## Pathological Iron Overloads Acquired in Captivity by Browsing (but not by Naturally Grazing) Rhinoceroses

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African black rhinoceroses (Dicerosbicornis) in captivity are affected by a number of disorders of high morbidity and mortality, including acute episodic hemolytic anemia. Hemosiderosis, the deposition of iron pigments in multiple organs, has been the most consistent necropsy finding in this population and has most commonly been interpreted as evidence of previous hemolytic events. Direct participation in necropsies of black rhinoceroses dying in captivity, and review of histopathology of previous necropsies, revealed magnitudes and patterns of tissue iron deposition that were incompatible with hemolytic disease alone, but instead were indicative of a true iron overload syndrome that progressed in severity with time in captivity. This interpretation was supported by quantitative analyses of necropsy tissues and serum iron analytes, including sera from four of the five extant species of rhinoceroses and from both captive and free-ranging black and white (Ceratotherium simum) rhinoceroses. Significant, often extreme, elevations in serum and tissue iron and ferritin concentrations and transferrrin saturations were observed in captive adult black rhinoceroses compared to all control groups. Similar elevations were observed in the few Sumatran (Dicerorhinus sumatrnesis) rhinoceroses available for study, but not in the two species of natural grazers (African white and Asian greater one-horned [Rhinocerosnuicornis]). These findings suggest that iron homeostasis in browsing rhinoceroses may be dependent on natural iron chelators, such as tannins, phytate, mimosine, etc., that may not be included as components of formulated captive diets. Excessive iron stores may contribute directly and/or indirectly to several of the other serious disorders threatening this species in captivity, such as susceptibility to infections in general, to tuberculous and exotic fungal pneumonias specifically, and to acute and chronic anemia, toxic hepatopathies, and stress intolerance.