

Adenovirus infection in bearded dragons Fact sheet May 2019

Introductory statement

Adenoviral hepatitis is a common cause of neonatal and juvenile mortality in captive bearded dragons (*Pogona* spp.) in the USA. Although adenoviral infection has been reported in both captive and free-living bearded dragons in Australia, there is little information on the prevalence of disease. Disease associated with adenovirus has only been reported in captive bearded dragons. Both free-living reptiles and captive populations are at risk from this virus in Australia.

Aetiology

Adenoviruses are medium-sized (80–110 nm), non-enveloped viruses containing a double stranded DNA genome ^[1]. Adenoviral infections have been recorded from a large number of reptile species including snakes, dragons, skinks, geckos, chameleons, monitors, crocodiles and tortoises ^[2]. Adenoviruses are generally regarded as being species specific and the majority of infections in bearded dragons have been caused by Agamid adenovirus-1 (AgAdv-1), as confirmed by PCR ^[1, 3-7]. However, there is one report of lizard atadenovirus infection in a western bearded dragon (*Pogona minor minor*), while AgAdv-1 has been found in a central netted dragon (*Ctenophorus nuchalis*), a species in the same subfamily as bearded dragons ^[8]. Given the high prevalence of AgAdv-1 in bearded dragons overseas it seems likely that some, if not all, of the adenovirus infections in bearded dragons reported before the advent of PCR were due to AgAdv-1 virus ^[9-12].

Natural hosts

Adenovirus infection has been reported predominantly in agamids of the *Pogona* genus. These include the eastern bearded dragon (*Pogona barbata*), central bearded dragon (*P. vitticeps*), Downs bearded dragon (*P. henrylawsoni*) and western bearded dragon. A case was also reported in a central netted dragon, which is in a different genus ^[5, 8-11]. AgAdv-1 infection has been confirmed by PCR in the central bearded dragon and central netted dragons, *Pogona henrylawsoni*, in the USA (Frye et al 1994). Adenoviral infection has also been reported in a savannah monitor (*Varanus exanthematicus*) that died without showing premonitory signs. In the USA PCR studies also confirmed the presence of adenovirus in a blue-tongued skink (*Tiliqua scincoides scincoides*).

World distribution

Adenovirus first emerged as a cause of hepatitis in an illegally imported, confiscated eastern bearded dragon in New Zealand in the early 1980s ^[10] and is now recognised as a common cause of

sudden death in captive bearded dragons in the United States ^[2, 3, 7, 12] and Europe ^[1, 4, 13]. Cases of adenovirus infection in USA have been reported in central bearded dragons and Downs bearded dragons, while all the European cases occurred in central bearded dragons.

Occurrences in Australia

Adenoviral infection is reported in both captive and free-living bearded dragons in Australia. Published reports in captive dragons have identified adenovirus infections predominantly in central bearded dragons, but also in eastern bearded dragons, a western bearded dragon and a central netted dragon ^[5, 6, 8]. Adenoviruses were found in two of four study populations during a small survey of free-ranging Australian bearded dragons in 2014-2017. Agamid adenovirus-1 was detected by PCR in an eastern bearded dragon from south-east Qld and a number of inland bearded dragons from western NSW. In addition, *Lizard atadenovirus A* (formerly helodermatid adenovirus 2) was detected by PCR in one of the inland bearded dragons from very small numbers of inland bearded bearded dragons from central Australia and western bearded dragons from Western Australia ^[14].

Epidemiology

Transmission is by the faecal/oral route ^[5]. There is anecdotal evidence of vertical transmission, but this has not been confirmed ^[15]. One study failed to detect evidence of the virus by PCR in the ovaries of adenovirus positive females ^[3].

Affected dragons are generally less than 12 weeks old ^[5], but disease can occasionally occur in older animals ^[1, 15]. Concurrent dependovirus (a virus in the parvovirus family that cannot replicate without the assistance of a helper virus) and coccidial infections have also been diagnosed ^[11, 12]. There appears to be no sex-linked predisposition.

The virus can be carried asymptomatically. A UK study of clinically healthy captive bearded dragons found a viral prevalence, based on PCR of cloacal swabs, of 40% in the 13–18 month age group and an 8% prevalence in animals over 24 months of age ^[13]. It is likely that bearded dragons are the natural hosts for both Agamid adenovirus-1 and lizard atadenovirus A ^[14]

Clinical signs

Clinical signs are often non-specific, including loss of appetite, weight loss, diarrhoea and weakness ^[5, 11]. Neurological signs, such as head tilt, opisthotonos, circling and limb paresis may be observed ^[5, 12]. Sudden death is a common occurrence ^[4, 5, 8].

Two separate outbreaks occurred in captive colonies of eastern bearded dragons. The only premonitory sign reported in the first case was haemorrhagic diarrhoea (Australian Registry of Wildlife Health [ARWH] case report 2756/1). The second case involved hatchlings from the same clutch that presented with severe dehydration and watery faeces and died soon after (ARWH case reports 5316/1, 5322/1). Another unrelated lizard was submitted that died after presenting with abdominal distension (ARWH case number 5858/1).

Diagnosis

Diagnosis is by signalment, histology and PCR. Oral and cloacal swabs or whole blood can be tested by PCR. A broadly reactive PCR in concert with a specific PCR for the most common genotype is recommended. Oral-cloacal swabs may be more useful than whole blood for detection although whole blood PCR may be diagnostic for viraemic, clinically diseased animals ^[5, 14]

Clinical pathology

Clinical pathology of adenoviral infection has not been described.

Pathology

Gross pathology is often unremarkable. The liver may be mottled, pale or reddish tan in colour, and friable. Histologically, acute diffuse hepatic necrosis is usually present (ARWH case reports 5316/1, 5858/1). Hepatocytes and cholangiolar cells may contain large amphophilic intranuclear inclusion bodies (ARWH case report 5316/1). Intranuclear inclusion bodies may also be found within the epithelial cells of the gastrointestinal tract, kidneys and exocrine pancreas (ARWH case report 5316/1) ^[1, 5, 16].

Differential diagnosis

- Dehydration
- Weakness secondary to suboptimal husbandry
- Hypocalcaemia
- Concurrent dependovirus and coccidial infection have been observed in neonatal bearded dragons infected with adenovirus ^[11, 12].

Laboratory diagnostic specimens and procedures

Procedures for specimen collection should follow those presented by Rose 2007 [17]. A full range of tissue samples should be taken. Neonates and hatchlings may be submitted whole (coelomic cavity opened) in 10% neutral buffered formalin for histopathological examination. Oral-cloacal swabs or whole blood can be submitted for PCR testing on live animals.

Laboratory testing includes histopathology and electron microscopy. Agamid adenovirus 1 and panadenovirus PCRs are available through Tim Hyndman (<u>t.hyndman@murdoch.edu.au</u>) at the School of Veterinary and Life Sciences, Murdoch University, WA, Australia.

Treatment, prevention and control

There is no treatment available. Affected animals should be culled from captive breeding populations. In contact dragons should be tested by PCR^[6]. All new lizards should be quarantined.

Adenoviruses are resistant to inactivation when outside a host and can remain infectious for long periods in soil, substrate, water and contaminated faeces. They can be inactivated by treatment for more than one hour with formalin, aldehydes or iodophors ^[5].

Surveillance and management

Wildlife Health Australia administers Australia's general wildlife health surveillance system, in partnership with government and non-government agencies. Wildlife health data is collected into a national database, the electronic Wildlife Health Information System (eWHIS). Information is reported by a variety of sources including government agencies, zoo based wildlife hospitals, sentinel veterinary clinics, universities, wildlife rehabilitators, and a range of other organisations and individuals. Targeted surveillance data is also collected by WHA. See the WHA website for more information <u>https://wildlifehealthaustralia.com.au/Our-Work/Surveillance</u> and <u>https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/eWHIS-Wildlife-Health-Information-System</u>.

There are a small number of cases of AgAdv-1 infection in free living lizards in the National Wildlife Health Surveillance (eWHIS) database. We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at <u>admin@wildlifehealthaustralia.com.au</u>.

Research

Adenoviral infections in bearded dragons are yet to be fully characterised in Australian outbreaks. Surveys to determine the prevalence of adenovirus in populations of free-living reptiles in Australia are required.

Human health implications

None.

Conclusions

Agamid adenovirus-1 is a common pathogen of neonate captive bearded dragons in the USA. Its presence has been detected in captive collections and in wild bearded dragons in Australia. Given the popularity of the *Pogona* genus as a pet species it is important to monitor the presence of this virus both in captive and free- living populations.

Acknowledgments

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Wildlife Health Australia recognises the Traditional Custodians of Country throughout Australia. We respectfully acknowledge Aboriginal and Torres Strait Islander peoples' continuing connection to land, sea, wildlife and community. We pay our respects to them and their cultures, and to their Elders past and present.

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