

1           Successful management of Heinz body hemolytic anemia associated with leek (*Allium*  
2                                   *ampeloprasum*) ingestion in a South American coati (*Nasua nasua*)

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20   Running title: Heinz body anemia in a South American coati

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1 Abstract

2 *Objective* - To describe the diagnosis, management and outcome of Heinz body hemolytic anemia in  
3 a South American coati (*Nasua nasua*) secondary to suspected leek (*Allium ampeloprasum*) toxicosis.

4 *Case summary* – A South American coati presented with Heinz body hemolytic anemia following  
5 addition of leeks to its diet for two to five days prior to initial presentation. Administration of a whole  
6 blood transfusion from an animal of the same species (conspecific) and supportive care resulted in  
7 immediate improvement in clinical signs. Normal behavior fully returned within six days of  
8 transfusion. Hematological evidence of anemia resolved by four weeks and there were no significant  
9 features of oxidative injury present by eight weeks following initial presentation.

10 *New information provided* – This is the first reported case of Heinz body hemolytic anemia, suspected  
11 leek toxicosis and administration of a blood transfusion in this species.

12

13 Key words

14 Zoo animal, *Allium* spp. toxicosis, blood transfusion

15

16 Abbreviation list

17 CPDA = Citrate-phosphate-dextrose-adenine

18 IM = intramuscularly

19 IV = intravenous

20 RI = reference intervals

## 21 Introduction

22           Heinz body hemolytic anemia is a rare but potentially severe condition in domestic and wild  
23 animals.<sup>1-5</sup> It is caused by the formation of denatured hemoglobin aggregates (Heinz bodies) within  
24 erythrocytes following exposure to an oxidant and subsequent erythrocyte lysis.<sup>6</sup> Oxidants include  
25 certain drugs, chemicals, heavy metals and plants, including *Allium* spp. such as garlic, leeks and  
26 onions.<sup>6</sup> Clinical signs may comprise anorexia, vomiting, diarrhea, lethargy, pale or icteric mucous  
27 membranes, hemoglobinuria and death.<sup>1-5</sup> Heinz bodies are identified on blood smears as rounded  
28 structures which are pale pink if Wright's stain is used, or dark blue with new methylene blue stain,  
29 within the erythrocyte cytoplasm, protruding from the cell membrane, or as free bodies in the smear  
30 background.<sup>6</sup> Other oxidative changes to erythrocytes include eccentrocytosis, pyknocytosis, and  
31 ghost cells.<sup>6</sup> Treatment involves oxidant removal, if possible, and supportive care.<sup>5</sup> Blood transfusion  
32 is indicated if clinical signs of anemia are significant.<sup>5,7</sup>

33           The aim of this report is to describe a case of Heinz body hemolytic anemia in a South  
34 American coati (*Nasua nasua*) associated with leek ingestion, managed with a whole blood  
35 transfusion and supportive care. To the authors' knowledge this is the first reported case of Heinz  
36 body hemolytic anemia, suspected leek toxicity and blood transfusion in this species.

## 37 Case Description

38           A 3 year 4 month old, 4.1 kg, male, South American coati (*Nasua nasua*) presented for a post-  
39 operative check 24 hours after partial digit amputation due to a phalangeal fracture. Clinical  
40 examination at the time of surgery had revealed dorsolateral displacement of digit four of the right  
41 forelimb, with purulent discharge at the nail bed, and moderately pale mucous membranes. Surgery  
42 and anesthesia had been unremarkable. A blood sample had been collected for hematology and  
43 biochemistry analysis and full body radiographs showed no abnormalities other than the phalangeal  
44 fracture. Meloxicam<sup>a</sup> (0.3 mg/kg, subcutaneously), buprenorphine<sup>b</sup> (0.01 mg/kg, intramuscularly

45 (IM)) and amoxicillin-clavulanate<sup>c</sup> (15 mg/kg, subcutaneously) had been administered intra-  
46 operatively and meloxicam<sup>d</sup> (0.1 mg/kg orally, once every 24 hours) and clindamycin<sup>e</sup> (11 mg/kg,  
47 orally, once every 24 hours) dispensed to continue from 24 hours post-operatively. At the post-  
48 operative check, the coati was slightly quiet and had regurgitated. Soaked pellets were offered 3-4  
49 hours later, which was followed by vomiting. The coati became progressively quieter and 3-4 hours  
50 later presented lethargic, dyspneic and tachypneic, in a sitting position with the head lowered in the  
51 corner of its enclosure. Blood results from the previous day became available and were compared to  
52 global reference intervals (RI) derived from serum or plasma samples collected from between 15 and  
53 100 zoo-housed South American coatis which were recorded with a normal health status at the time  
54 of blood sampling.<sup>8</sup> Hematology revealed marked anemia (PCV 0.110 L/L (11.0%); RI 0.277 – 0.508  
55 L/L (27.7 – 50.8%)) with moderately hemolyzed plasma (Table 1). On blood smear examination  
56 (Modified Wright's stain), numerous erythrocytes contained Heinz bodies, which were typically large  
57 (1 - 2  $\mu$ m), protruding, and often multiple per cell (Figure 1); brilliant cresyl blue staining  
58 demonstrated Heinz bodies in 100 % of erythrocytes, with up to 15 per cell (Figure 2). Ghost cells  
59 and eccentrocytes were present in moderate numbers, often with attached Heinz bodies (Figure 1).  
60 No polychromasia was noted. Complete blood count performed using a hematology analyzer<sup>f</sup>  
61 revealed a mildly increased mean corpuscular hemoglobin concentration (487 g/L (48.7 g/dL); RI  
62 282 – 391 g/L (28.2 - 39.1 g/dL)). Mild neutrophilia ( $18.95 \times 10^9$  cells/L ( $18.95 \times 10^3$  cells/ $\mu$ L); RI  
63  $1.796 - 14.715 \times 10^9$  cells/L ( $1.796 - 14.715 \times 10^3$  cells/ $\mu$ L)) was observed, although the neutrophil  
64 count was similar to values reported in wild South American coatis with a low parasite load and may  
65 be within normal limits.<sup>9</sup> Moderate numbers of neutrophils displayed mild cytoplasmic foaminess,  
66 occasional basophilia, and low numbers of Döhle bodies (Figure 1). Moderate numbers of reactive  
67 lymphocytes were present. Biochemistry performed on heparinized plasma using a biochemistry  
68 analyzer<sup>g</sup> revealed mildly increased bilirubin (18.0  $\mu$ mol/L (1.05 mg/dL); RI 0.70 - 12.0  $\mu$ mol/L (0.00  
69 – 0.70 mg/dL)) and urea (11.4 mmol/L (31.9 mg/dL); RI 1.90 - 9.30 mmol/L (5.40 – 26.0 mg/dL)).

70 Glutamate dehydrogenase was also elevated compared to RI in other domestic carnivores (56.0 U/L;  
71 dog RI < 6.0 U/L; cat RI 0.0-0.4 U/L; ferret RI 0.0-2.5 U/L).<sup>10-12</sup>

72 A thorough husbandry review with keepers revealed that between five and ten grams of leeks  
73 (*Allium ampeloprasum*) had been added to the diet of this coati and one other animal of the same  
74 species (conspecific) which shared its enclosure up to four times daily for two to five days prior to  
75 initial presentation, defined as the day when partial digit amputation was performed and the initial  
76 blood sample was collected. The maximum total leek intake was therefore 200 grams, equivalent to  
77 48.8 g/kg body weight. There was no known exposure to other oxidants associated with Heinz body  
78 anemia.<sup>6</sup> Mean corpuscular hemoglobin concentration was likely falsely increased due to  
79 hemoglobinemia and the presence of Heinz bodies, while hyperbilirubinemia was also a reflection of  
80 increased bilirubin production in the face of hemolysis.<sup>13</sup> Neutrophilia with mild to moderate toxicity  
81 and reactive lymphocytes indicated an inflammatory response and antigenic stimulation, likely  
82 associated with the nail bed infection. Mildly increased urea was suggestive of pre-renal azotemia or  
83 protein catabolism.

84 The coati was placed in an oxygen chamber. Tachypnea and dyspnea improved but worsened  
85 when oxygen therapy was discontinued. Given the severity of anemia and associated clinical signs, a  
86 blood transfusion was performed. A young, healthy, vaccinated, male neutered, full sibling conspecific  
87 with no history of ill health, nor access to leeks, was selected as a blood donor. Both were  
88 anesthetized, the recipient induced with 8% sevoflurane<sup>h</sup> delivered in 100% oxygen via facemask,  
89 intubated and maintained with 4-5% sevoflurane<sup>h</sup> in 100% oxygen, and the donor induced with  
90 medetomidine<sup>i</sup> (0.05 mg/kg, IM) and ketamine<sup>j</sup> (3.92 mg/kg, IM), intubated and maintained with 2%  
91 isoflurane<sup>k</sup> in 100% oxygen. The PCV of the recipient and donor was 0.100 L/L (10%) and 0.300 L/L  
92 (30%) respectively. The blood collection and administration sites were aseptically prepared.  
93 Intravenous (IV) catheterization was initially unsuccessful in the donor coati therefore  
94 medetomidine was reversed with atipamezole<sup>l</sup> (0.25 mg/kg, IM) to resolve presumed medetomidine-

95 induced hypotension. Subsequently fifty milliliters of blood were collected from the donor coati via  
96 IV catheters in the lateral saphenous and jugular veins into syringes containing citrate-phosphate-  
97 dextrose-adenine (CPDA) in a 1:7 ratio of anticoagulant to whole blood. A combination of 2.5 ml  
98 syringes containing 0.31 ml of CPDA and 2.19 ml of whole blood, 5 ml syringes containing 0.63 ml of  
99 CPDA and 4.37 ml of whole blood and 10 ml syringes containing 1.25 ml of CPDA and 8.75 ml of whole  
100 blood were collected. These were transfused to the recipient via an IV catheter in the lateral  
101 saphenous vein in boluses at an initial rate of 1 ml/kg/hour, increasing to 3 ml/kg/hour after 15  
102 minutes then doubled every 15 minutes to reach a maximum rate of 24 ml/kg/hour. Respiratory rate,  
103 heart rate and body temperature were monitored continuously and were stable. A crystalloid  
104 solution<sup>m</sup> (10 ml/kg/hour, intravenously) was administered via a separate IV catheter in the cephalic  
105 vein throughout the procedure. The recipient vomited once on recovery from anesthesia; otherwise  
106 the donor and recipient recovered uneventfully.

107           Provision of leeks was discontinued and a conspecific which shared an enclosure with the  
108 coati examined visually. No clinical signs of anemia were observed. During the subsequent 24-hour  
109 period, the recipient coati was isolated in an enclosure with minimal furniture to reduce activity  
110 levels. The coati was brighter than prior to transfusion, its mucous membranes were less pale and it  
111 was no longer dyspneic or tachypneic at rest; however, the respiratory rate increased from 20 to 40-  
112 50 breaths per minute on exertion. The coati appeared comfortable using its right forelimb therefore  
113 meloxicam was discontinued. Over the subsequent 24 hours, the coati was bright, eating small  
114 amounts and showed no dyspnea or tachypnea. Respiratory rate was stable at 28 breaths per minute  
115 when mixed with conspecifics in its normal environment. A free-catch urine sample was collected.  
116 No abnormalities were observed on dipstick<sup>n</sup> or microscopy and the specific gravity was 1.005. On  
117 days three and four following transfusion, the coati was brighter and climbing normally, with no  
118 dyspnea or tachypnea. Mucous membranes remained slightly paler than those of conspecifics. The  
119 coati was eating a normal amount, although slightly slower than usual, and was sleeping lower down

120 in the enclosure than normal. On day six following transfusion, the coati had returned to normal  
121 behavior and the digit amputation site had healed, therefore antibiotics were discontinued.

122 Four weeks after initial presentation, a blood sample was collected under general anesthesia  
123 to reassess anemia. Anemia had resolved (PCV 0.34 L/L (34%)) (Table 1). There were occasional  
124 eccentrocytes (< 1 per 100 x hpf), indicating minimal residual oxidative injury. Mild macrocytosis  
125 (approx. 1 – 10 per 100 x hpf) and moderate codocytosis (approx. 11 – 20 per 100 x hpf) were  
126 present, consistent with erythrocyte regeneration. Moderate numbers of schistocytes (approx. 3 – 8  
127 per 100 x hpf) and low numbers of keratocytes (approx. 3 – 5 per 100 x hpf) were noted for the first  
128 time, indicating erythrocyte fragmentation. Neutrophilia and neutrophil toxicity had resolved;  
129 however reactive lymphocytes were still present, suggestive of mild antigenic stimulation. Serum  
130 biochemical analytes were within the RI for zoo-housed coatis.<sup>8</sup>

131 Eight weeks after initial presentation, a health examination was performed under general  
132 anesthesia to reassess erythrocyte fragmentation. There were very rare Heinz bodies present,  
133 indicating minimal residual oxidative injury, and mild macrocytosis (approx. 1 - 10 per 100 x hpf)  
134 and codocytosis (approx. 5 – 10 per 100 x hpf) were observed, consistent with erythrocyte  
135 regeneration (Table 1). On serum biochemistry profile, glutamate dehydrogenase was elevated  
136 compared to the initial blood sample and compared to RI in other domestic carnivores (99.0 U/L; dog  
137 RI < 6.0 U/L; cat RI 0.0-0.4 U/L; ferret RI 0.0-2.5 U/L).<sup>10-12</sup> However, there are no published RI for  
138 coatis, other hepatic parameters were within normal limits and no clinical signs of hepatic disease  
139 were evident. Echocardiography and abdominal ultrasound examination showed no abnormalities.  
140 One year following initial presentation, the coati remains clinically well.

#### 141 Discussion

142 To the authors' knowledge, this is the first reported case of Heinz body hemolytic anemia in  
143 a South American coati (*Nasua nasua*). *N. nasua* is a member of the Family Procyonidae, Superfamily

144 Canoidea and Order Carnivora therefore the ferret and dog represent its closest domestic relatives.<sup>14</sup>  
145 Heinz body hemolytic anemia has previously been described in dogs, cats, horses, ruminants, and a  
146 ferret, and has rarely been reported in captive or free-ranging wild animals, including Atlantic  
147 puffins, common marmosets, cotton-top tamarins, herring gulls, koalas, murrees, a red panda and a  
148 river otter.<sup>1-3,6,15-18</sup> Heinz bodies have been described in the erythrocytes of healthy cats and several  
149 wild mammals, but not in healthy coatis.<sup>1,6,19-21</sup> Heinz bodies can cause hemolytic anemia via: 1)  
150 trapping and lysis of affected erythrocytes in the spleen as they are less deformable; 2) lysis of  
151 affected erythrocytes within blood vessels as they are fragile due to binding of Heinz bodies to the  
152 cell membrane or oxidative damage of membrane proteins; and 3) phagocytosis of affected  
153 erythrocytes by splenic or hepatic macrophages as antigens form on their cell membrane when  
154 hemichromes attach to erythrocyte membrane band 3 proteins.<sup>6</sup>

155         Leeks (*Allium ampeloprasum*) are considered the likely oxidant which caused Heinz body  
156 formation in this case. *Allium* spp. contain organosulfur compounds which are absorbed across the  
157 gastrointestinal tract and metabolized to form highly active oxidants.<sup>5</sup> They are not described in the  
158 wild diet of *N. nasua*, nor are they offered as part of the captive diet in zoos.<sup>14,22</sup> In two previously  
159 reported cases of Heinz body hemolytic anemia associated with *A. ampeloprasum* ingestion in a  
160 domestic shorthair cat and cattle, *A. ampeloprasum* was offered for eight or ten days; however, the  
161 amount consumed was unknown.<sup>23,24</sup> The toxicity of onions (*Allium cepa*) in dogs and cats has been  
162 evaluated. Consumption of 5 g/kg of onions in cats or 15-30 g/kg in dogs can cause clinically  
163 important hematologic changes and ingestion of 0.5% of an animal's body weight in onions at one  
164 time is consistently associated with toxicity.<sup>5</sup> A high single dose or smaller doses over several days  
165 may result in Heinz body anemia.<sup>5</sup> Up to 48.8 g/kg body weight of leeks had been added to the diet  
166 over two to five days prior to initial presentation therefore the dose of leeks offered to the coati may  
167 be associated with toxicity. However, species susceptibility is highly variable, dogs and particularly



168 cats being highly susceptible, mice, rats, goats and sheep far less susceptible and humans being more  
169 resistant.<sup>5</sup>

170 Other oxidants associated with Heinz body hemolytic anemia include drugs (paracetamol  
171 [acetaminophen], aspirin, benzocaine, cetacaine, ecabapide [DQ-2511], methylthioninium chloride  
172 [methylene blue], phenacetin, phenazopyridine, phenothiazine, propofol), heavy metals (copper,  
173 zinc), other chemicals (methionine, naphthalene, oil, phenylhydrazine, propylene glycol, skunk musk,  
174 vitamin K<sub>3</sub>), and plants (*Brassica* spp., wilted red maple (*Acer rubrum*) leaves, possibly other types of  
175 maple and other *Allium* spp. including Catalan spring onions, Chinese chive, garlic and  
176 onions).<sup>4,6,15,17,25</sup> Increased Heinz body formation and reduced PCV has also been associated with  
177 skeletal muscle myopathy due to vitamin E, selenium and protein deficiency in common marmosets,  
178 and secondary to diabetes, hyperthyroidism and lymphoma in cats.<sup>6,16</sup> No exposure to these oxidants,  
179 nor evidence of these diseases, were identified in this case.

180 Most of the clinical signs in this case were similar to those previously associated with Heinz  
181 body anemia.<sup>1-5</sup> Gastrointestinal signs tend to occur first after ingestion of *Allium* spp., and clinical  
182 signs associated with anemia take several days to develop, with the most severe being 5-6 days post-  
183 ingestion.<sup>5,26</sup> As expected, the most severe signs in the coati were observed 2-5 days after leeks were  
184 added to the diet. However, several typical clinical signs were not noted, such as anorexia, diarrhea,  
185 icterus and hemoglobinuria.<sup>1-5</sup> Absence of hemoglobinuria and icterus was likely due to hemolysis  
186 not being sufficiently severe. An unusual feature of this case was that Heinz body anemia was  
187 identified on hematology performed during assessment of a phalangeal fracture. It is possible that  
188 the coati was experiencing weakness associated with anemia, which may have predisposed it to  
189 falling and fracturing a digit; however, we suspect that the phalangeal fracture and Heinz body  
190 anemia were unrelated as it is unlikely that there would have been sufficient time for purulent  
191 discharge to develop at the nail bed in association with the fracture if the fracture occurred following  
192 development of anemia.

193           The clinicopathologic features of Heinz body anemia in this case were similar to those  
194 previously described, including anemia, hyperbilirubinemia, Heinz bodies, eccentrocytes and ghost  
195 cells.<sup>6</sup> However polychromasia and reticulocytosis were absent, likely due to insufficient time for  
196 development of a regenerative response or dampening of regeneration by the concurrent  
197 inflammation.<sup>6</sup> Furthermore, reticulocytes may have been missed on the brilliant cresyl blue stained  
198 smear due to the overwhelming presence of Heinz bodies hindering a thorough search for similarly  
199 staining aggregates of ribosomal material. Bilirubinuria, hemoglobinemia and hemoglobinuria were  
200 also absent, likely due to hemolysis not being sufficiently severe.<sup>6</sup> Methemoglobinemia is often  
201 observed in Heinz body anemia cases; however, methemoglobin levels were not measured in this  
202 case.<sup>6</sup> Heinz bodies may be identified on a fresh blood smear stained with Romanowsky stains;  
203 however, they are best seen on blood smears stained with new methylene blue or brilliant cresyl blue  
204 stain.<sup>9</sup> Heinz bodies vary in size, from 1-2  $\mu\text{m}$  in dogs, guinea pigs and rabbits to one third of  
205 erythrocyte diameter in cats, and the total mass of precipitated hemoglobin is indicative of the  
206 severity of the toxic injury.<sup>13</sup> Heinz bodies in the coati of this report were large and numerous,  
207 consistent with a severe toxic insult.<sup>13</sup> More frequent monitoring of erythrocyte parameters and  
208 physical examination could have provided useful information on the resolution of Heinz body  
209 hemolytic anemia in this case; however, this was not pursued as anesthesia would have been  
210 required for each follow-up examination and sample collection due to the non-domesticated nature  
211 of this animal.

212           Management of Heinz body hemolytic anemia involves removal of the oxidant, if possible, and  
213 supportive care. If oxidant ingestion occurred within 2 hours of presentation, unlike in this case,  
214 vomiting can be induced and activated charcoal administered following emesis.<sup>5</sup> Intravenous fluid  
215 therapy is recommended to prevent hemoglobin nephrosis and replace losses from vomiting and  
216 diarrhea.<sup>5</sup> This would not have been tolerated conscious therefore it was only performed during the  
217 anesthetic period. Supplemental oxygen therapy may be required if anemia is severe and a blood

218 transfusion is indicated if the PCV is less than 0.100 L/L (10%), if the PCV has dropped rapidly to less  
219 than 0.200 L/L (20%) in dogs or 0.150 L/L (15%) in cats or if the animal is showing significant clinical  
220 signs of anemia, as observed in this case.<sup>5,7</sup>

221 A donor coati was chosen following several of the criteria for donor dogs and exotic small  
222 mammals, being young (3 years 4 months old), neutered, male, fully vaccinated, in good body  
223 condition, with no signs of ill health or history of infectious disease, and of the same genus and species  
224 as the recipient.<sup>27,28</sup> The donor was also chosen as it had had no access to leeks. The donor coati was  
225 5 kg which is slightly higher than the median body weight derived from 146 zoo-housed South  
226 American coatis aged 3-4 years old and its PCV measured under anesthesia immediately prior to  
227 transfusion was 0.300 L/L (30%) which is within the RI derived from 100 healthy zoo-housed South  
228 American coatis (0.277 – 0.508 L/L (27.7 – 50.8%)).<sup>8,29</sup> It is unknown whether erythrocyte antigens  
229 exist in coatis; cross-matching prior to transfusion might have provided useful information and is  
230 recommended for exotic small mammals due to lack of knowledge regarding blood types.<sup>28</sup>

231 Approximately 10% of total blood volume can be collected from donor dogs with no expected  
232 adverse effects, therefore 50 ml of whole blood was obtained from the 5 kg donor coati in this  
233 report.<sup>27</sup> Assuming coatis have a similar blood volume to dogs, 50 ml of whole blood with a PCV of  
234 0.300 L/L (30%) should increase the recipient PCV from 0.100 L/L (10%) to approximately 0.141  
235 L/L (14.1%).<sup>7</sup> CPDA was used as this is the anticoagulant of choice for small animal blood  
236 transfusions and a gradual increase in the transfusion rate from 1 ml/kg/hour with continuous  
237 monitoring performed to reduce the risk of transfusion reactions.<sup>27</sup>

238 Other than vomiting on recovery from anesthesia, no acute adverse transfusion reactions  
239 were noted.<sup>27</sup> The blood transfusion was successful at immediately resolving clinical signs of  
240 lethargy, dyspnea and tachypnea at rest. Complete clinical recovery to normality was also more rapid  
241 than that described in dogs with experimentally-induced onion toxicity which did not receive a whole

242 blood transfusion.<sup>26</sup> Resolution of anemia was complete by four weeks after initial presentation, as  
243 described in dogs with experimentally-induced onion toxicity.<sup>26</sup> The appearance of schistocytes and  
244 keratocytes at four weeks, indicative of erythrocyte fragmentation, is unexplained. Causes in dogs  
245 and cats include vascular abnormalities, turbulent blood flow, microvascular injury, disseminated  
246 intravascular coagulation, dyserythropoiesis, hepatic disease, glomerulonephritis,  
247 hemangiosarcoma, myelofibrosis and chronic doxorubicin administration.<sup>30</sup> Formation of  
248 microthrombi in the whole blood used for transfusion and subsequent microvascular damage may  
249 have been responsible.<sup>27</sup> Use of a blood administration set and/or blood filter is recommended for  
250 blood transfusions in human and veterinary patients to remove microthrombi and could have  
251 reduced the post-transfusion RBC morphologic changes seen in this case.<sup>27</sup>

## 252 Conclusion

253 The authors describe successful management of Heinz body anemia in a South American coati (*Nasua*  
254 *nasua*) secondary to suspected leek (*Allium ampeloprasum*) toxicosis. Administration of a whole  
255 blood transfusion from a conspecific resulted in rapid resolution of clinical signs and the coati made  
256 a full recovery.

## 257 Footnotes

258 <sup>a</sup> Metacam<sup>®</sup> 5 mg/ml solution for injection for dogs and cats, Boehringer Ingelheim Vetmedica GmbH,  
259 Ingelheim am Rhein, Germany.

260 <sup>b</sup> Buprecare<sup>®</sup> 0.3 mg/ml solution for injection for dogs and cats, Animalcare Ltd, York, United Kingdom.

261 <sup>c</sup> Amoxycare LA Suspension for injection 15% w/v, Norbrook Laboratories Limited, Newry, Northern  
262 Ireland.

263 <sup>d</sup> Metacam<sup>®</sup> 1.5 mg/ml oral suspension for dogs, Boehringer Ingelheim Vetmedica GmbH, Ingelheim  
264 am Rhein, Germany.

- 265 <sup>e</sup> Antirobe<sup>®</sup> Capsules, Zoetis, London, United Kingdom.
- 266 <sup>f</sup> ADVIA 2120i, Siemens Healthcare, Surrey, United Kingdom.
- 267 <sup>g</sup> ILab600, Instrumentation Laboratory, Munich, Germany.
- 268 <sup>h</sup> SevoFlo<sup>®</sup> 100% w/w Inhalation vapor, liquid for dogs, Zoetis, London, United Kingdom.
- 269 <sup>i</sup> Sedastart, Le Vet B.V., Oudewater, The Netherlands.
- 270 <sup>j</sup> Vetalar<sup>®</sup> V 100 mg/ml Solution for injection, Zoetis, London, United Kingdom.
- 271 <sup>k</sup> Isocare 100% w/v Inhalation vapor, liquid, Animalcare Ltd, York, United Kingdom.
- 272 <sup>l</sup> Sedastop, Le Vet B.V., Oudewater, The Netherlands.
- 273 <sup>m</sup> Aqupharm 11 (Hartmann's) Solution for Infusion, Animalcare Ltd, York, United Kingdom.
- 274 <sup>n</sup> Multistix<sup>®</sup> 10 SG Reagent Strips for Urinalysis, Siemens Healthcare Diagnostics Inc., Camberley,  
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345

346 Table 1. Peripheral blood erythrocyte parameters in a South American coati (*Nasua nasua*) with  
 347 severe Heinz body hemolytic anemia at initial presentation (Day 0) and at follow up examinations  
 348 27 and 62 days following initial presentation.

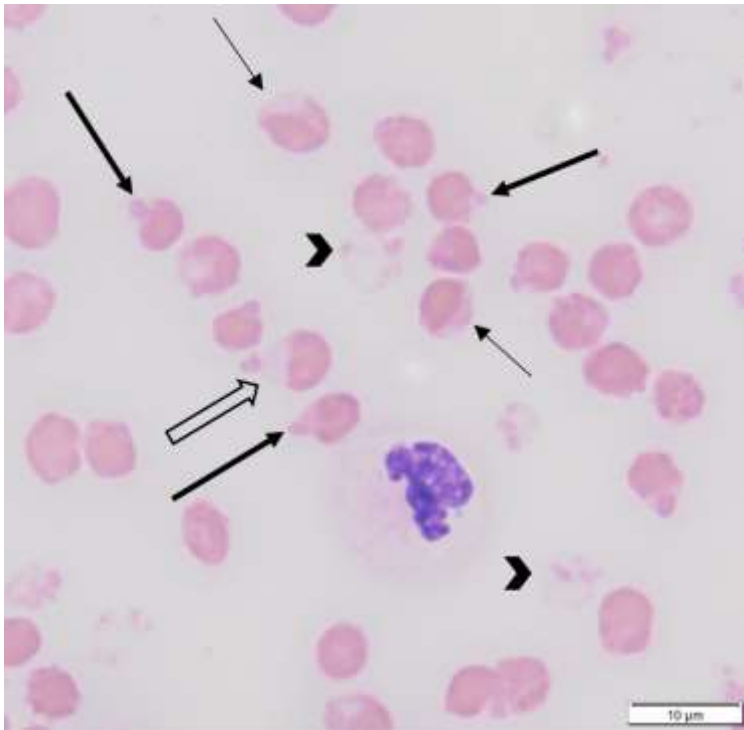
Erythrocyte parameter	Reference intervals	Test result		
		Day 0	Day 27	Day 62
RBC count, x 10 <sup>12</sup> cells/L (x 10 <sup>6</sup> cells/ $\mu$ L)	3.88 - 8.10 (3.88 - 8.10)	2.68 (2.68)	5.35 (5.35)	5.96 (5.96)
Hemoglobin, g/L (g/dL)	88.0 - 151.0 (8.8 - 15.1)	65.0 (6.50)	109.0 (10.90)	110.0 (11.00)
MCV, fL	39.7 - 71.2	49.7	55.2	59.4
MCH, pg	13.6 - 24.7	24.3	20.4	18.5
MCHC, g/L (g/dL)	282 - 391 (28.2 - 39.1)	487 (48.7)	371 (37.1)	312 (31.2)
RDW, %	N/A	21.1	19.8	17.1
PCV, L/L (%)	0.277 - 0.508 (27.7-50.8)	0.11 (11)	0.34 (34)	0.34 (34)
Heinz body presence	-	Numerous	Absent	Very rare
Eccentrocytosis	-	3 - 8 per 100 x hpf	< 1 per 100 x hpf	Absent
Ghost cells	-	3 - 8 per 100 x hpf	Absent	Absent
Schistocytosis	-	Absent	3 - 8 per 100 x hpf	Absent

Keratocytosis	-	Absent	3 - 5 per 100 x hpf	Absent
Codocytosis	-	Absent	11 - 20 per 100 x hpf	11 - 20 per 100 x hpf
Macrocytosis	-	Absent	1 - 10 per 100 x hpf	1 - 10 per 100 x hpf
Hemolysis	-	Moderate	Mild	Absent

349 MCV = mean cell volume, MCH = mean corpuscular hemoglobin, MCHC = mean corpuscular  
350 hemoglobin concentration, RDW = red cell distribution width, N/A = not available. Values for RBC  
351 count, hemoglobin, MCHC and PCV are given in Système International units followed by  
352 conventional units in parentheses. Hemolysis was assessed based on the color of the plasma.  
353 Reference intervals are derived from serum or plasma samples collected from between 15 and 100  
354 zoo-housed South American coatis which were recorded with a normal health status at the time of  
355 blood sampling.<sup>8</sup>

356 Figure legends

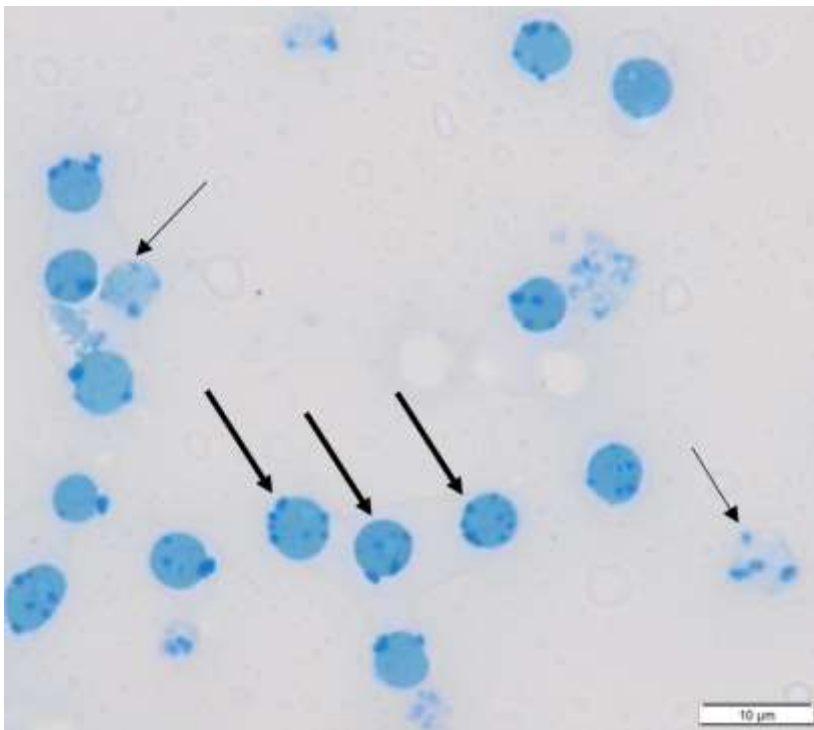
357 Figure 1. Blood smear from a South American coati (*Nasua nasua*) with severe Heinz body hemolytic  
358 anemia. Note the large, protruding Heinz bodies (thick arrows), eccentrocytes (thin arrows),  
359 eccentrocyte with attached Heinz body (double arrow), ghost cells with attached Heinz bodies  
360 (arrowheads), and a neutrophil containing Döhle bodies. Modified Wright's stain, 100x objective, oil  
361 immersion. Bar = 10  $\mu\text{m}$ .



362

363

364 Figure 2. Blood smear from a South American coati (*Nasua nasua*) with severe Heinz body hemolytic  
365 anemia. Note multiple, dark blue staining Heinz bodies within and protruding from erythrocytes  
366 (thick arrows) and attached to ghost cells (thin arrows). Brilliant cresyl blue, 100x objective, oil  
367 immersion. Bar = 10  $\mu\text{m}$ .



368