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INCIDENCE OF LYMPHOCYTOSIS AND LYMPHOID LEUKEMIAS IN BEARDED DRAGONS (POGONA VITTICEPS) AT A PRIVATE DIAGNOSTIC LABORATORY

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or this 6-month period of 2012, a regional diagnostic laboratory in Chicago, Illinois received 31 separate hematology sample submissions from bearded dragons (*Pogona vitticeps*) sent in from several public and private reptile collections and veterinary practices. Of these samples, there were 5 that exhibited moderate to marked circulating lymphocytosis on the complete blood count and differential. This indicates a presumed incidence of 16% of all hematology submissions in this reptile species over this period. This was of concern for a potential over-representation of lymphocytosis in this species, and possibly an underlying alternative etiopathogenesis in addition to spontaneous neoplastic transformation. Two of these 5 individuals were determined to be most likely lymphoid leukemias and 3 were likely transient reactive lymphocytic responses to non-neoplastic unidentified conditions based upon progression or lack thereof. Other criterion considered was a worsening clinical course, minimal response to therapy, or euthanasia due to disease complications. In addition, 2 cases had follow up CBC's and one principle case was followed closely and monitored with serial hematology examinations, as well as other ancillary of tests to attempt to characterize the lymphoid population in this animal. A sequence of DNA was found in this animal's blood which was similar in arrangement and character to the sequence for Feline Leukemia virus (FeLV) and the genome identified for Bovine Leukemia virus (BLV) as well. This sequence could not be verified due to the lack of a normal genomic sequence for Pogona vitticep established or identified for this species.



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Table 1: 5 cases of moderate to marked lymphocytosis in bearded dragons

	Date of sampling	Animal data	Lympho-cyte count / ul	Cell morphology / additional finding	Tentative diagnosis	Outcome
Case 1	March 10, 2013	5 year old female	20,064	Marked toxic heterophilia, and sepsis evident. Increased lysed cells and count may have been higher	Secondary reactive lymphocytosis related to infection and sepsis	Animal died a short time later
Case 2	May 7, 2012	11 month old, sex unknown	15,480	A few reactive forms	Unknown -possibly transient reactive lymphocytosis	No further submission from this animal.
Case 3	April 23, 2012	2 year old, sex unknown	24,112	Cells appeared small/intermediate and somewhat reactive (larger, darker basophilic cytoplasm with open coarse chromatin, and sometimes indented nuclear forms).		
	June 19, 2012		32,419	Progressively increasing lymphocytosis –and similar morphology.	Potential chronic lymphocytic leukemia (CLL)	Lost to follow up
Case 4	July 16, 2012	4 year old, female	13,968		Unknown -possibly transient reactive lymphocytosis	No follow up submissions.
Case 5	September 7, 2012	6.5 year old, sex unknown; the animal appeared to have minimal clinical signs or illness	117,888	50-60% of the lymphoid cells were large lymphoblasts. Concurrent heteropenia. No anemia or thrombo- cytopenia.	Acute lymphoblastic leukemia. (ALL)	
	September 21, 2012		184,230	Developing anemia – non-regenerative with continued heteropenia and thrombocytopenia now seen as well. Similar lymphocyte morphology.		
	October 01, 2012		457,920	Progressive anemia – non-regen- erative with similar lymphocyte morphology evident.		
	October 17, 2012		244,880	Stabilizing anemia – non-regenerative with similar lymphocyte morphology; although many lysed, fragmented and apoptotic remnant cells present. Note: Likely another 100,000 cells could be added to the lymphocyte count if these were countable.		
	November 05, 2012		672,980	Likely another 28,336 cells could be added to the lymphocyte count however these were unclassified cells based on severe pleomorphism. Persistent non-regenerative anemia.	Persistent fulminating ALL	Clinical condition had worsened significant- ly with anorexia and severe progressive ataxia. Elected eutha- nasia on November 6, 2012

Biography

Dr Rand Wilson has joined leading veterinary diagnostic laboratory Finn Pathologists in the newly created role of Head of Clinical Pathology. Dr Wilson received a BSc in Wildlife Biology in 1984 and a Doctorate of Veterinary Medicine in 1988 from Colorado State University. He spent the next 20 years practising small animal, exotic and avian medicine and surgery and undertaking PhD studies in clinical pathology and infectious disease. He

became a member of the Royal College of Veterinary Surgeons in 2008 and achieved Board Certification in Clinical Pathology in 2008 from the American College of Veterinary Pathologists. Arriving in the UK in 2008, he gained experience in the veterinary labs sector before taking up his new role at Finn Pathologists, one of the UK's leading veterinary diagnostics laboratories in Harleston, Norfolk.

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