Resource use and food plant preference of the Bornean elephant in the Lower Kinabatangan wildlife Sanctuary, Sabah

Megan English
Global Visions International Elephant Reintroduction Programme

Optimal foraging theory predicts site re-use (recursion) as a function of the quality of the site, extent of its last use, and time since its last use because these influence site resource status and recovery. We used GPS collars, behaviour and site sampling to investigate recursion of foraging sites for two elephant herds (*Elephas maximus borneensis*) in the Lower Kinabatangan Wildlife Sanctuary, Borneo, over a 12-month period. Recursion occurred to 48 out of 87 foraging sites and was most common within 48 hours or between 151–250 days, indicating two different types of recursion. Recursion was more likely to occur if the site had previously been occupied for longer. Moreover, the time spent at a site at recursion was the same as the time spent at the site on the first occasion. The number of days that had passed between the first visit and recursion was also positively correlated with how much time was spent at the site at recursion. Habitat type influenced the intensity of site-use, with more time spent at recursion within riverine/open grass areas along forest margins compared to other habitat types. Unlike other elephants that live in a forest environment, the Bornean elephant preferred sites containing species from the Poaceae (specifically *Phragmites karka* and *Dinochloa scabrida*) over other plant types including gingers, palms, lianas and woody trees. Recursion, therefore, is a common behaviour used by the elephants and its pattern suggests it may be a foraging strategy for revisiting areas of greater value. The qualities of recursion sites might usefully be incorporated into landscape management strategies for elephant conservation.

Genetic basis of iron storage disease (ISD) in captive black rhinoceroses (*Diceros icornis*)

Donald Paglia, MD[1,3], Rose Linzmeier, PhD[2], Elizabeta Nemeth, PhD[2], and Tomas Ganz, MD, PhD [1,2]
Departments of [1]Pathology & Laboratory Medicine and [2]Medicine, David Geffen School of Medicine at UCLA
[3]UCLA Hematology Research Laboratory

Many mammalian and avian species develop iron toxicity (iron storage disease, ISD) when misplaced from natural ecosystems of their evolutionary origins.[2] When properly studied, all African black (*Diceros bicornis*) and Sumatran rhinoceroses (*Dicerorhinus sumatrensis*) in captivity exhibit evidence of progressive ISD, whereas African white (*Ceratotherium simum*) and Asian Greater One-Horn (*Rhinoceros unicornis*) do not. Pathological effects of ISD are caused by iron-catalyzed reactive oxygen species (ROS) that induce significant morbidity and mortality, often without overt clinical signs or symptoms until irreversible organ dysfunction develops. Recent collapse of the U.S. Sumatran rhino captive breeding program can be directly attributed to ISD that could have been prevented by measures, such as periodic phlebotomies, that are established standards of practice for human ISD.[5] In the wild, iron homeostasis among ISD-susceptible species likely relies on selective consumption of native forage containing compounds with high avidity for dietary iron, allowing enteric passage without systemic absorption.[1] Since iron balance in most vertebrates is regulated through the hormone hepcidin,[3] we compared black and white rhinos by sequencing genes known to be involved in human hereditary hemochromatosis caused by hepcidin deficiencies. Most had identical sequences in black and white rhinos (hepcidin, ferroportin, HFE, hemojuvelin, BMP6), but a mutation in the hepcidin regulator, transferrin receptor 2

(TfR-2), was identified that had G28A amino acid substitution. It remains to be determined if this substitution alters TfR-2 function. We next focused on differences potentially affecting red blood cell (RBC) development and survival, since such defects also cause ISD in humans and mice. Using unbiased mRNA sequencing of liver and spleen mRNA, we identified two mutations in proteins important for RBC function. These mutations were unique to black rhinoceroses compared to white rhinos and all other mammalian species. One mutation was identified in the SLC28a2 gene, which codes for the concentrative transporter for adenosine.[4] Since adenosine salvage is essential for maintenance of BC adenine nucleotides,[6] this mutation may be relevant to the extremely low (2-5%) RBC reserves of adenosine triphosphate (ATP) that are uniquely characteristic of rhinoceroses compared to almost all other mammals. Another mutation was found in EPB4.1, an erythrocyte membrane protein. Functional consequences of these mutations remain to be determined, but they could enhance RBC vulnerability to environmental stresses mediated by ROS, initiating a cycle of low-grade hemolysis and inducing increased iron absorption. Iron-catalyzed ROS in turn could worsen RBC damage, as has been observed in β-thalassemia, thus causing a vicious cycle of RBC destruction and iron overload. Understanding the genetic basis of RBC vulnerability and predisposition to ISD in black rhinoceroses should contribute to more effective captive management of browser rhinos, tapirs and other ISD-susceptible species.

References:

1. Candra D, Radcliffe RW, Andriansyah, Khan M, Tsu I-H, Paglia DE. Browse diversity and iron loading in captive Sumatran rhinoceroses (Dicerorhinus sumatrensis): A comparison of sanctuary and zoological populations. J Zoo Wildl Med. 2012; 43(3)S 65-S72.
2. Clauss M, Paglia DE. Iron storage disorders in captive wild mammals: The comparative evidence. J Zoo Wildl Med. 2012; 43(3)S6-S18.
3. Ganz T, Nemeth. Hepcidin and disorders of iron metabolism. Annu Rev Med. 2011; 62:347-360.
4. Linzmeier R, Thompson, LaMere S, Lee P, Paglia DE, Nemeth, Ganz T. Regulation of iron balance in rhinoceroses. Proc Am Assoc Zoo Vet; 2013. p. 36-3
5. Paglia DE. An historical perspective: Iron overload disease in browser rhinoceroses. Proc Intl Rhino Keep Assoc, Chester, U.K. 2015.
6. Paglia DE, Valentine WN, Nakatani M, Brockway RA. Mechanisms of adenosine 5'-monophosphate catabolism in human erythrocutes. Blood 1986; 67:988-992.

Case report: treatment of iron storage disease in a black rhinoceros (*Diceros bicornis*)in western Europe

Gerda R. van Sonsbeek,DVM
Rotterdam Zoo

In January 2015 Vungu, a 15 year-old black rhino male, housed at the Rotterdam Zoo since 2013, became less active, had less appetite and laid down for prolonged periods. Weekly blood samples were collected for hematology and clinical chemistry. As black rhinos are sensitive for iron storage disease, a monitoring program for this disease was initiated[1,2]. Monthly serum samples were sent to the Veterinary Faculty in Utrecht for measuring serum iron (Fe2+), iron saturation and total iron binding capacity. GGT levels were very high (up to 131U/L, ref: 6-54U/L3) and iron saturation was up to 99%. In most species a saturation of maximum 70% is acceptable. In horses GGT is a reliable indicator for liver damage. Attempts to visualize the liver by ultrasonographic examination failed. Because of the concerns for his physical condition and behavior, a long-term large volume phlebotomy treatment was initiated. At first the procedure was done under general anesthesia (a combination of Immobilon (2.25mg