Arizona Wildlife Podcast

Transcript: Episode 12 – Amphibian Fungal Disease

(Please note: this podcast was recorded live from a public presentation. It was not a rehearsed speech. This transcript attempts to capture the dialogue as it was spoken. At times when the speech was difficult to hear or understand, a good effort was made. These rare cases are noted in the text.)

The content for this episode came from a public speaking engagement at the Arizona Game and Fish Headquarters in Phoenix on December 4, 2008. The topic of the talk was the Chiricahua leopard frog.

Listen as Mike Sredl, reptile and amphibian program coordinator for the Arizona Game and Fish Department, describes a deadly disease affecting many amphibians across Arizona and the world. Following the live recording, the audio was edited and Eric Proctor, a Department educator, interjected appropriate questions to link the content.

ERIC:
So, Mike, I've been hearing a lot about this fungus that may be responsible for amphibian die-offs around the world. Can you tell us a little more about this?

MIKE:
Actually, it was only identified as a…as a disease in 1998. People were seeing mysterious die-offs. In fact, I, in the early '90's, was doing some population studies and saw mysterious die-offs and population crashes. And I was fortunate enough, uh, at one of my mark-recapture sites – it's a kind of a study where you do, where you estimate populations – that…that one of my mark-recapture sites a big die-off happened and I happened to preserve all of those animals informally. And it turns out that this pathogen, you can do histology or tissue sectioning. And you can find the…

PARTICIPANT:
The lesions.

MIKE:
Yep, exactly.

PARTICIPANT:
Are they…do they attack the brain and neuro system? Or…

MIKE:
No, it's…it's, uh, rather odd because it's a very superficial infection. It's only the top two layers of skin. The…essentially the, uh…

PARTICIPANT:
The epidermal, subcutaneous…

MIKE:
Epidermal, yes. And…and it's not even subcutaneous, really, um. It's the stratam corneum and the stratam granulosum, which are the top two layers in amphibian skin. So, it'd be like a person dying of

zits. You would think that something that was so superficial couldn't be so lethal. But you have to imagine that amphibian skin is…is really, kind of more like an organ. I mean they…they respirate through their skin. They take in ion – ionic exchange. Unlike our skin, which is…is fairly, you know, a fairly strong barrier to the outside world. Amphibian skin is not that way.

PARTICIPANT:
What do you guys think is the major contributing factor? Is it environmental cause?

MIKE:
To? To kicking off these die-offs? Or…

PARTICIPANT:
To starting the fungal infection?

MIKE:
Right. Boy, a lot of people have…in fact, uh, my major advisor at ASU has been investigating this. And people are looking for those triggers.

It appears that cold weather plays into it. You know, you get a cold snap and we…we will generally see die-offs around those cold…in fact just today I was over in Cave Creek, uh, swabbing because we can…now there's a genetic…a PCR like the O.J. Simpson trial. When…when they did the genetics for…for O.J. Simpson. And there's a PCR test that you can do to detect the fungal pathogen. And so you just do skin scrapes. You can do it on live frogs. It's a non-destructive sampling, real easy. Well they…they had, uh, several dead frogs there that I also, uh, swabbed their skin.

So, we don't know what the triggers are. Cool weather appears to be somewhat of a trigger. But people have looked at the amphibian immune response and they're not seeing any depressed, classical T-cell, which is a…

(INAUDIBLE COMMENTS FROM PARTICIPANTS)

MIKE:
…part of the immune system. They don't appear to make antibodies. They don't really appear to de…detect the pathogen, uh, very much at all. So it's…it's a puzzle. And so people have speculated, "Well, it's a disruption of an organ…the skin." It's…maybe they're secreting some kind of toxin. Immuno-compromised individuals are susceptible. They really haven't seen much in the way of immune response yet. The…it's still a rath…rather mysterious disease. But people are starting to get a little better handle on some of the triggers.

PARTICIPANT:
Is this the same disease that affects all the amphibians like salamanders, newts, all that stuff? Is it the same kind of fungal infection?

MIKE:
It is…it is, yeah. And it's affected, uh, pretty much, um, all of the major groups of amphibians, yeah.

PARTICIPANT:
And it spreads outside the southwest, all over the world?

MIKE:
It's…it's, yep. Uh-huh. The…as I said you could do it retrospectively, you could look in histology. We did a study with, uh, museum specimens and we found it as far back as 1972 in some museum frogs in Arizona…southeastern Arizona.

The…the oldest specimens in the entire world are from South Africa in 19…1938 or something like that. They were from *Xenopus* which is, uh, uh, kind of the white lab rat of the amphibian world. It's used in a lot of studies of…of behavior and…and…and neurons and they actually use…widely use *Xenopus* as a pregnancy test. So it…*Xenopus* was shipped all over the world including the U.S. So, the hypothesis is that…is sort of the "Out of Africa" hypothesis that *Xenopus* and the biomedical trade shipped those frogs all over the world. The pathogen somehow escaped and spread to the wilds of the areas where *Xenopus* was shipped. And that's how the disease became so recently introduced to many of the world's amphibians.

But then there are genetic data that aren't really consistent. There's little to no divergence in the strains of this path…pathogen which you would expect that supports the "Recency" hypothesis. You know, because they're all more or less the same, closely-related strains. But there is not the structure across the landscape that you would expect that would support the "Out of Africa" hypothesis. So it's…it's a little muddled and conflicting like that.

PARTICIPANT:
The frogs that you treat and release, are they immune or they're just clean when you let them go and then…?

MIKE:
Yeah, yeah. There are some medications that are thought to…to have a fairly long lasting effect and…and perhaps even, and I've heard the person who is investigating this use the word immunity, but it's not sort of the classical…

PARTICIPANT:
Right.

MIKE:
…definition…of…of immunity as an innate resistance…

PARTICIPANT:
Right.

MIKE:
…either due to the immune system response. It's not that kind of immunity. I think it's just a long lasting resistance.

The work on this pathogen has exploded over the past ten years, which it's really only been known for about a ten year period.

PARTICIPANT:
Are they doing gene studies?

MIKE:
Lots of genetics, yep. People…people are studying it's…it's…essentially they're looking at the host, pathogen, and the environment. So, you know: the triggers, uh, in the environment, the genetics of the pathogen, the ecology of the pathogen, uh, the ecology of the frogs.

Some frogs, uh, can…can bask. If they get hot enough. The pathogen doesn't survive above 30 or so degrees Centigrade. So there are treatments where you can heat up frogs or…or some frogs appear to climb up higher and bask longer if they're infected. So there…there seems to be a behavioral response on the part of some species of amphibians.

If it weren't…weren't so tragic, it's really a fascinating sort of story because the pathogen has a complex life cycle. So it's…it's got an infective stage that is in the skin. And then there's a dispersal stage which is kind of like a…a little sperm – flagellated organism – that goes out and finds another frog and colonizes. It only affects adult amphibians in the skin because it is a decomposer of keratin. Adult amphibians have keratin in the skin, tadpoles don't. The only place that tadpoles have keratin is in their beaks. They've got little mouth parts that are like kind of teeth that, uh…and then they've got a little beak that…that they munch their food. So, the tadpoles can get infected but only in the mouth. You've got a host and a pathogen both with complex life cycles where the…the food, so to speak, of the pathogen is in different parts of the body of the individual and it's…it's rather…like I said, if it wasn't so tragic, it's a…it's a very cool host-pathogen story.

PARTICIPANT:
Whenever they are raised captively, how do they get exposed to it? Is it just inherent in the breeding population and it gets transferred to the eggs?

MIKE:
It probably is, yeah. There are probably reservoirs of infected individuals out in the wild, uh, and then there's also the possibility of within collections, like at a zoo, cross contamination. In fact, that probably happened at the, uh, National Zoo when they first described the disease.

This podcast is brought to you by the Environmental Education section of the Arizona Game and Fish Department and is made possible through the Heritage Fund and the Arizona Lottery. For supplemental information and activities for this podcast, please visit www.azgfd.gov/focuswild and click on the podcasts button.

Thank you.