



WINN FELINE FOUNDATION

For the Health and Well-being of All Cats

637 Wyckoff Ave., Suite 336, Wyckoff, NJ 07481 • www.winnfelinefoundation.org
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39th Annual Winn Symposium – June 29, 2017

“Ending FIP – Is There Hope?”

Transcript of Audio: Introductions, Dr. Niels Pedersen on FIP and Q&A

[Steve Dale]

How many of you here happen to have a cat? Alright, so first of all, for those who haven't raised their hand, I can help you, and I think there are people in this room that can help you, too. This is the 39th Annual Winn Feline Foundation Symposium, and what a brilliant idea this was to bring veterinary professionals – so there are many veterinary professionals in the room. Can you guys raise your hands? There are some folks here who are, I believe you say it's cat fanciers? Is that the right terminology? People who are responsible for breeding our cats – well, you're not all responsible for breeding my cat but are responsible for breeding cats just like our Roxy at home, a Devon Rex cat. How many here are fanciers? The idea of bringing veterinary professionals together with fanciers together with incidentally, cat lovers, just folks out there that love cats was not the idea of anyone who's currently involved with the Winn Feline Foundation. This is the 39th annual. So, 39 years ago, at the very first symposium, Niels Pedersen I believe was there, and he spoke with Dr. Fred Scott, who I've had the honor of meeting from the Cornell Feline Health Center, as it was known at the time. That happened in either 1979 or 1978, we're not too sure, but here's what we are sure of. The person whose idea that was who will, by the way, in three weeks, receive the American Veterinary Medical Association Humane Award; a person who was not only the leader but the steward and the visionary behind the Winn Feline Foundation for well over a decade; a person who many of you know, and if you don't know her, you know her name. She is in the room. Please help me acknowledge Joan Miller. [applause]

I don't know how many times Joan has been on the radio with me and, last night, some of us from the Winn Feline Foundation were on WGN radio. I work at WGN here in Chicago, and I didn't introduce myself. My name is [Steve Dale]. I've been on the board of the Winn Feline Foundation for 12 years. Joan has been on the radio with me, I don't know, 10 times over the years, something like that, and there are people that will say “Well, I'm on the radio, I'm fairly articulate.” I will tell you, this lady blows me away. Absolutely a brilliant person who I learn from every time I talk with her. So, the Winn Feline Foundation I say is responsible, for those of you who said “Yes, I have a cat,” for pretty much everything we know about cats, and I believe my next function is to introduce the immediate past president of that organization so he, therefore, is responsible for everything we know about cats - Dr. Glenn Olah.

[Glenn Olah]

Hello. My name is Glenn Olah. I am now the immediate past president as of yesterday, so all I can tell you is that I love cats. That's why I'm involved with Winn. I think the bottom left of the slide says it all for me. I think that's the vision statement for this organization. It's so true - every cat, every day, benefits from Winn-funded research. I think everybody should just remember that, because it is so true. I'm just going to talk briefly about Winn. Actually, you all know about Winn or you wouldn't be here, right? Yeah. So, I'm sort of talking to the choir, aren't I? But this, believe it or not, we had to have a strategic planning session for the last two days, which was very intense. This was our old mission statement. You know what, I don't have a slide with the new mission statement, but it's much simpler, much easier, at least for me, to remember. Basically, that Winn advances feline health through research and education. It's saying pretty much the same thing here, but I can remember it now! But I also can remember every cat, every day benefits from the Winn Feline supportive research. So, anyway, let's move on here. Just reiterating, Winn is about cats. A nonprofit organization set up almost 50 years ago and all about cats. There's no other organization in the world that is solely, solely dedicated to funding feline health studies.



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I like this graph. My background is physics, so I like graphs. The main thing from this graph, with the horizontal line being time, is we have a positive slope. Positive slope is the money coming in because of all of you, all the donors, all the people who support Winn, and you can see the growth. The two lines - one is the main Winn Great review session, which is the top line, and then we're also responsible for another session in the fall, called the Miller Trust, also a positive slope, which is good. This past year we've had a great year. We've funded over \$360,000 directly going to feline health studies. That's good! Thank you. [applause]

Okay, yes, I am the immediate past president. Now, I'd like to introduce to you our new president. It's my honor to introduce you to Sheila Norton. She's been -- well, you can see in her background. This is exciting to have her as our president. She's been associated with Winn for at least the last six years as a reviewer, so we know her. She has a background in nonprofits. Oh my gosh, how did we find you? Anyway, I'd like for you to meet our new president, Dr. Shila Nordone.

[Shila Nordone]

I just wanted to take the chance to say hello to all of you and let you know how excited I am to be a part of this dynamic board; a group of individuals who are really driven every day to meet this foundation's mission, and I definitely want to meet all the passionate donors that have supported Winn for 50 years. That's an incredible accomplishment. I hope to meet each and every one of you at some time while I'm in this role, but I also want to take the chance to say thank you to Dr. Glenn Olah. Where are you, Glenn? (Come back up here!) He has lead this organization as president of our board for three years. He is an incredibly driven guy. In every one of these meetings, he keeps us on task and keeps us focused on our mission. So, all of us on the board wanted to say thank you to Dr. Glenn Olah for his service and his passion for cats.

[Glenn Olah]

Thank you. I don't think I have anything more to say. This is a surprise. Thank you very much.

[Steve Dale]

Oh, so I'm doing this, which is good. That's okay. Some number of years ago, this lady called us at the Winn Feline Foundation and said, "I believe that I would like to start a fund. It was like "I'm going to build a show" and she did. She has an army of followers. She calls them her family. All across not just the part of the country she lives in, not just America, not just North America, but the world - Susan Gingrich. [applause]

[Susan Gingrich]

I never turn down an opportunity to talk, especially about FIP. In the end of 2004, our precious little Bria was diagnosed with tentative FIP. I was fortunate, because I had a vet who had FIP experience, and she knew that she needed to rule out other things before determining, most likely, it was the FIP. She also sent me to an internal medicine specialist to rule out other causes but, as it turned out, the reason for Bria's abnormal breathing and for fluid in one side of her chest was most likely FIP. Bria was a fighter. She had such spirit. She fought for almost four months with wet FIP, with only a couple of drainings and pred. When she left us on April 19th, 2005, she left us unable to forget her or forget FIP. I wanted to do something; Jim wanted to do something, and we approached the Winn Feline Foundation with a proposal to start the Bria Fund. I knew Winn superficially, because I belonged to the National Birman Fanciers, and I knew that Janet Wolf was the executive director, so I submitted a proposal to her, and she was very enthusiastic about it, but she made it very clear that, if the board approved it, I would have ownership of it. I would be expected to lead the Bria Fund, to get donors, to help make sure that research that's so important to end FIP was funded. Fortunately, the board approved, and the Bria Fund was announced at the Cat Writers' Association conference in November 2005. It took a couple of years of fundraising before we were able to



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fund a couple of studies. Since that time, we have funded 21 studies through the Bria Fund. What I didn't know before coming to Winn was that Winn had a long and deep history of funding FIP studies. Many, many, many years. Winn has funded more individual studies than any other nonprofit. We have made tremendous progress in the last 11 years towards understanding FIP, better tests, better understanding of treatments that may be able to help and more and more research. I think the Bria Fund and our pushing it helped other researchers also be interested in FIP research, besides the Dean of FIP Research, Dr. Pedersen. We've funded studies from not just this country, but also out of the country. They have helped us solve pieces of the FIP puzzle, but it isn't finished. We have some cats that it looks like are being helped by prophylactic things, and they're able to live with FIP as a chronic disease, but it's very few cats. The most exciting research in my whole over 11 years of dealing with FIP, is the research that Dr. Pedersen is doing right now. It's amazing. I mean, to think that there are cats that potentially can be cured of FIP. It's ongoing. We don't have all the answers yet, but my goodness when we lost Bria, we had no hope. There was nothing. We have hope now, and we have proof that there are things that potentially can cure cats with FIP and to help cats dealing with FIP. We've come far, but we have further to go. Research and research donations for FIP are more important than ever. Dr. Pedersen talked to us yesterday about the potential for FIP research concerning antivirals. We all have got to do what we can do to raise FIP awareness, to encourage people to give to FIP research, to give to the Bria Fund in particular, because we are different. Unlike other nonprofits, every single dollar you give to the Bria Fund only goes for FIP research. None of it is used for university overhead or for Winn's administrative cost. There's still a lot of education, a lot of attention that's needed. There is more continuing education for vets, but much more is needed. Cats are still misdiagnosed and die needlessly because vets don't have the information that they need to know that FIP has changed a lot since they were in veterinary school.

Thank you for coming, and we appreciate your being here. I do want to acknowledge a few people that are here. These are individuals that have been involved with Dr. Pedersen's clinical research. We have Walt and Scott, who have Flora. Flora was in Dr. Pedersen's clinical trial last year. It's been over a year since she started the treatment. The treatment ended a while ago, and she's doing very well. August 6 was the last dose, and she has no symptoms. She's not on any medications related to FIP. Bloodwork is normal, and I'll let Dr. Pedersen talk about his second clinical trial, which is going on. We have Deb and her husband Jamie here. They have Luna, an adorable little Savannah. She's six months old. She's the new trial and doing well, and we're very fortunate that the people I just introduced have shared their experience to a certain extent with the Facebook Groups community.

Also, I want to introduce Peter Cohen - partner, Hero. Welcome. I mainly want to introduce Peter, but just a little bit -- I became aware of Peter through our Facebook group. We have two FIP groups - one is called FIP Fighters, on Facebook, and we have one on Yahoo called FIP Cat Support. They have been amazing for sharing information about FIP. Some of you that dealt with FIP years ago probably also know that besides it being terrifying and depressing, it's also a very lonely disease, when you didn't know anybody else that ever experienced it, and the social media has really helped us reach out and to connect, support people dealing with FIP or maybe not dealing with FIP, in some cases, and that's how I met Peter, because Peter started sharing his experience with Dr. Pedersen. Peter is my very close brother in our FIP family. Many of you may be in our family and not even know it, but anybody that ever experienced FIP is in our family. None of us wanted to experience it, but we are in the family, and we hate FIP. It's cruel and unforgiving, and it's our enemy, and we work closely for FIP research and to end FIP. Peter, would you like to say a few words?

[Peter Cohen]

Hello, everyone. A lot of people! My name is Peter Cohen, and I am a co-founder at Zen By Cat. I am standing here, in front of way too many people, way outside my comfort zone, because I wanted to do something useful in the fight against FIP. The stark contrast between Miss Bean losing her battle with this horrible disease and Smoky



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winning his is what motivates me. To convince people that it just takes a lot of us giving a little each week or each month to raise the funds that are needed to give all cats afflicted with FIP the chance at life that Smoky received. Dr. Pedersen and the other amazing researchers that he works with have done, and are doing, the hard work. They have found drugs that will end FIP, and they are having success. Instead of asking for donations to find a cure, we are now actively seeking donations to fund a cure. FIP survivors, like Smoky and Flora and Oakley and Luna and others, clearly show that a cure is possible. Cats play an important role in our lives. We bond with them, we take care of them. Some would say we serve them, and we love them. As their human guardians, it is our responsibility to get the funds needed to get these drugs to market. Many of the people in this room have been fighting this battle a lot longer than I have. I am humbled that so many people could keep in this fight for so long and not give up. Raising money is really hard. Please reach out to me, and let's work together to find a way to end the heartache that is FIP. Thank you.

[Steve Dale]

We're going to watch this film put together by Peter. It's very emotional. I've already seen it. It'll make you cry - it made me cry. Bear with me.

Zen by Cat Movie

We've been rescuing cats for almost 30 years. Once I started adopting, it kind of became an obsession, then it just kind of snowballed. We currently have 22 cats, which sounds insane, and it wasn't planned. This is Smudge. There's Poppy Seed, Cheesecake, Chocolate, Secret, Donut, Vanilla, Smoky, MiniBean, Mikan, Climber - I'm forgetting a whole bunch. I can't remember any more!

If you're a cat person, to me, the moment that the cat trusts you, because cats are usually wary, I just find that aspect of it to be amazing. I like to adopt cats that are hard to place. We take black cats because of superstition they are hard to place and I like to take shy cats, because most people don't want them. All cats are different. Some cats want to be social, some want to be in their own space, and we started building catwalks about 25 years ago. The house is filled with walks, it's filled with fountains, both for the sound and because the cats drink from them. We have music playing all the time, because I and the cats like it. The reward is we have this Zen-like experience for us and the cats, twenty-two cats that are 22 different sentient beings.

One of the things that I experienced, personally, when you lose -- I mean, we lose cats all the time, that's part of having animals in your life, but when you lose a kitten to FIP, there's something especially horrific about it. It changes you. My first experience with FIP was four years ago. We had adopted two kittens and one of them, Peanut, was diagnosed with FIP, and I had never heard of it, and it's this horrible disease that usually attacks kittens and older cats, and it's always lethal. Peanut lasted five days from diagnosis to the time we had to euthanize her, but at the time I was told it was really rare, and I thought, I've been doing this for 25 years, this is the first time I saw it, I'll never see it again. But then, last year, we adopted two more kittens, Vanilla and Miss Bean, and Miss Bean was diagnosed with FIP, but through the magic of the internet, and the fame of my cats, I was put in touch with UC Davis - they were starting a drug trial, and Miss Bean was accepted. Unfortunately, she didn't respond well to the drug. She went up and down over a four-week period, and there was always hope.

It's really hard, as humans who care for cats, we have to decide, you know, how long to let them fight when they're fighting things like this, and Miss Bean kept having a chance. Ultimately, she didn't make it, but they learned a great deal from studying her. I found Smoky, another FIP kitten, who had been accepted into the program, and Smoky was diagnosed with FIP when he was about 15 weeks old. To knowingly adopt an FIP kitten was a little scary, knowing what I had just gone through and that I was probably going to go through that again, but Smoky,



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unlike Miss Bean, did respond - one of the rare ones that did. There were a couple of times when he almost died, and we had to decide, do we keep going or do we give up, and luckily we kept going. He's one of the most stoic, nothing-stops-him cats. He went through 12 weeks of twice-daily painful shots, that was the protocol and he would never run away. He's now completely recovered, and he's now one of six in a row that they think they've cured. They're afraid to say the word 'cured,' but he's been in remission six months almost, and he is now one of the very few cats who's survived FIP. He's a pretty special cat. The researchers who have been studying this, they've been studying it for 50 years, and only this year is the first time they were actually able to cure a cat, and so that inspired us to use the fame of Zen By Cat to raise money for the researchers to speed this drug. My main business is construction - I have a construction company, which is how I could build all these catwalks, and for years people have asked me, "Can you do this in my house?" and we're selling the catwalks in kit form, and they'll be like ours, they'll be floating, and the proceeds of that will be going toward Zen By Cat, which goes toward FIP research. I've teamed up with this amazing company called ViviPet.

Before I started this business, I worked in a CPA firm, had a very stable life, and then I adopted a cat. His name was Tex. Unfortunately, Tex passed away a couple months right after I adopted him because of FIP wet. This disease is very horrible to me, because it's like it happened so fast, and for the very beginning few weeks, we couldn't tell what's this disease was. After I saw Smoky - he survived it from the research. That's a big hope to me, so I could remove a very dark spot in my heart. Basically, I started this business beginning with the feeders. I hope this business could support the research of FIP, the low awareness of the cat owners and also the shortness of funding of the research institution. Make this disease that's for decades and decades, very hard for cats to survive from it.

I belong to a lot of social media sites that these groups for FIP, both private and open groups and, every day, the story is the same. You see new members joining and saying, you know, I adopted a kitten for me or my kid, and he got a cold, and suddenly I'm told I have to euthanize it, and it's so horrific. You are so helpless. You are told there's nothing you can do. The idea of Zen By Cat is sign up \$5 a month, \$10 a month. If we could get thousands of people to sign up with \$10 a month donations, small donations, we could create a steady revenue flow for the researchers, and we can also educate people and bring awareness, and the fact that they have cured kittens says they're on the right track, they just need funding. These people are heartbroken, and they want to do something, and we're trying to give them a constructive way to do something. That's my great hope for this.

[applause]

[Steve Dale]

Thank you for coming to all of you, and to Peter, thank you very much, and many of you have traveled from out of state to come here, so thank you. I've had this opportunity several times now in my career to introduce Dr. Niels Pedersen, and I've often said that he wrote the book on feline health, the first book on feline health, and people say, "I'm sure, that's a nice thing to say, wonderful." He wrote the first book on feline health! It's right here! Dr. Pedersen, I will be getting your autograph later. I've got the first book on feline health. I could name all the awards he has garnered over his career, but then you would never be hearing him, because we'd have to give up this room by the time I'd still be rattling off all the awards. I will say one of them - the AVMA Winn Feline Foundation Award from 2012. If there's a retrovirus in cats, he has studied it, and a couple of years ago, he said he is determined, before he absolutely retires from UC Davis, where he's currently professor emeritus of veterinary medicine and epidemiology, that before he leaves, he is going to do something - he is going to kick open the door so we can all do something about FIP. He has done that. We're going to hear about it. We're going to see about it right now. Please help me welcome Dr. Niels Pedersen.



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[Niels Pedersen]

It's my pleasure to be here to speak to you. I'm really happy to have join Joan (Miller) here and also say some really good words about Peter, who obviously takes good care of her - really, always an inspiration to me, and I can remember starting off my career with a few people that seemed to have faith in me, and you were one of them. (Audio problem) Anyway - what can I say. Okay, let's just talk about this disease that I've studied since my second year of veterinary school, in 1964. Published the first paper on this disease along with a graduate student in pathology in 1965, and so this has been a real quest for me, and it isn't an easy disease to study. You're right, there was not a lot of money to study it. It is the most complex infectious disease as far as I'm concerned, and I've worked on HIV, I work on SIV, I work with FIV, I work on all sorts of diseases. I can tell you that this disease is the most complex from all aspects of any infectious disease of man or animals, so it hasn't been easy to unravel the layers that you have to unravel. The secret sits in the middle and knows kind of thing, and we heard in this thing, so we've been kind of circulating it around and around, peeling off the onion layer at layer. I think we've gotten to a point now where it becomes obvious what the best approach is to this disease, and we'll talk about that at the end, but I want to talk to you now about the disease. A little bit maybe you already know about it. There will be some pictures in here that are a little graphic and shocking, but I want to shock you, because this is grossly the worst-looking disease that you could ever imagine, and we actually see that cat from the outside, but when you look inside and see how terrible those lesions are and see how those cats must be suffering, because if that was a human, they would never suffer like that with those types of lesions. So, we'll talk a little bit about that.

Okay, there is a little mistake in this - I am no longer director. I was the founder of the Center for Companion Animal Health. I was its director for 18 years, and it is now celebrating its 25th year, and so I'm proud of that accomplishment. Okay, just to tell you that I do have cats, Gerry and I have cats. These are the two, we're in a heat wave in California, and so our cats take it easy during the day, so they find a cool spot near the house, and they are two great buddies. By the way, I do have a whole pile of cats that, when they come out of their one-year quarantine, yes, that means they're cats that have survived FIP, experimental FIP, but we have to keep them for a year before we can declare them not at risk with the disease. I have about 20 of them that need homes, and so these are the greatest cats on Earth. They're obviously random-breds, but they are really great cats. So, let me know.

Okay, coronaviruses. These are what we call RNA viruses, and I like to throw science into my talks, so some of you may remember some of your biology, some of you may not know what I'm talking about, but I hope to try to make it clear, but there are basically two types of viruses - RNA viruses whose nucleic acid is ribonucleic acid, and our DNA viruses. DNA viruses are like herpes virus, and those are the classic DNA viruses. Most of the viruses that attack us are RNA viruses, so the flu, rhinoviruses, noroviruses, coronaviruses, HIV, HTLV. You just go on and on. Those are all RNA viruses, and one of the things that we'll bring back to that point is that these are pathogens that adapted themselves to every species of animal, so every animal species has its own little coronavirus, and several have more than one, and most of these coronaviruses are familiar to us as causing bronchitis, they are viruses that attack the lining of the respiratory tract and of the intestinal tract, so they're causes of winter dysentery, avian bronchitis, diarrhea in calves, so they are very common to us as pathogens. I want to talk about one virus in particular, and the nomenclature gets a little bit woozy as far as these viruses. A lot of the Europeans like to just lump them all together and call them feline coronavirus, but there is no virus that exists out in nature called feline coronavirus as a pathogen. That's a subspecies name, and the viruses that we're interested are two, and one is called feline enteric coronavirus and one is called feline infectious peritonitis virus. Those are the two viruses that are pathogens that we deal with as pet owners, as cat owners, cat breeders, cat lovers, as well as veterinarians. So, the enteric coronavirus is ubiquitous throughout the world in both domestic and wild felids of all sorts - lions, tigers, so forth and so on, and this is its nomenclature right here, and I just want to let you know that we're interested in these two that are called biotypes, FECV, feline enteric coronavirus and FIP virus, and they come in a couple of forms and so, again, these are RNA viruses. They mutate a lot, and they adapt themselves and



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change a little bit, so there are different types of them out there, but the one I wanted to talk about is feline enteric coronavirus. This virus is all over the place, and it's spread by fecal/oral spread, so we have carrier cats, often younger cats, that are shedding this virus in their feces and, by the way, interestingly, because cats have a very short intestinal tract - they have one of the shortest intestinal tracts of any species that we deal with; they don't have a rumen and a reticulum, an omasum, an abomasum, a spiral colon, and all those other things that we see in these other species. We just have a straight tube, it's that long, and that's because they eat meat, and they don't need a lot of fancy digestive processes to deal with it. Strangely enough, the enteric coronavirus infection of cats because of this anatomy, is not really a pathogen at all. They don't really get sick, most of them. They may get a mild diarrhea that lasts a day or two, but it's really not a disease of any importance in terms of its own ability to cause disease, and it's going to be found, being a fecal or oral pathogen, it's going to be found in any area where you have a lot of cats, and especially kittens, because kittens are the ones that are going to be the most susceptible. In nature, if the mother is allowed to nurse her kittens, the infection starts about nine weeks of age, so the mother gives them some protection for the first few months of life, which she does for a lot of other things as well, a lot of other infections. Usually, the earliest we see this infection in nature would be about nine weeks of age, and it's going to be obviously in high-density cat populations where kittens are part of the equation. You need kittens in there, because basically what you're creating is an oven or a furnace, and you just keep stoking it with wood and keeping the fire going, and you can even get it hotter by throwing in more wood, and what is the wood? It's kittens. You just keep throwing that into the mix. We see it in dense urban or rural free-roaming cat populations, and you've seen these if any of you have gone to Athens, in the Parthenon in Athens, you've seen all the cats. If you've gone to the Coliseum in Rome, you've seen all the cats, and there are many places like this in the world. In fact, when I go to Turkey, I've been to Turkey several times with my wife and we're in a little restaurant outside, or in Israel, and surrounding us are cats, and they always end up under my feet for some reason.

But anyway, this is a picture. I also want to make a point right here, just an interesting thing. Most of the cats you're going to be seeing are red. Now, the red gene confers extra strength and health and some vigor. So, the red gene is becoming dominant all over the world, so if you look at pictures of cats anywhere in the world that are community cats or feral cats, whatever you call them, you'll see they're all torties and reds, right? That's an interesting thing, and we see this occasionally in these high-density populations. I want to make the point here, because Flora comes from a park in Peru. She was rescued from a park in Peru and Walt brought here to the United States, and so she's an example of coming from one of those types of environments. Now, the number one environment that we are seeing kittens from - and, by the way, the incidence of FIP is really climbing, it's really going up. I don't think that's just because of better knowledge of that. I think it's actually there are just more and more cases of it occurring, because more and more of our kittens are coming from these specific environments, and the number one environment by far we're seeing them from is from kitten foster rescue groups, and this is probably obvious because what happens is that many of these kittens are raised without their mothers. They're bottle-fed, and some of them not under the best circumstances, because what's happening is the shelters are pushing back on the number of cats that they're taking, because they don't want to be responsible for euthanizing all these excess cats, so they push back now, and when they push back, who's going to take them all in? Basically it's going to be these groups of people that are loose organizations, that are basically volunteers, and some of these may have 200 cats in a house, some of them may only have one litter in a house, and so you have everything in between, and so this is another area. These are kittens that are often weaned earlier, may not never have nursed, that are in a very dense environment. They're exposed to massive amounts of viruses, the enteric virus, so it's a perfect kind of environment for FIP. I mentioned conventional shelters, these would be city, community, SPCA type things, county shelters and that. Again, we don't see as much from them anymore, because they're pushing back all of those kittens. The high-risk kittens, they're pushing back into these kitten rescue organizations and so it's a little unfair that the kitten rescues are taking the brunt of it. Believe me, I'm not criticizing kitten rescues, I'm just saying that that's what's happening. I do, and I've talked to the board about this, I that I think that people should realize



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that cats are not pets that have gone feral. The cats that are out there in the community running around are not somebody's pets that have gone feral. These cats have evolved with humans since the Neolithic period, when our ancestors changed from hunter-gatherers to farmers. We had grain, we raised grain, and the mice came in from Asia by the flood where they came from, and they were associated with humans, they evolved with humans in Asia. They flooded over into the Middle East, into the Fertile Crescent, and guess what, the desert cats came in around those communities, because there was plenty of food in terms of rodents. Also, protection from their predators as well, because their predators weren't going to come near the humans and so there was this close association that occurred and then, slowly, the same thing happened. Now, realize dog domestication occurred now, I think, 45,000 years ago, much further back, when dogs were with us on the hunt when we were hunter-gatherers, chasing the game north and south, during the great ice ages. Basically, that relationship also was the same kind of thing. Dogs started as a certain type of wolf and started associating more with humans, because associating with humans allowed them both food and protection, and then, slowly, slowly by what we call positive selection, in this case not deliberate maybe, but by positive selection, they became closer and closer and eventually they were part of our homes and our hearts, so this is exactly what happens with cats. I would like people to know that community cats or cats that are out there are not pets that have gone wild. They don't need to all be taken in and found homes for. We need to deal with and accept that. When Fish and Game says that we need to get rid of the cats from all of our parks because they're artificial, we want to get back to the way it was when the Indians were here. Well when the Indians came there were mastodons and all sorts of beasts that they got rid of so let's talk about and be truthful about evolution, it's a normal process whether we like it or not. Cats are the principal small carnivore in all human ecosystems, the principal small carnivore in all human ecosystems. Without them, we know what happens, there are plenty of examples in that we get overrun with rodents. I can attest to you from having a cat, there's no beast like a cat that can kill mice faster than a cat. They do a very good job of keeping that population down. Of course, the other source are pedigree catteries, kittens from pedigree catteries and in the case of pedigree catteries we think that there's more that heritable components are also involved to a greater extent than in the random-bred populations. We know from our research on genetics that the genetic susceptibility that's in pedigree cats to FIP is what we call polygenic and involves hundreds of different negative polymorphisms that are brought together and sometimes are concentrated by what? Inbreeding. The more inbred and inbreeding that you have, the higher the risk. We've done some work in some breeds and the heritability in pedigree cats is probably about 50%. In other words, accounts for about 50% of the incidence of the disease. You're going to ask me which breeds have the most problem, and I'm not going to be able to tell you that answer because all the breeds we see the most FIP in are also the most popular breeds so, you know, you really have to do some true incidence studies, which is something, by the way, that I suggested that Winn actually study. Is there a breed susceptibility or not?

Okay, so let's talk about enteric coronavirus infection. The initial infection, if they're being weaned normally, nursed is around nine weeks of age, it's unapparent before that. Now, if we look at, this is just shelter populations, and I'm showing you some graphs. I've got a little pointer here. Just look at this first thing right here and look at the light blue, and these are the percentage of kittens that shed coronavirus in their feces at the time they're brought to the shelter, so these are kittens coming from the outdoors. So, you can see that 30% of them are already shedding by the time they get to the shelter but, within a week or two, that goes up to 60%, so you can see how fast this infection can spread. It really likes these environments, and the host cell, this green is fluorescent staining for a virus. This happens to be what's called a villi in the intestines. This is like your intestine has finger-like poles, and at the tips of the fingers are the cells that do the major transport of electrolytes and nutrients and so forth, and that virus attacks only those cells. Just think of this virus. It has evolved to attack a single cell type in the body, and it happens to be that cell type that's in the intestine. So, what happens during the primary infection is that this is pretty widespread in the bowel, but as the infection proceeds, the infection settles into the colon, mainly, where it persists in the colon, and then you see this pattern here where again, graphs, I know you hate graphs, but if you look at the blue line, you can see this is the level of virus being shed, and you could see that goes straight up, and then



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you see it slowly goes down, and it kind of goes up and down and then slowly disappears over and period of many weeks, so it takes a long time for these cats to develop immunity, and they slowly build up immunity and then, associated with that, antibodies come out, and then, the strange thing about this, but not so strange, because we know this for all mucosal infections, is that when they lose the infection by getting immunity, they also lose their immunity, and so then can be reinfected. So, their immunity keeps going, they lose, this is common with all of our enteric infections, is that infections of the linings of our respiratory tract and intestinal tract are often short-lived, immunity is short-lived, and you can get them again. That's why you keep getting colds and that, some of these things just keep going.

I'm going to skip over that and say so what? Why do I spend all this time talking about enteric coronavirus? Because from enteric coronavirus, that's where FIP comes, and I'll talk about that a little bit just now, but I want to say one thing. Before we knew that FIP virus is just a simple mutation that occurred during this enteric coronavirus infection phase, we were so confused, because we didn't realize we were dealing with two viruses that were 99.8% identical to each other that were in the same animal, but that did exactly totally different things. One of them was this little simple pathogen just in these little gut cells, and then this other one was this nasty virus that got into the lymph nodes in our intestinal tract and then got into these cell types called the macrophages in the lymph nodes and became like tuberculosis. Now, tuberculosis is the nearest disease to FIP in humans is tuberculosis and, in fact, bovine tuberculosis looks exactly like FIP in cats, but bovine tuberculosis has been almost eliminated completely as a cause of disease in cats and in humans by getting rid of it in our dairies, by pasteurizing our milk and testing our cows and that. Interestingly, 100 years ago, the best indicator of the presence of bovine tuberculosis in a herd was tuberculosis in the cats that were in those environments.

So, bovine tuberculosis in cats and in humans is a very good model. Okay, so FIP, during this primary infection, during this lengthy infection, this lengthy period, and maybe even during periods of secondary infection - reinfection - we have this situation where these viruses, I told you, they are RNA viruses. RNA viruses have to convert their RNA to DNA in the cell before they can replicate themselves, so this RNA-to-DNA change involves a process called reverse transcription, because the normal process of the cell's function is for our DNA to make RNA and then RNA to make protein. That's the normal course, but these viruses have turned that upside down, so they take their RNA, make it into DNA, and then they make it into RNA. So, this is called reverse transcription, and so this term, reverse transcriptase, you know, right? Because of the AIDS epidemic, HIV, you know that term, reverse transcriptase, because HIV is so intricately involved in this reverse transcription, like any RNA virus. Okay, so, this mutation occurs, and we know that this mutation occurs quite often, and it involves three spots in the viral genetic makeup, and three distinct areas that you have to have mutations occur. We know what types of mutations occur in each of those spots. We don't know what those mutations do in terms of making those horrendous pathogens, but we know which types of mutations occur. We know the mutations, and these mutations, as I said, occur... We don't know what percentage, but one experiment that we did suggests that up to 20% of the time, during the initial phase of the enteric virus infection, probably 20% of those kittens will produce a mutant that will get into a lymph node, get into a macrophage, that's capable of causing FIP. Now, we know the incidence worldwide of FIP is 0.3%, so that's 3 out of 1000. We know the incidence in a cattery or a kitten rescue or shelter is from 1% to 5%. It's about that. We know, then, that it doesn't match up. If 20% of these cats have a mutant capable of causing FIP and the incidence is 1%, what does that tell us? It tells us that most cats can fight this infection. They have the ability to mount an effective immune response, so that's something that we didn't know before. The assumption is that it's 100% lethal. No, it's not 100% lethal. It's only a little bit lethal, but to those that can fight the infection off, that can develop a protective immune response, those are the ones that are going to be your problem. Okay, so let's get that out of the way. We also know now that what happens is that when that mutation occurs, it often sits in those lymph nodes, just like tuberculosis. Now, remember that 40% of the people in the world have TB in a lymph node in their chest. Many of you do, but it doesn't do anything, it just sits there. In



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fact, it's good for most of us, because it keeps perpetuating our immune response, it keeps our immune response alive, but we know that certain people start to lose it, and some of them lose it gradually and get sicker and may only just have a cough or something, and what did they do in the old days? If they could, they'd send you down to the sunny climates and put you on the beach and rest you, let your immune system build back up again, but we also know that, in some cases, the tuberculosis can become consumptive. It can just consume you. It can just keep getting worse and worse and spreading and spreading and spreading and consume you, and that's what happens in FIP. So, 40% of you have -- maybe not of you, but 40% in the world have TB and don't know it, but only a small percentage of you are going to be sick, so that's the kind of thing we're dealing with. Now, we have two forms of the disease, and what happens is that some of these cats, it sits around in a lymph node, doesn't do anything, but in some cases that just kind of simmers away, it cooks away at different speeds and different rates depending on the heritable resistance of the cat, depending on the stress factors that are thrown at it. Early spay/neuter at 8 to 10 weeks of age, being taken from your mother when you're a newborn and not allowed to nurse. All of these things are going to have an influence on how these cats react to this situation. Basically, they're going along with it sitting in there and then what happens is some of them, it eventually starts to build up, just like in most cases of consumptive TB. It gets worse and worse, and then it becomes clinical, and in the earliest clinical stages, you may only see what's called dry FIP. You may see enlarged lymph nodes in the abdomen or the chest or something like this. It may be very limited, or it may spread to the brain or the eyes but, in most cases, what you see is you see this belly full of fluid, or that 1 in 5, 1 in 10 cases, a chest full of fluid. In the chest disease, by the way, the disease usually starts in a single lung lobe, it's focalized in a lung lobe, and that lung lobe is really badly involved, and then it leaks fluid into the chest slowly, and when that fluid builds up to a certain level they can't breathe, and then you have to drain the fluid, and then they can go for several weeks, and then you have to drain it again, that type of thing. Basically, that's where we are at. What I'm trying to say is that those cats, once they get to the point of having effusive FIP, they have really lost it. At that point, their immune systems have totally collapsed. The cats that have dry FIP, they're still fighting it, and some of them can live a long time, as some of the work of Dr. Al Legendre and those have shown, those cats can live for months and a year or more, some of them, in a state of health, but once they go wet, it can go pretty fast. Although I can tell you I have seen a cat that has wet FIP for over a year, running around with a big belly full of fluid and seemingly otherwise normal. There are all sorts of little strange twists to this thing.

We don't know how long the actual disease course is. Why don't we know? You'll see a lot of literature, they'll tell you, "Well, the average disease course is four weeks, five weeks, six weeks." What kills most cats with FIP? Can anybody tell me what kills most cats with FIP? Euthanasia solution. They don't die a natural death, and so it makes it hard, because every one of you has a different idea of how long you want to keep these cats alive and how much you want to fight and how much symptomatic treatment you want to give, and so forth.

Why is it not taken seriously? I don't know. It has a tremendous emotional and financial drain on people that have to fight it. This financial drain and emotional drain I don't have to tell you about. The financial drain is getting to be quite horrendous. We recently took a cat in that had been diagnosed with FIP, thoracic FIP. He had to go to an emergency clinic then back to their regular veterinarian and had some fluid drained and all that, and a week and a half of this was \$6,000, so this can be a big financial drain, because it hits people so fast. It's just like everybody says it's all of a sudden, your cats healthy and then all of a sudden, it's got FIP. What are you going to do? You've got to do something and so you get brought into this thing, and it can be both an emotional and a financial drain. It is increasing worldwide but, again, I can't tell you the exact figures, because those studies haven't been done.

Okay, what are the major risk factors?



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We did a study of major risk factors, we did a study, Janet Foley and I did a study, which is actually one of the most quoted papers in the Journal of Feline Medicine and Surgery over all time, and so what some of these studies do is trying to determine what are the risks, and so this dealt with both catteries and shelters, and so what we tried to do is take away all the husbandry issues - you know, overcrowding and all those other things - and they really weren't found to be as important as we had thought, and when it came down to just what the nitty-gritty was, the greatest risk factor that increased the incidence of FIP was how many cats in that group were shedding in their feces, so the more shedders there were, the more apt they were to have FIP. The magnitude of the shedding, so not only the percentage of the shedders, but how much each of them were shedding. The more each one of them were shedding, the higher the incidence. Then, also, we looked at antibodies and the more cats in that population that had titers of 100 to 400 or greater, the more apt you were to see FIP. Age was definitely a predisposing cause, with most cases being in the 4- to 29-month age range, and there was a genetic predisposition. We could see certain males were more apt to be associated with certain breeding, and that is not because certain males, that male is X-linked or Y-linked characteristic of just males; it's because certain males bred to certain females. The female also can have these bad risk factors, but if you have a good male that doesn't have many risk factors, and you breed him to a whole bunch of females that don't have these risk factors, that's going to look really good, right? That male's going to look good, but if you have a male that has a lot of risk factors, and you take and you use him on a whole bunch of females that have a lot of risk factors, that's when, all of a sudden, you will see several litters where you may have one or two or more cases of FIP in those litters. That lead to our suggestion that if you do anything genetically, do not use "at risk" males at least don't use males because males produce more kittens and have a greater influence on the situation than females, not because males are genetically worse than female.

Okay, so you all know this picture of this distended abdomen and you all know this fluid, this yellowish fluid and it's varying color. The more yellow it is, the more inflamed the intestine is, the more microhemorrhaging is occurring in the intestine, the more severe the disease is. The more fluid, the lighter yellow it is, the less cells it has, the less inflammatory that is, the longer the cat will probably survive. There's a lot that you can tell. The important thing for a veterinarian... This is why veterinarians tell me that it's hard to diagnose FIP. When you get a young cat coming in with an extended abdomen that comes from one of those environments, you pull this kind of fluid out - what else? You have your diagnosis right there. You don't even need to take any fancy blood tests after that.

I wanted to show you where this fluid's coming from and why. This is looking at the abdomen of one of these cats, and you can see this thickening, you can see the lesions on the spleen, you can see this omentum is thickened and yellow from hemorrhaging in it, you can see the fibrin on the liver, and certainly you can see this ton of fluid, hundreds of milliliters of fluid in the abdomen and, by the way, there's no reason to take that fluid off unless there's so much fluid that it's causing problems with pressing on the diaphragm and breathing, because if you take that fluid out, it's just going to come back again, and then you're just going to take all that protein and fluid, you're just going to dump it out, and then the cat has to take it out of its own muscles and waste it. Chest fluid, yes, you have to get it out and, actually, chest fluid doesn't come back nearly as fast as abdominal fluid.

In the case of dry FIP, the lesions are more concentrated in certain areas, and so the immunity in wet FIP is such that it allows the infection to just spread all over the place and FIP is a disease of small blood vessels, so it is basically an inflammation of the small venules that line the intestines and what's called the omentum and the mesenteries and that. Now, in the dry form, there is more immunity, and the immunity tends to push the virus back into certain sites and cause more like the classical what we call granulomas or tubercles or tuberculosis. These are more like tumor-like lesions, and we don't usually see the fluid, and you can see these lesions on the kidneys, and all of those lesions are surface-oriented, so all of those lesions started as inflammation on the surface, and then, as



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immunity damped all that surface inflammation down, you have these residues of infection that extend down from the surface into the normal kidney or, in this case, tissue.

This is a cat that has a hepatic lymph node. Again, you notice the yellow. That's because of hemorrhage in there. You see these lesions on the liver and then here is another lymph node that's enlarged in this cat. So, these cats are going to have fever, they're going to be losing weight slowly, they're obviously going to be ill but not maybe as ill as the ones that come in with the wet FIP. Now, in some cases, the FIP can also attack the eyes. In this case, you see the cloudiness in the eyes, both eyes. This is inflammation of the iris, it's called anterior uveitis. You see some redness there and hemorrhage, so this cloudiness is from inflammation, inflammatory fluid and cells behind the cornea, and here we have another where we have these precipitates, these are called mutton-fat precipitates. These are fairly diagnostic of FIP, they're not seen... These are aggregates of inflammatory cells, and they're called mutton-fat precipitates because it looks like somebody's taken a handful of mutton and thrown it up against the window. We're seeing that from the other side. There are these kinds of interesting terms that come about in both medicine and veterinary medicine. They all come from agriculture, by the way and are ultimately from the home.

This is Sebastian.

Sebastian has neurologic FIP. You can take my word for it - this cat is totally incoordinated, so he's going to move around, he's totally incoordinated. Incoordination is a prime sign of neurologic involvement with feline infectious peritonitis. Interestingly, cats that have ocular or neurologic FIP often don't have lesions in their abdomen and other places, so the virus kind of just ends up going into the brain and causing an encephalitis, and it can involve the spine as well. Okay, so what about diagnosing it? This is where it gets a little bit where I don't understand. Veterinarians have come to this conclusion that FIP is a difficult diagnosis to make, and I cannot understand that. Maybe because I know the disease so much that I don't need to do all of this stuff, but it doesn't seem to be that unobvious, and the interesting thing is that there are many cases when the owner, especially the cat owners that are familiar with cats, like breeders, they know it's FIP before the veterinarian knows it's FIP. Basically I think the reason is that it has been a fatal disease and so, basically, they're uncomfortable with telling you "Your cat has this fatal disease called FIP," because you're going to ask them, "Are you sure, doc? Are you 100% sure?" The doc says "I can't be 100% sure, maybe I can be 99%, but I can't be 100%." Then you get sucked into this thing of trying to come up with other diagnostic tests that are more and more complicated, that are only like 70% accurate. They may be definitive, but they're only 70% accurate, so for instance, if you have this cat with this fluid, and the fluid is characteristic in every way, and the history is characteristic, and you say, "Well, I'm not going to set the diagnosis unless I take some of that fluid," and you analyze it and detect the viral genome by polymerase chain reaction, PCR, or whatever. Then you send it out, then it comes back negative, 30% of them do. Then what are you going to say? Here's your definitive test that's not definitive. Then you may try another test, another test, and so this is where you get led down into these expensive kinds of things.

This is not my slide by the way. This is a slide that's taken that supposedly tells you how you can make a diagnosis of FIP. Now, I would like anybody to see if they could figure this one out, okay? But, interestingly, when you get to number five, right in the middle, so you get to five right here, it says treat, and treat is pred, feline interferon-omega, that's been proven years ago by tests not to work. Why is this still used? Costing hundreds of dollars and bringing it illegally to this country to use it. It doesn't work, so why bother? You can use - there's polyprenyl immunostimulant, which is kind of popular, but remember, this has not even been proven to be an immunostimulant, number one, and number two, admitted even by everybody that it's only for cats with the milder forms of dry FIP and their only evidence is that it can prolong life about 100 days. At \$300 a week, that's okay, if you believe it works that's okay but anyway, this is what the treatment is so really, basically no treatment. This is what we're going to hopefully talk about today.



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Diagnostics

I'm going to go through this fast. There are just simple diagnostic tests that are non-definitive in that they don't say, "Yes, he has this virus 100% certain." There are tests that are very simple, like the complete blood count - just a complete blood count with serum proteins. Just looking at the fluid and analyzing it; what is the protein content of the fluid? What are the cells that are in that fluid? What types of cells are in that fluid? You can do all of that very simply. You don't need a lot of stuff and this is just a history on a cat. What are we going to look for? We're going to look for what is called the signalment. The signalment is the age, breed, origin, gender. The gender could be either male or female. Some say males more, I haven't seen it that way. It is definitely a disease that tends to occur in younger cats, you saw the figures, 4 to 29 months of age. By the way, interestingly, something else that is happening now is that we're seeing FIP in older, much older cats, aged cats. If you have an old cat in a household, this is what happens; we have two old cats in a household, we have three old cats, we may have four, and they slowly die, and you end up with one. You decide that that old cat that's 12 or 13 years old needs a buddy, you know, that's what he needs. So, what do you do? You go out and you get a kitten! Now, those poor old cats have been in that household, or that apartment or whatever, isolated, for years. You go out and get a kitten. Where do you get the kitten from? Nine times out of ten, you would get it from a rescue, because that's what you do, that's what your heart tells you to do. You go out and you get a kitten, you bring it in, and remember what I said, with time they lose their immunity and become susceptible to reinfection. Here this old cat is, you bring another one in, 60% chance or more that he's shedding in the feces. You've infected him, so here he is. Now his immune response system is not as good as it was when he was young, because he's old, and their immune systems are just like our immune systems, as we get older we start seeing all these interesting things happen.

Okay, so we're just asking for age, breed, origin, situation. We look for failure to thrive, especially if it occurs in younger cats. Poor hair coats. There's this cyclical fever that is antibiotic-unresponsive and that's so typical, and every cat that we get is on antibiotics, and I cannot figure out why. Why is every cat that we get with FIP on antibiotics? Yeah, they're on steroids, many of them, but there's also a mistaken belief that if you put an animal on steroids, you always need to put them on antibiotics. On the other hand, it's okay to put them on an immunostimulant and prednisolone at the same time, which doesn't make any sense either, because one's supposed to stimulate the immune system; one's supposed to take it down. Okay, then you're going to look for the typical signs, the eye signs, the neurologic signs, you're going to look for masses in lymph nodes, in the kidney, and so forth and so on, and that's really about all you need to do to make a diagnosis, that's about it and the fluid is very characteristic, as I'd said.

This is kind of a characteristic complete blood count picture of a cat with FIP, forget about these liver enzymes because they go all over the place, the albumin levels are low usually, the total protein is high. Why is the total protein high? It's because the globulin levels are high. We have, if you do what's called an A/G, albumin-globulin ratio, it's going to be very low, because the albumin's low, the globulin is high and so you look down at this, and you see, just like here, you see three principal signs that could go wrong with the other signs that you see from your physical history. Then you go to this one, and you look at the white cell count. White cell count can be high, low, or in between, but the important thing is many of these cats are anemic, so you can see they're anemic, their red cell values are below normal, and that's called the anemia of chronic disease. That means, if they have anemia, that means that they've been sick for longer than you think, so that's the anemia of chronic disease. Then if you look down here at the white cells, the main thing that you're going to see, especially with cats with wet FIP, not so much with dry FIP, notice that the lymphocyte counts are low. Low lymphocyte counts, and the lymphocyte is a cell that fights the infection, so it is an important cell in the immune system; it's low, and that's evidence that their immune system is being knocked down. A complete blood count is the simplest test that you can do. We always test for



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FeLV, because when FeLV existed as an important disease, it no longer does; when it existed, it is a potentiator of FIP, so when FeLV was rampant, it wasn't uncommon about 40% of our FIP cases were also FeLV-infected. FeLV infection is a potent immunosuppressant and allows the FIP virus to flare up. The antibody titers, especially in dry FIP, are often high, especially if they're done by the old classical immunofluorescent antibody test. There are other tests that people do that involve you having to take some sample of a tissue or the fluid and, basically, you're going to take those tissues or fluids and you're going to look for the presence of the virus, even the viral proteins, the specific FIP viral proteins or the RNA of the virus.

Here's a disease – this is a bladder wall, this is bladder muscle. This is the lining of the bladder, and it should only be one layer thick, and you can see, it's been totally infiltrated with edema fluid and these nests of inflammatory cells, which are mainly the macrophages that are containing virus. Now, if you were to do this test with immunofluorescence, you can see that lesion, that's that little foci of macrophages, and every macrophage is staining green, which means that it's full of viral antigen. Now, the problem with immunofluorescence is that this has to be fresh tissue, and you have to cut this with a microtome and freeze right away, so this isn't done very often, but it is really quite sensitive to take a tissue that has lesions in it, freeze it, and then do frozen sections and do this procedure, but the more common one is called immune-peroxidase. It often goes by immunohistochemistry. Here's the intestine, that's the intestinal villi right there and this is the wall of the intestine. This is the muscular layer of the intestine right here and this is that single layer that should be the serosal surface, a single membrane, it should only be one membrane thick, but you see it is also thickened and infiltrated by inflammatory cells and these little foci. In this case, the tissue has been fixed with formalin, which is the common way that we fix tissue, and this can be cut very easily, without frozen tissues and that, and so 99% of the immunohistochemistry is done with formalin-fixed tissue by this immunoperoxidase methodology, which is unfortunate, because it is less sensitive than the other, but it can be done, but it has to be done properly. If you look here at that little square where that little group of cells is, and everything that contains this red stain contains virus, so these are virus-infected macrophages. Now, these are definitive tests for the virus. Now, there are also what are called polymerase chain reaction tests, where you take these same tissues or fluids, then you analyze them for the presence of not the viral proteins but of the viral RNA, the genetic nuclear material. Supposedly, those are supposed to be very sensitive and very specific. The problem is that in the way the samples are taken and maybe they're not taken properly or good enough, but probably about 30% of the samples that are taken and tested by PCR come back negative, falsely negative, which can really give you problems, because how do you deal with a false negative when everything else is positive? How do you weigh this test compared to all the other tests? I'm telling you and a lot of veterinarians will tell you that this is the definitive test, but it isn't. The definitive tests are long before that. This is only confirmatory. If it's positive, there, you have your confirmation. If it's negative, all those other signs have to be taken into consideration.

Okay, let's go into treatment. This will be the last part, and so if there are any questions about FIP that anybody has, if you want to hold them to the end, or you want to ask questions because my students know, or knew, that if you don't slow me down, it'll go right past, and if the question's important to you then it'll be important to others as well.

Okay, so treatment. Firstly, we know that most of the cats will die of this disease, but I'm here to tell you that a lot of them can live a lot longer than you think, as you've already seen, so if you're the type of person, the minute you hear FIP, you euthanize them, then they have a lifespan of a few days. If you fight on and fight on and on and symptomatic treatment and all those things, some of these cats can live for weeks, months, and quite amazingly long periods of time, because cats are really tough individuals. Some of them can come into some sort of a balance with their disease, where they can kind of survive in their own way with it. Immunostimulants and immunosuppressants have no curative power, so prednisolone is only anti-inflammatory. That'll make them feel



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better, eat better, may alleviate their signs for a while, but it will not change the outcome. Immunostimulants, I don't believe in them, I'll tell you right now there are a number of them on the market. They're basically plant-based, or bacterial-based extracts. They're called biologics, they're not drugs, they're biologics. None of them have been tested in cats, in research cats, to show any immunostimulatory effect. They're just marketed, and I don't know how they got in, but they're marketed without any test results on research cats that show definitely that this compound, if given to cats, stimulates some aspect of their immune system. None of that is shown, but no matter what they have no curative powers. They may prolong life, if you believe some of them, they may prolong life, but it's only for a short period. So, what are we left with?

Vaccines would be the answer for this disease, obviously. There is a vaccine on the market for this. It is used by some people, forget it. The American (International) Feline Practitioners Association. Others will tell you that they don't recommend it, because I could go into details why it's not effective, but I'm telling you, it's just not, and so it's not something that you should waste your money on. There have been many, many people that have claimed to have developed vaccines that are effective against it and have developed drugs, mostly biologics, different compounds, cyclosporin A, blah blah blah. These things appear in the literature that claim that they can do something against FIP and, believe me, they don't do anything like what they say. The results are not repeatable. All of these vaccines in the past that have come out with this flurry of being great answers have turned out to be a complete bust. In fact, I went to a meeting and I listened to the head, the representative of all veterinary biologic companies in the world saying that, if somebody brought him today a vaccine for FIP, he'd tell them to go away. That tells you where it is, and I'm not saying it can't be vaccinated against, but we're in a situation that's very similar to tuberculosis and HIV. There are just some diseases you can't vaccinate for, so let's face it. Anyway, just as with HIV, with HIV time was lost, several years were lost because everybody had hopes for a vaccine and they did not put their effort in the antiviral drugs, and then what happened is people that were working on antiviral drugs, they were the ones that were starting to show some success. The vaccines started to drop out one at a time, but over about a 10-year period, so this was kind of a waste, because everybody thought, vaccines, vaccines, vaccines, we've got to prevent this. It's almost like the war on cancer. The first war on cancer was looking for viruses and all the money was spent on looking for viral causes of cancer, and now all these people that were working with carcinogens were saying, "We're not getting any money. What about me?" So, eventually, it switched around and so, anyway, I think it did slow us down on trying to do that.

Antiviral drugs

Now, what happened here is really quite simple. This is an RNA virus. Most of the viruses that we had antiviral drugs for are RNA viruses. RNA viruses contain many of the same genes, similar genes that do the same thing. Many of them have a reverse transcriptase gene that transcribes their RNA to DNA. Many of them have a protease gene that cuts their proteins into usable segments. This is typical of RNA viruses to have these similar genes, which means that all RNA viruses have similar targets. They have similar targets that you can attack them by, and so it was when I read a paper from Yunjeong Kim and her group at Kansas State University, great collaborators by the way, they were researching these protease inhibitors against this typical type of protease that you would find in RNA viruses, and they were doing most of the work on calicivirus as a model of norovirus, and you know what noroviruses are, because those are the bane of the cruise ships, right? The ones that gives everybody diarrhea. They published that they had a compound they thought would also work against coronavirus. I contacted them and said, "Okay, this is great research, love it. You have a drug. I have a model. I can test this drug. Let's work together. I know this disease from the research, the experimental aspect, and I know it from the clinical aspect as well, so let's work together." This is the outcome of this research, of the first drug we worked, it was a protease inhibitor, and that drug is called GC376. That trial is over so, basically, I can tell you that the results of that are going to be published in the Journal of Feline Medicine and Surgery, because that's the journal where it's most



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appropriate to be published, because that's where the audience is, and we're going to publish it so that everybody can have open access to it, so you can download it without any cost as well. Hopefully in several months that will happen. To make a long story short, we treated 23, 24 cats, but a couple of them died within a few hours of coming in because they were so sick, but out of the 20 cats that we treated, we saved seven, so there are seven cats out there that we saved. The rest of the 13, almost every one of them we made normal for a period of time, a few weeks, a couple of months or so. All those 13 relapsed with disease; most of them with neurologic disease, with brain infections, which we couldn't treat, because these drugs did not penetrate that barrier, the blood-brain barrier. Why do we have a blood-brain barrier, why do we have a placental barrier? The placental barrier is to save our progeny from getting killed by infections in the mother and that. The blood-brain barrier, so if we take in some toxins that it may give us a gut ache or do some other things, but it's not going to put us into a coma. If it puts us into a coma, we're going to be dead. I'm not talking about modern times, I'm talking about thousands of years ago when we evolved, or millions of years over which we evolved. We have this blood-brain barrier, which is great for keeping out things from getting to our brain, but it makes problems when you're trying to treat something that involves the brain. That's the outcome of that.

I want to just show you a schematic of a typical RNA virus, and this happens to be HIV, but it could be a coronavirus as well. This has to attach itself to the membrane or float around the brain or in the lumen of the bowel and get onto a cell and then basically it becomes fused with the membrane and then its internal RNA and all that is kind of instilled into the cytoplasm of the cell. Then this first step is called reverse transcription, and this is where the viral RNA is converted to DNA, and this is the first step in all RNA viruses, and so this can be inhibited by what are called nucleoside or non-nucleoside RT inhibitors, and so these are NRTs and NNRTs, this is what they're referred to. Lovingly, they're referred to as nukes, and these are the mainstays of HIV treatment, the nukes, the NRTs and NNRTs. They inhibit this conversion, so they inhibit the replication of the virus very early in the scheme of things. Now, if you go on, that DNA has to be converted to RNA. You can see these RNAs here, and that RNA gets converted to DNA, and this DNA then has to get converted to what's called messenger RNA, and it's this messenger RNA that produces the proteins that are going to help in your body functions and that. In a normal body, DNA is always transcribed to messenger RNA, and this is only seen with RNA viruses, this reverse transcription. Then, we're going to make protein, and then this protein all comes together in the surface along with copies of the viral RNA, and then we make new viruses, and so protease inhibitors work in -- RNA viruses, because they're very small, they don't have a lot of genes, and so instead of having a gene for every protein, they have often one large gene that makes a very large protein, and that protein is called a polyprotein. A polyprotein is made up of a whole bunch of individual proteins that are all linked together like sausages. Then there has to be some means for the virus to then cut that polyprotein into the individual proteins, so it has another gene called the protease gene that makes this protease. The protease - pro means protein, -ase means cut - cut proteins. The protease cuts these polyproteins into the individual proteins, which then allow the assembly of these new viruses, so protease inhibitors work late in the replication cycle. The nukes work early in the replication cycle, and so nukes and protease inhibitors are the mainstays of HIV treatment. The nukes are the main ones, and the protease inhibitors are thrown in there often to prevent resistance from occurring. HIV drug resistance occurs quite rapidly but, by having several drugs at different places in the replication strategy, if a virus mutates to become immune to one of these nukes, then it'll be still susceptible to the protease inhibitors down here. Using drugs in combination like that is not just for a synergistic effect or to increase and make them better, inhibiting the virus, it's also one of the main functions, but using several drugs together is to prevent these drug-resistant units from arising.

Now, in the case of hepatitis C virus, you remember there's been a great flurry of research in the last few years in clinical applications, whereas before you couldn't treat hepatitis C. Hepatitis C is a disease of people, the generation below me more. In hepatitis C, about 70% of the people exposed to hepatitis C get immune to it, 30% become chronically infected so they will get chronic infection in their liver cells, and this chronic infection in their



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liver cells is what causes their liver cancers and hepatitis and all that down the line. Basically, the first treatment against this was the combination of interferon, which is a drug that stimulates the immune system and a relatively poor antiviral called ribavirin. They use interferon and ribavirin together, this awful, painful treatment that is prolonged because interferon makes you sick. Basically, they had about a 70% cure rate with that. Now, what they've done is they've developed a battery of protease inhibitors, which they've used against hepatitis C, and with those they're getting 90%, 95% cure rates with those drugs, without all the side effects of the interferon. Protease inhibitors are very effective but you don't have to worry about resistance with hepatitis C so much because you're only treating for 8 to 20 weeks. Whereas with HIV you're treating forever, because the minute you stop treating it's going to come back up again so resistance is a bigger problem with HIV. That's why you would want to use combinations of drug. In the case of our disease, our disease is more like hepatitis C so we will not worry as much about resistance.

This is the paper that is coming out in in the Journal of Feline Medicine and Surgery which documents the first cures of cats with a protease inhibitor and so, as I said, we're going to make this freely available to everybody and, believe it or not, I had one reviewer who did not believe that I knew how to diagnose FIP, so that's the way life goes.

This is GC376.

It was made both at Kansas State University with colleagues at Wichita State University, and we did the animal studies at University of California. Flora is a product of that study. She's one of the seven survivors, and Smokey, so those are two of the seven survivors. Oakley – there are others in that mix as well. Some great cats. What can I say? This kind of breaks them down into what they were. We had some... They were mainly younger cats, but there were some older cats. Now, I have to tell you that you might say, "Well, 20 cats is not very much," but to tell you that, when we were pushing K State and Wichita all the time to make more drug, and they had to make it by old-fashioned chemistry. Now, those of you that know about old-fashioned chemistry know that you're sitting there in your laboratory with all sorts of glass and tubes and so forth all over the place, and so we were pushing them really hard to make enough drug, because when we did the laboratory experimental work, in only two weeks, we could cure cats with experimental infection in two weeks. We knew with natural infection it could be worse, but in natural infection, we learned that it required 12 weeks to even get near the cure. We were using drug up at much greater rate. What we learned is you don't need 100 cats or 200 cats. If you had 20 of the right cats, you can learn everything you need to know, if you do it right, and you learn everything from each one of them to the max. That's all you need, and so basically what we learned was that... They came from all over the country, by the way. We even had one cat, Flora, that came from Peru. We had cats from all over. I had people offer me \$200,000 if I would let their cat in the trial, and I said, "I'm not going to let your cat in the trial unless it's the right cat and the right owner." We had one, Smokey, where the owner was accepted and then couldn't pay for the trip to Davis. We paid for everything else, but they couldn't pay for the trip to Davis and follow-up blood tests that we wanted, and that's the one that Peter adopted so kindly and took over and was a success story. Some people might say, "Well, Peter was given some special privilege in that." No, he wasn't. Peter did a special thing for another person that was accepted in the trial that couldn't come. We had different forms, we had a number that had dry or wet FIP, and what do we mean by dry? It means that they had it localized in the lymph nodes and some tissues and then, at the end, it just went wet, suspected dry or wet, and then a whole bunch of them, we couldn't find any evidence that they had dry before that, and then a few that were just pure dry FIP.

I can just make a long story short and say with protease inhibitors, the most successful treated were the young cats with wet FIP. The kittens less than 18 weeks of age with wet FIP, no signs of dry FIP preceding that or anything, those were the ones that were the most successful. The ones that were the least successful, the ones that most apt to



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developed neurologic disease or recurrence of non-responsive disease were the older cats, and I don't mean aged, I mean cats who were maybe 1 year or 1½, but miracle of miracles, we had one cat that was six years old, his name was Krathos and he belonged to a veterinarian in southern California, Krathos all of a sudden just started developing fever and weight loss. This huge cat, he was a big cat, just started wasting away and had fevers and everything and so he was worked up and he had the typical blood picture, the high globulins, all that stuff, the anemia of chronic disease. He had, on palpation and on ultrasound, he had an enlarged lymph node, a mesentery lymph node. His disease was localized to the mesenteric lymph nodes and so, basically, what they did is they biopsied those. They took some of that and looked at it, and yes, it looked compatible to the type of inflammation, and so we took that, and it had a high antibody titer to the FIP virus.

We took him in and we treated him and his story is here. When we treated him – at first, we didn't know how long to treat these cats so we're only treating him for two weeks. If you look, this is just his body weight. We're following his body weight, so we follow different parameters; we follow their temperatures, we follow their body weights, we follow their CBCs, their anemias and their total proteins. We monitor all of that to see where we're going and so he did feel a lot better, he started eating and gaining weight. Then when we stopped the treatment, his fever came back, relapsed. We said okay, treat him again. We treated him again for several weeks and again he responded but then relapsed again, but he didn't have neurologic disease. Once they develop neurologic disease we knew couldn't treat him anymore, but he was just going back to the same old thing again, so we treated him again. Then he bumps up for a longer period of time, he bumps up to this, and all of this time he's in remission. Then, all of a sudden, relapse again. Then we've treated him for 12 weeks because by that time we had figured out that maybe 12 weeks is the time. We then treated him for 12 weeks, and here he is eating and gaining more weight. He's now been in remission for two-thirds of the year; many, many months, and no obvious sign of relapse. We see the same thing in other cats that successfully were treated over 12 weeks, that there was a progressive gain of size and weight. There are a lot of simple criteria that you can use to monitor these cats. You don't have – there's nothing complicated or magical about monitoring your success on these cats. This just shows the viral levels in the fluid, the ascites fluid before we started the drugs.

This is the virus levels for these different cats. You see, within a few days, these virus levels dropped really fast and, by the way, as one might predict, these are the cats that did better than this cat over here because he responded much better to the drug. The virus levels went down pretty fast.

One thing that we had is one of our cats had eye disease and we treated him and within a week, his eyes were completely cured. We said that's great but it didn't make sense, because the eye has a barrier too; it's part of the brain, so it has that same barrier that the brain has. Basically, to make a long story short his relapse was only transient and in a few weeks, he developed neurologic disease and died. What we found out was, we knew ahead of time not to treat cats with neurologic disease, but what we learned with protease inhibitor was that if they had eye disease they would respond dramatically, we had others that responded too, but they would always get brain disease so if they have eye disease, don't even start with that treatment.

What were the side effects?

This is one of our cats, Oakley and what the drug does is it inhibits the development of the permanent teeth so if you are treating... Remember I said the most successful cats were under 18 weeks. Their full eruption of their permanent teeth is about six months or so, five or six months so basically, what happened is that the drug would inhibit the formation of the permanent teeth so that they would be smaller than normal and slower than normal in erupting. What would happen is that you would have these retained teeth, where you would have retained like you can see a retained baby tooth here. This is a permanent tooth, this molar, but this fourth premolar is nothing but a



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baby tooth and you can see the other little teeth on the inside. You see those little bumps right there? Those are the permanent teeth coming in, and they were so small that they weren't able to push out the permanent teeth. Now this is Oakley now – excuse me, this isn't Oakley. I'm trying to think of this one's name, but anyway. It is the same one you saw in the last one, but you can see now that there are some teeth missing, obviously, but there's nothing wrong with this dentition. In other words, they have their canines, which is the important thing and they have their incisors and they have this one large premolar, but they're missing some of their other teeth but that's the main problem that we saw with the younger cats. This particular drug can cause a lot of staining, and it does cause, if you get some of the drug into the skin itself, it causes a blister, it causes skin reactions and you can get some scarring subcutaneously. Most of these, probably three fourths of these cats have these little areas where the skin had died and regenerated and then, obviously, no hair because the new skin doesn't have follicles in it and the scar doesn't. Several of them had subcutaneous areas, little round pea-like nodules which we are following closely, and we will remove one of them. Basically, as a precaution because you know about the fibrosarcomas, the injection induced fibrosarcomas in cats so we want to nip that one in the bud and if those subcutaneous scars are too large we'll take them out.

Okay, here is Flora.

Some pictures of her. We had fun with Walt and we had fun with Flora. She is a great cat.

This is Smokey.

I tried to find the best picture. This is the best picture of Smokey. He loves his owner. He follows him all around, sleeps with him. They have a special bond with each other, and I do think he's one of the most unique-colored cats I've ever run across and Smokey is such an appropriate name for him.

Okay, so let's talk about – the last thing I want to talk about is what we're working on now. I was head of the virology unit at the primate center during the AIDS epidemic, during part of the AIDS problem. We were working on the SIV - simian immunodeficiency virus - model of HIV, and SIV in rhesus macaques was just like HIV and the viruses are closely related. There was a little company down in the Bay Area called Gilead that was just starting and up, and they had this compound called PNPA, this group of compounds that they were trying to prove and this was at the time that everybody was interested in vaccines and not as interested in drugs. They were trying to prove this drug, so we helped test that drug in primates in the SIV model and found that it was very effective in SIV and it was very effective in the neonatal monkeys when we were studying neonatal transmission and that. We like to think that we did them a great service because we provided them with valuable information that saved this class of drugs. I don't need to tell you that Gilead Sciences now covers seven blocks in Foster City with dozens of high-rise buildings that you can't see the top of, hardly. I wish I would have bought stocks in the company. I knew them when... but I didn't. Anyway they published an article a year or so ago where they were working on drugs for MERS and SARS, which are the Middle Eastern Respiratory Syndrome and the Sudden Acute Respiratory Syndrome, which are coronavirus infections in humans, highly fatal, not humanized totally yet and then they are working on Ebola. Now, Ebola is an interesting story, because Ebola is an RNA virus and showing how mutations are important. Before this last outbreak that covered huge parts of West Africa, Ebola was only a little disease that occurred in little outbreaks out in villages, way out in the sticks. Someone, a native out there would get a hold of a monkey or some species that carried the Ebola virus, and then he'd eat it, and then they would have spread it by intimate contact to a group of people. The virus wasn't fully humanized, so all these little outbreaks usually kind of fizzled out they kind of scared the hell out of us. There were a few cases, very fatal, and that type of thing but they never went anywhere. Basically, what happened in this last outbreak is the virus humanized. It went through another mutation and humanized, and by being humanized what it means is that now it can be transmitted human to



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human, and that's always a great fear with avian influenza that it's finally going to humanize itself, and then we would have a real problem on our hands. These things are – and remember, chimpanzees were the host of HIV, and it didn't disease in chimpanzees, it was only when it got into humans and humanized in humans and then it spread from there. Gilead was working on, basically, drugs for Ebola and they published a study that showed that they had a nucleoside inhibitor that would protect monkeys against experimental Ebola infection. I said "That's kind of neat because, although Ebola isn't a coronavirus, it's an RNA virus, and it may be typical." I got ahold of the co-founder of Gilead, Norbert Bischofberger. Really neat guy, Swiss. Anyway, I got a hold of him and said, "Norbert, I see you've got some interesting research to do – do you could share some of your compounds," because these companies make these compounds by the thousands. I told the group last night that this isn't going out into the Amazon and collecting thousands and tens of thousands of different drugs and then going to the laboratory and seeing if any of them have effect on cancer or bacteria or anything. This is all science now – you can actually make the proteins of a virus in three dimensions. You can determine where the receptors are that you can target. You can actually find out how you can make drugs that will target those receptors. This is all science now. This is not hunting for things out there as much. Anyway, I knew that when they make all these compounds they made different iterations slightly takes because what they're trying to do is find the compound that fits in one of these little places the right way so they make all these different iterations. I said, "Just give me your iterations – give me the ones that you don't want, the ones that you're not going to patent or you're not going to market," and so he went to one of his virology groups and said "There's somebody here that wants to do some work in animals." Most of the group did not want to work on animals, they had no interest in animals, because that's not their thing. But there was one guy, just one guy, that said "You know, that sounds interesting. Let's do it, just for the sake of cats." That is all it took, and so they gave us a group of compounds. We had a grant from the Winn Foundation, Brian Murphy and myself, to screen those compounds. We screened those compounds and we found one of them that just was amazing in tissue culture at inhibiting virus and very safe. We took it into animal trials, and we did what's called a pharmacokinetic study to determine how much of the drug it would take to sustain blood levels over a 24-hour period that would effectively inhibit the virus. We found that one dose, once a day, at a relatively low level of drug, would inhibit 100% of the virus in the blood. Then that was enough to take it into a field trial.

There are a lot of people that are upset at me. I don't do Facebook, because I have two groups, I'm like Trump, I think. I have two groups; those that hate me and those that love me. Basically, the ones that hate me hate me for different reasons but one of them is that I used experimental cats. These are purpose-bred for research, they're not. We do find homes for everyone that does not need to be put down. We did that work and it worked quite well so then we decided to do the field trial. You cannot do a field trial under our rules at the university. You just can't take a client's cat and say "I've got this stuff that I want to poke in your cat and see if it works or not." That's not easily done, so you've got to give them evidence that whatever you're going to do has some efficacy or some reasonable chance of working and we also don't want to get into this thing of having placebo controls. By the way, you do not have to have placebo controls. There's nothing in the rulebook about placebo. That's old-fashioned. You have to have controls, but they don't have to be placebo controls. They can be natural controls and that type of thing.

We provided our institutional animal use and care committee with the data showing, "Yes, we have a drug that has a high chance of being highly effective against this disease in nature. Will you okay it?" We did some bouncing back and forth about how we were going to do this, and so forth and so on. It is not easy. It took us six months to get permission from the institution committees to use it and now we're in the midst of using it. I can say that we're 24 cats into this particular trial. This is a lot different, working with Gilead, and I'm not saying anything against K State because they have worked their little tails off. You know, when you've got this company that you just say, "Okay, we're going to take it into field trials. I want 50 grams of this stuff, two weeks' time" and then they put an order to their chemists and say 60 grams, and here it comes, a vial of 60 grams. That makes life a lot easier. In



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those 24 cats now, the furthest one now is probably around 11 weeks. You saw Luna, some of you today, and Luna is out nine weeks.

Basically, I just wanted to tell you about this class of drugs, these are nucleotide inhibitors. They inhibit this step of RNA to DNA and they do it in an interesting way. The drugs prevent the DNA copy from being made. You have the RNA copy here and then the RNA copy is taken by this reverse transcriptase enzyme and it causes a DNA copy to be made. This copy is being made by adding nucleosides one at a time so you can imagine this would be a big ladder going up over here. What happens is that this compound, it actually gets in there and it puts phosphorus moieties onto these nucleosides that are going to come in here and make this chain and, actually, it gums up the works... It's like putting a rock in a gear, a bunch of gears. Basically, it just takes it out and, as I said, this is a very early stage of replication so the earlier in replication you can stop this, the better off you are.

Here is Luna. She was a pistol, boy! She's a fighter and those of you that saw her here. Actually, she's the third hybrid cat that we've had to treat for FIP in this scheme and they're all doing well out there. I thought that she would be a problem, because one thing is that she does not like to be restrained. She's so active it's unbelievable. She does not like to be restrained. We had to really wrestle with her to get her injections and get her blood but Deb says that she just sneaks up there and she puts a dish of food down there and sneaks up and grabs some skin and pokes the needle in, and that's it, so that's a lot easier than most people have had.

To make a long story short, of these 24 cats that we have out there, or that we started treatment on; three we couldn't treat because they had neurologic disease right from the onset. We know that right off the bat, because within 24 to 48 hours, these cats' fevers drop just like that to normal. Within three days, their activity and their appetites all come back again. Now, if that fever does not come down within that first five to seven days that we treat them, we know we've got a problem on our hands. So far, strangely, because we would have predicted we would have had more problems with neurologic disease that we had, the only problems we've had with neurologic disease are the three that had neurologic disease to start with. All the rest of them are still out there, and we ship them drug every two to three weeks and they're still out there. The longest one is 11 weeks, which is getting near the time period of the 12 weeks. During this same period, I can tell you, of the 20 cats that we had, we lost 13 with the protease inhibitor. We haven't lost any that have gotten past that first stage yet with this particular drug. Now, the advantage of this drug, I mentioned this, is that if you have a company like Gilead behind you it is really interesting because Gilead, at first, they said, "Whatever you find, if you find this is of value to you then you can have it, you can have the rights to it and use it." They still will give us the rights but their enthusiasm for the drug has gone way up. As we gain more and more positive results, their enthusiasm goes up. Now they're all talking about it, "We're going to find the company to market this with, and we're going to help you," and so forth and so on, which is great. I'm not going to be dealing with all the things that they're dealing with in the protease inhibitors where they have to try to find companies and find this and that. It's really important to have a company that has dealings with the FDA. Now, remember, all drugs have to be approved by the FDA. It's not the USDA. USDA is only animal vaccines and biologics. All drugs - FDA. That's a whole different ballpark, so when you have people that know about all these procedures, it makes it a lot easier.

Am I optimistic? As optimistic as my Scandinavian nature will allow me, yeah! [laughter] I think we will have our answers, but, if we don't, what we've done and this is what Yunjeong does, I want to say this with our work with the protease inhibitor. It may be that this isn't the drug that's going end up being wherewithal to end all or that EVO will be the drug, but what we've shown is that there will be a drug. We just have to get into those cupboards and those drawers of these companies that have all these compounds that are being made for various viruses, and remember that these are all RNA viruses, they're not anything special beyond that. We can use the data from one RNA virus to fight another RNA virus and that. Anyway, that's all I have to say. I think that somebody said that



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FIP is the worst possible enemy. I look at it this way, some people look at their enemies as enemies. I look at enemies as being learning experiences. They're worthy enemies, if I can use this term. I think what I've learned in my 50 years is FIP is a worthy enemy. Okay. That's it. [Applause]

Okay, let's do questions.

[Dr. Pedersen]

So, I've just been told that the question and answer period comes after some business. So, what's your question? If you allow me to answer it, because you've already asked it.

[Question]

So your saying that EVO 984 drug is crossing the blood brain barrier?

We don't have evidence for that yet. In the monkey experiment with the Ebola, they did look at the levels in the brain and it did get in the brain at low levels, just as GC376 got into the brain at low levels. Most of these drugs do not get into the blood/brain barrier, drugs that are like this. My suspicion is, and then basically on our failure on those three cats that had neurologic disease, that it may get in enough for those cats that have early disease maybe to prevent brain infection but not enough to cure it once it's there.

[Steve Dale]

Okay, so we're going to - I promise! We're going to answer your questions. That's sort of the highlight, and I know that Dr. Pedersen enjoys actually taking questions. How many here had a cat who has succumbed to FIP? I wish I had a camera to take that picture. That's like 90% of the room. How many of you were astounded if that is a safe word, by what we just heard? How many of you, five years ago, and that's not long ago, in cat years or our years. How many of you, five years ago, would have thought we'd be hearing this today? There are one, two extraordinarily optimistic people in the room. I will go to Vegas with both of you people! [laughter]

Here is what we're going to do. That very first slide, the very first slide that we saw was Winn Feline Foundation welcomes the 39th Annual Symposium or something to that effect. Did you see that slide? The very first, that was taken the other day when we all went to see the Chicago White Sox play. Now, the Chicago White Sox, how many are from Chicago here? Okay, that's 10%, 15% of you, or in the Chicago area. The Chicago White Sox said, you know, this organization sounds really good. We don't understand exactly what you do, but it sounds really good and important. Here's what they gave me. They gave me plastic. They also gave me a baseball jersey worn by Robin Ventura, who was a third baseman for the Chicago White Sox for many years and their manager for many years. (Dr. Pedersen: "That's my size!") Well, we shall see! So, here's what I'm going to quickly do, and I will not do it for anything less than \$500. I'm going to try to live auction this off. If it doesn't work here tonight, I will find another way to raise money for the Bria Fund. This specifically goes for FIP studies for the Winn Feline Foundation Bria Fund. I'm not an auctioneer, but I'm going to give it a go.

Anyone here for \$500 for this jersey? Yes, we have a taker right here, it's Peter. I'm not stopping though. How about \$550? You think I'm stopping there? You're a White Sox fan? - I never knew that. \$550? Going once. It is not signed. I thought it was going to be, to tell you the truth, but it is not signed. I do have a letter of verification, and I have the lady's name at the Chicago White Sox if that doesn't work, and you're welcome to call her. Jo Simmons is her name, actually. \$550? Come on, going once. Peter, this may not fit you, so I... Dr. Pedersen clearly says this will fit him. (I didn't say, I just said I could get into it!) [laughter] \$550? Going once, going



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twice. Peter, you are now a Chicago White Sox fan! I'll bring it down to you in a moment, and that does go to the Bria Fund. Yes, sure.

We met Susan Gingrich who began the Bria Fund, a board member of the Winn Feline Foundation. Now, there are other board members in the room, and I will ask my colleagues on the board to stand up and receive attention for all the work they do with the Winn Feline Foundation. [applause]

I also have two other people in the room that I'd like to acknowledge. One is... There are a lot of veterinarians in the room, but there is only one Dr. Shelly Rubin. If you're from the Chicago area, you know this man. Dr. Rubin.

Now, have any of you seen a concert, ever? No one has seen a concert in this room? Of course! Oh, by the way, here's what I've learned. Have any of you been to Carnegie Hall? Yeah, I know you guys have been to Carnegie Hall. I've never met anyone personally who's performed at Carnegie Hall. I've gotten really close, because isn't it your daughter that did or a Pedersen? – and Gerry herself? Ah, that is what I thought, Joan! So, Gerry, Mrs. Pedersen, she came here to deal with us low-lifes after performing at Carnegie Hall! Isn't that nice?

Here's what I was saying though. Have any of you seen a concert, and you'd said yes, you've seen a concert. Have any of you seen a concert where the performers happened to be only feline? You think I'm kidding about that. The Acro-Cats! Some of you actually know what I'm talking about. Samantha Martin is right up here! Samantha. She actually goes around the country with performing cats demonstrating that cats can learn, too. It's not only about the D-word - those dogs - it is also about cats. The Winn Feline Foundation is also about the person who steers us, the person who leads us and that is our executive director and, by the way, they have a farm, and they make the greatest jelly in the world. Maybe she'll let you buy some and the funds go to the Winn Feline Foundation, too. Everything goes to the Winn Feline Foundation. Dr. Vicki Thayer.

[Vicki Thayer]

Certainly, one of the things I've learned from being around our honored MC is that cats are the Rodney Dangerfields of pets. They're not the first class citizens; they're oftentimes not the second. They're really the third or fourth, and so it's really important, I think, to emphasize the evidence-based medicine, the research that we need to do, we need to promote for cats. I've been very involved in organized veterinary medicine, like the AVMA House of Delegates and working alongside Dr. Shelly Rubin in the House of Delegates, and they may establish priorities when people talk about how important evidence-based medicine is, or research is but, if you're going to prioritize it, it falls way down the list, and that's not a good thing oftentimes for cats because, as I said, they're third or fourth class citizens. I went to one of the most recent veterinary internist meetings, where where the top research gets reported, and out of oral and poster research abstract, 21%, 22% were related to cats and 75% canine. Now, that's a shame in my book, so that's why oftentimes my mantra is to say "Felines first," because so much of the research is being done for dogs and cats are way down the list when that happens. Also, again, we want to raise awareness, and one of the things that we're utilizing, as we mentioned, this is coming up on our 50th anniversary. We're rolling out early, like right now, we're at the CFA annual, and next year, of course, 2018 is the 50th anniversary. One of the ways we're raising awareness is to bring out weekly Winn Wins but we're also going to go into a campaign that we call Cures for Cats day that's going to be on October 21st, and that's at the American Association of Feline Practitioners meeting in Denver, on October 21st. We've been working very hard to do our 50th anniversary book, and that 50th anniversary book is going to come out on that day. It also will have a very unique cover by everyone's favorite artist, Jamie Perry, so this is going to be something very special, and we're really wanting everybody to try and think about Cures for Cats day. Let's raise the awareness of cats. I hope as you go home, please consider your folders, your little green bag with the homeless kitten, take it home, adopt it. There's a great pen in there that is related to our 50th anniversary, and a treat - it's chocolate - Kit Kat bars for you



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guys. Really, the last thing is, we can talk about Winn's impact. It's been, I think, pretty doggone high, but the impact really comes from you, and you've helped reach the point over the years, supporting Winn, helping to support and trying to find those answers for FIP to where we're here seeing Luna and hearing about Smoky so we want to say thank you. Thank you very much. [applause]

[Steve Dale]

Okay, now we can do what many of you have come to do and that is to take questions for Dr. Pedersen, so I'll invite Dr. Pedersen back up here. I will ask you to stand up when you ask your question. We don't have a... Oh, we do have a handheld mic somewhere in here, actually. If this works like I'm hoping it will, then I will run around to you but, in the meantime, we'll start, so just raise your hand, stand up and ask your questions loud and clear.

[Question]

I was not clear on the nucleotide inhibitor, are you using that on wet, dry or both?

[Niels Pedersen]

We are using it on everything, except neurologic disease and ocular.

[Steve Dale]

Next question. I'll go to you first.

[Sue Robbins]

Hi, Dr. Pedersen, Sue Robbins. I have a couple of questions, actually. The first one is what is the timeline that you anticipate for your trial on the EVO984?

[Niels Pedersen]

Let me say how these trials go. We'll probably close the trial for these first 20-some cats, and then we'll digest what happens, you know, over the next 12 weeks or how long it takes. Then, depending on the results, we'll open that up again for maybe a larger, more select trial, but we're not going to treat hundreds of cats. This first trial is basically very much like what we did with the protease inhibitor. It just kind of gives people enough information. For instance, the GC376 is already being synthesized in China and sold, and people are buying it, but you have to be very careful about what you're buying, because is it chemically pure? I don't know. Then, I think the important thing is I know that one group in China is using it, but they don't know how long to treat it, they don't know the potential side effects, they don't know which cats are going to be the most favorable ones to treat, which ones not to, but I couldn't feed them that information, because, first of all, I don't want to feed anything that's not legal. The second thing is that the results are now in the hands of the Journal of Feline Medicine and Surgery and it's now their property until it's published and made freely accessible. I can't just give out everything, they want an article, right? They want an original article.

[Sue Robbins]

I think the bottom line question that I'm inquiring about is, assuming that it goes well, what kind of a timeline do you envision for the drugs to be on the market and available for our general... Is that a feasible question at this time? Five years? Ten years?

[Niels Pedersen]

The favorite answer when somebody in a mouse model cures cancer and then the question the reporter always asks is "How many years is it going to take for this knowledge to be used in humans?" The answer is always five years, and of course it never is five years it never comes at all most of the time. Anyway, the answer is that it's not going



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to be five years but it's not going to be one year. It's going to be, again, it really depends on how well this trial ends up going. I mean, you know, it could be we get out there and all these cats relapse; all these cats that are so doing marvelously out there, just like we had with the protease inhibitor. They're doing so well and all of a sudden, two thirds or so then just drop out. We don't know if that's going to happen, so I can't answer your question. The question, I suppose, would be - can you find a company? Let's say the worst case scenario is that you could only cure one third of the cats, and only these cats with a certain type of disease and that type of thing. Is there a company that's willing to take that on and spend the money to take it through the FDA approval processes and market it.

[Sue Robbins]

And make it reasonably affordable but...

[Niels Pedersen]

Yeah, that's right. You know, it costs \$200,000 to treat a hepatitis C patient - \$100,000 to \$200,000, and so that's not possible. I do know from what we've done that people are willing to spend several thousand dollars to treat their cat. I know there's a market there and what's happening now is that there are new companies. I'll give you an example. For instance, Gilead, they were working on compounds for human lymphoma, going to treat lymphoma in humans. They had some compounds that, for some reason, the people at CSU - Colorado State University - tested some of these compounds in dogs with lymphoma. Lymphoma is a very common tumor, if you get a golden retriever, you know about it, or others. Basically, they had favorable results with this in dogs. It wasn't a compound that they would want to market in humans, because it probably didn't give much advantage over compounds that were already existing for humans. So, basically, the group at CSU, I guess this is no secret, they started a separate company that's basically, it's an independent company. It's just how life is now, some entrepreneurial person just starts another company using those compounds, marketing those compounds. I think it's called VetDC or something like that. Basically, those are their first two compounds. If you think about Gilead with PNPA, that's where they started, so this little company starts with that. Now, that and Gilead is feeding into that, you understand, because those are Gilead compounds marketed for veterinary use. Gilead doesn't have a veterinary wing to it and so you can see that happen, and there are several other little veterinary companies that are in the same boat. Gilead may be working with us going to a company like that and saying here, I know you're only interested in cancer right now, but here are some other things that you might be interested in as a startup. I also know at least one huge veterinary company is also very interested in antivirals for FIP. I think that part is going to be taken care of. I think there's interest out there. I think it's just a matter of what kind of information we can feed to convince one of these companies to take it on, and then I think it's going to also depend on the FDA. In the FDA, the rules are changing a lot, they have a licensing procedure for major drugs for minor species and they also have drugs for highly fatal diseases which are the rare diseases that are highly fatal. They have different licensing pathways, like for an antidepressant or an NSAID or something that could be really cumbersome and take a long time. For some of these specialized things it could be a lot faster, so you're asking me a lot of questions that really depend on what happens and, ultimately, it's going to depend on how much good information we can give to these people that want to take it on. In other words, if we can show them that we have drug that will cure a lot of these cats, I think there are going to be takers, and they go pretty fast depending on how FDA reads it. Now, if this is a human drug, a common human drug, for let's say depression, antidepressants or something; they would definitely want our results to be repeated at three or four other places. They wouldn't just accept our results. They would want other trials done. So, I don't know there. So, they could look at this and say, "Hey, we're not going to accept this unless it's done at Cornell or it's done at NC State or somewhere else, at Utrecht or wherever."

[Sue Robbins]

Thank you.



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[Steve Dale]

How many of you, out of curiosity, have given even \$1 to the Bria Fund? Well, first of all, thank you very much, and you should know you've made a difference. Dr. Pedersen and the board talked yesterday, and one of the things he told the board is something he just said now... Probably, for starters, we're going to need to replicate this with another investigator somewhere else at another facility. That, of course, takes money and that's what the Bria Fund is for. So, if you think, okay, the Bria Fund is over, it's done its job, that is, unfortunately, I wish that were the case, but it is not the case. We absolutely still do need your help.

[Dr. Shelly Rubin]

Dr. Pedersen, we realize that only a very small percentage of the coronavirus affects a small percentage of cats. Have there been genetic markers for breeders that are having an issue in their cattery, let's say? Have they identified genetic markers? And, if so, is it useful, and if not, do you recommend...

[Niels Pedersen]

Are you talking about genetic markers that are host markers for susceptibility or resistance?

[Dr. Shelly Rubin]

Right, exactly.

[Niels Pedersen]

Yeah, we did a study with Birman cats in Denmark. They have a high incidence of FIP, and we did what's called a genome-wide scan, and we looked for genetic differences in cats that were over a certain age that had not had any history of FIP in their pedigree with a group of cats that had FIP, and so what we found were definite areas in the genome of cats that were susceptible that had FIP that were not present in the cats that didn't have that in their line. They were multiple what we call hits, but they were in genes that made sense that would influence the immune system and that kind of thing. So, what we found is that none of them reached what we called the height of significance. When you do these genome-wide analyses, there are very stringent rules for what is significant association. Ours were below significant, but they were very good. They were below, and that was because we didn't have enough numbers. We need more numbers. Then what we did was, we looked at our random-bred cats, and our random-bred cats in our colony, over the years, every time we infected a group of cats with FIP, 20% of them are resistant, so from this certain breeding colony where we get our cats, 20% of the cats had a natural resistance to FIP and 80% die or go on to treatment. Basically, what we decided to say is well, let's look at those 20 that are resistant compared to the 80. We did the same thing, we did the genome-wide (GWAS) in those but then we came across a whole different set of peaks than we saw in the Birman cats, and some of them didn't make much sense as to how they would relate, so then we decided, okay, so let's assume that these cats that are immune, this 20%, that it's a simple genetic, one genetic change that is making these cats resistant, but wouldn't that be nice if we could do that? If we could find that polymorphism and this simple gene, we could just introduce it into all these breeds of cats and they'd all be resistant. This is the study I got into a lot of trouble with, because we used a lot of cats, and so basically, what we decided is, we're going to breed resistant cats to resistant cats, and then we're going to test whether their kittens are more resistant. Now, if this was a simple genetic trait, their kittens should be more resistant than the parents. When we did that, the kittens were more susceptible. Instead of 20% being resistant, there was only 10% resistant, so we took that 10% and bred them to each other, and they were all susceptible, so what that says is and that's why I discussed it in the board meeting; this is a complex polygenic trait that involves probably hundreds of what are called risk factors. In our evolution, we have accumulated positive risk factors and negative risk factors, and we all have them in different proportions, and that's why if we get into a big disease, we're not all going to die. Even with HIV there's 20% survivors that are resistant, or 16% or whatever it is.



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Basically, what that says, I used this like a pinball machine, I said it's like playing pinball. You throw the ball up at the top and then you've got all these little flippers and bumpers and everything like this, and the more flippers and bumpers you have, if you've got some skill, you can keep the ball up there. Eventually, you're going to lose, it's going to get down there. Then as you inbreed, which is what we're doing, normal inbreeding for simple genetic traits, that's what you've got to do, you've got to concentrate that genetic trait because you're artificially selecting for it, so it's like coat colors, your setting a coat color or fixating it. With these polygenic traits, what happens is that you're losing flippers. As you inbreed, you have less and less flippers and bumpers and so when you throw the ball up there, it goes straight down to the bottom. That's the nature, and positive risk factors that are in one group of cats, or negative risk factors, may be totally different than the positive or negative risk factors in another group of cats. That's why in Europe there is this one test offered by this Polish thing, you know, if you're heterozygous for a certain polymorphism in a certain gene that constitutes that your cats are more resistant, so the breeders get that test, and then it's like me, I had my DNA ancestry, because I had some idea that I might be something different than I am. It came back a little red circle right around Denmark, Norway, and Sweden. There I am! A little red spot right there. Basically, you know, they are looking at their evidence for what limited study they had, but then the strange thing about this was that, to have resistance you had to be heterozygous, you had to have only one copy, not two. Then I said, "How do you breed for heterozygous in a population?" That's outbreeding, to increase resistance for a heterozygote, which is called heterozygote advantage. The more heterozygote genes you have, the more of your genes are heterozygote, you have a heterozygote advantage. I was saying in a way, that's kind of an interesting test, because the breeders actually were using it the right way, they'll be breeding for more and more heterozygous, which is good. They thought this was some simple thing, where you just test your cats and then use it in some simple breeding thing, but it's so complex, that I wouldn't even begin to suggest it.

[Steve Dale]

Alright, let's try for one more very quick, because we're going to be thrown out of the room. One more very quick question and very quick answer. Who will be the lucky winner of the last question here?

None? Okay, I will ask a question. I've got the microphone, I can do that! Dr. Pedersen, you've done a whole lot in your career. There isn't a feline disease that doesn't have your touch on it in some way, often significant way. Of all the things you've done, for all the years, human or cat years, you've been doing what you do, is this the most significant thing?

Not counting the Carnegie Hall appearance by your wife. [laughter]

[Niels Pedersen]

That probably is the most, but anyway. I could be corny and say my kids and different things like that. It's impossible to say, because okay, with feline leukemia, my colleague, Hans Lutz of Switzerland, and myself, we did the monoclonal and made the FeLV diagnostic tests that are used all over the world, which caused complete cure. It's not the vaccine that's prevented or wiped out FeLV as a disease, it's the testing and finding out which are the carriers and not using them, getting them out of the population. When we did that, we pushed FeLV back out into nature, where it's only like 1%, 2% infection, and most cats that are exposed are older and they get over it anyway. We pushed FeLV back into nature, where it's not a significant disease, whereas in the 60s, 70s, and parts of the 80s, it was in all of our catteries, it was in all of the cat households, it was all over the place. I take that as a major accomplishment. Then when we look at the discovery of FIV, saying that you've already got two retroviruses in cats, you can't have a third one. I said, well, why not, if we have two, we could have three. I felt we wrapped that up very fast because we determined that it was transmitted by aggression, meaning through male aggression, that it was a disease mainly of outdoor cats that infected cats, just like in HIV-infected people. We're not infectious to other cats as long as they didn't engage in risk behavior and so keeping them indoors takes away that risk behavior



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and so that made possible. Then we also said that FIV, they can live a long time with it, there's no need to put them down right away. You treat them well and get them back on good nutrition, so that's important. Then with FIP, we get to this point, and so you're asking me to say which of my kids I love the most, you know? [laughter]

[Steve Dale]

Right, that's the greatest line to end it on. You always end with a punchline - I've learned that. Please help me thank Dr. Pedersen. [applause]

You were the first to hear much of the information, almost all the information...

The End