Adenovirus in Bearded Dragons (*Pogona vitticeps*)

**ABSTRACT**

Little is known about the pathogenesis of adenovirus in the bearded dragon. Like other viral diseases in reptiles, adenoviral infection is difficult to diagnose ante mortem (before death). A better understanding of the disease process and its detection is necessary if we are to treat the animals affected with the virus and prevent this virus from spreading throughout the captive population. This discussion will examine the clinical presentation of the affected patient and my work on determining viral presence ante mortem, as well as understanding its pathogenesis. Detecting the presence of the virus is important, but preventing its spread is essential. By understanding the pathogenesis we can better accomplish this. Originally, it was believed that the virus was passed vertically. More recently, some have proposed that the fecal-oral route or an intermediate host passes on the virus. The goal of my work is to help better define this process.

It is sometimes difficult to differentiate virally infected animals from animals suffering from calcium deficiency or coccidiosis. Fecal examination can rule out coccidiosis, and a thorough review of husbandry can rule out calcium deficiency. Diagnosis becomes complicated when multiple disorders are present at the same time. While there are no specific signs of adenovirus infection, a good history, physical exam and fecal can yield some strong suspicions. Detection of viral presence in the patient while alive would be a useful tool for the veterinarian.

Ante mortem and postmortem histopathology and electron microscopy were performed on tissues taken from three symptomatic clutches of dragons including the mother. Tissue samples were collected on random animals from ages one-day-old to one-year-old and the mother. Electron microscopy was also performed on fresh stool samples from this population. A review of these findings will be presented.

**INTRODUCTION**

Since their introduction into the USA pet trade in the early 80's, the Inland Bearded Dragon (*Pogona vitticeps*) has grown in popularity and is now estimated to outnumber the green iguana. It is the most popular reptile species kept as a pet in the United States and it is estimated that over 500,000 captive bred babies are produced each year in the USA. Adenovirus is a relatively new disease. The first reported case in the USA inland bearded dragon (*Pogona vitticeps*) was in 1996. There have been only 3 reported cases of adenovirus in bearded dragons. The first was by Julian, A. & Durham, and P. 1990 in a female *Amphibolurus barbatus* at the Auckland Zoological Park in Australia. The second was by Frye, F. et. al. 1994 in Rankin’s Dragons *Pogona henrylawsoni* at a private breeder in Illinois, USA. The third was Jacobson, E. et. al. 1996 in *Pogona vitticeps* with animals from Iowa and California USA mentioned. My first case was in 1997 in a clutch of one month old *Pogona vitticeps* from Illinois USA. Since that time there have been many cases diagnosed all over the country. An exponential increase in the cases in the past three years has been noted. Because to this point we have only been able to detect this disease after death.
using postmortem pathology, little is known about the pathogenesis of the adenovirus in the bearded dragon. My work has focused on detecting the virus in the live animal and getting a better understanding of its pathogenesis.

**DISCUSSION**

Accurate diagnosis is the key to containing this disease’s spread throughout the entire population of Bearded Dragons. History, physical exam, histopathology, fecal stool exam, and fecal stool EM negative staining are the tools I use to diagnose the presence of adenovirus.

**History and Physical Exam** are essential in identifying potentially affected animals. This disease presents itself differently in different age groups. The age groups can be divided into three ranges, 0 to 4 weeks, 5 weeks to 12 weeks, and greater than 13 weeks. In all groups it is essential to determine if proper husbandry is utilized. It is important to determine if proper UVB lighting, day and night temperature, diet, and supplementation are being provided. In cases where there are husbandry shortfalls, they should be corrected and the animals reassessed in 4 weeks. Parasitism should be eliminated by running a fecal floatation on all suspect animals. Clutches that incubate normally but have poor hatchability or have more than 10% death before 4 weeks should be suspect. Affected animals that are between 5 weeks and 12 weeks may appear week, have diarrhea, poor appetite, failure to thrive, have seizures and death. Dragons over 12 weeks that grow at a slower rate than expected while appearing normal in all other ways should be suspect. If two or more clutch-mates are kept together and one is much smaller, suspicion should be high. Also, some animals can carry the adenovirus and have no clinical signs. These are the hardest to identify. If any animals are determined to have the virus, their parents should be suspected to be carriers and proper tests for viral presence should be done.

**Histopathology and Fecal Stool Exam** can be run either ante mortem or postmortem. The virus has been found in all types of tissue but is consistently found only the liver and small intestine. The problem with these tissues is that they are not always positive in affected animals. An ante mortem liver biopsy can be collected safely by an experienced reptile veterinarian. However, negative liver biopsies can occur in affected animals. Post mortem, liver and small intestine samples should be collected at a minimum, but all other tissues should also be taken. Intestinal parasitism with Pinworms, Coccidia or bacterial overgrowth can mimic the viral disease. In some cases, both intestinal parasitism and virus are present. It is essential the run a fecal stool exam on all suspected cases, and if parasitism is detected administer proper treatment. Parasitized animals should be then be re-evaluated in 4 weeks for proper growth. If growth is still retarded, further tests should be run.

**Fecal EM Negative Staining** is where I have concentrated my work. Feces are placed in a special fixative and sent to a university that has an electron microscope where the virus particles can be detected in the sample. In 100% of affected animals, this test has been positive and 100% of unaffected samples have tested negative. After a number of trials using known virally infected feces, it has been determined that the stool sample must be put in the fixative within 90 minutes of being voided. After that time, the test is less accurate. If it is not possible to submit the sample within that time, the sample can be frozen and later thawed and fixed. Known positive samples that have been frozen for up to 46 days have had 100% positive results. The importance of this work is that this test can be run ante mortem and without the risk of anesthesia or surgery and is much less costly.

Little is known about the pathogenesis of adenovirus in the bearded dragon. Possible modes of transmission are vertical, fecal-oral, and through an intermediate host. Vertical transmission is when the virus is passed directly from the parents to the offspring. In order to test this method, two virally positive symptomatic normal size adults were bred together. The eggs were laid in a 50% play sand and 50% coconut fiber mixture sterilized by autoclave and mixed with distilled water. The mother’s ventral surface was washed daily with Lemon Quat (a viracidal disinfectant) for the last week prior to laying the eggs. The eggs where
then incubated in the sterilized coconut fiber moistened with distilled water and incubated at 84 degrees Fahrenheit until hatching. The hatchlings were then put into a new 10 gallon glass tank that was disinfected with the Lemon Quat and lined with sterilized surgical drapes. They were fed Rep-Cal juvenile bearded dragon pellets moistened with distilled water. At 4 weeks old freshly voided stool was submitted for fecal EM negative staining. All of the hatchlings were positive for adenovirus. These results show that the virus is transmitted from mother to offspring. It does not determine if the virus came from the ovaries or from stool as the eggs passed through the cloaca—(the common area that stool, urine and eggs pass through before leaving the body). Surgical removal of the eggs at the time the positive female indicates she is ready to lay should determine the point of infection. If the hatchlings are negative, the virus is acquired from the stool in the cloaca. If they are positive, transmission is from the ovary.

In order to fully understand the pathogenesis of the adenovirus, we must test the other possible modes of transmission. Future work will test the fecal-oral mode by force-feeding virally infected stool to negative animals and running fecal EM negative staining. To test the intermediate host hypothesis, virally infected stool will be fed to crickets and in turn, the crickets will be fed to negative animals which will ultimately be tested with fecal EM negative staining for diagnosis. When these modes are defined, we will have a complete understanding of the pathogenesis of the virus. When we understand the modes of transmission, we can define a plan for prevention.

CONCLUSION

Until we fully understand the pathogenesis of the adenovirus, we should take all the precautions possible to prevent its introduction into our collections or spread to others. For this reason, I recommend following these precautionary steps. Fecal EM test ALL breeder animals. This will allow for clean breedings and production of virally free offspring. Quarantine ALL new introductions until fecal EM tests are run. This will prevent introduction of the virus into your collection. Feed only commercially processed foods. Until it is determined that crickets are not a vector, play it safe. Feeding commercial foods also prevents Bearded Dragons from contracting Pinworms and Coccidia. Finally, always maintain proper disinfection and hygiene. Animals that are proven virally free or are the offspring of them should be sold at a premium—this will more than offset the added cost of testing.

The potential for this disease to devastate your collection is high. If it continues to spread throughout the industry, it could ruin the market. For these reasons, ethical considerations should always be considered if an animal is diagnosed with adenovirus. The main question is should any animal suspected of or diagnosed with Adenoviral infection be sold or bred? At this time, this author believes the answer is no. Since many of the asymptomatic carriers can live a long and natural life, they can be kept separate. Precautions must be taken to prevent the accidental spread of the virus if one chooses to keep positive animals.