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Health of the Forest Rhinoceros of Southeast Asia: Sumatran and Javan Rhinoceros

ROBIN W. RADCLIFFE AND KURNIA OKTAVIA KHAIRANI

Introduction

The rhinoceros living in the rainforests of Southeast Asia now survive almost entirely within the boundaries of Indonesia and represent the most threatened species of the Rhinocerotidae family. Following Bergmann's rule, the Sumatran rhinoceros *(Dicerorhinus sumatrensis)* and Javan rhinoceros *(Rhinoceros sondaicus)* are smaller than their African and Asian relatives living outside of tropical environments.[1] For both species for which conservation has historically included (Javan) or currently includes (Sumatran) attempts at a managed breeding program, our understanding of their biology and associated threats from disease draws us to a singular conclusion: their health is integrally linked to the native forests. Invariably, captive browsing rhinoceros, Sumatrans included, are susceptible to captive-induced disease in the form of gastrointestinal ailments, infectious disease, enhanced susceptibility to ocular disorders, and most concerning of all—due to its insidious onset and irreversible progression—iron storage disease (ISD). The objective of this chapter is to summarize in one place the most current state of health knowledge in captive and wild settings for these unique forest rhinoceros representing two diverse genera (Table 100.1).

Sumatran Rhinoceros (*D. sumatrensis*, Fischer 1814)

Taxonomy

Known by its colloquial name of the "hairy rhinoceros," three subspecies of Sumatran rhinoceros are recognized. *D. sumatrensis sumatrensis* is found in just three protected areas on the island of Sumatra (Way Kambas, Bukit Barisan Selatan, and Gunung Leuser National Parks), whereas *D. sumatrensis harrissoni* is found in exceedingly small numbers in Sabah (Malaysian Borneo) and Kalimantan (Indonesian Borneo) on the island of Borneo.[2] Although there are unsubstantiated claims that a small population persists in Mynamar, *D. sumatrensis lasiotis* is presumed extinct.

Infectious and Emerging Disease

Bacteria, Viruses, and Preventative Medicine

Sumatran rhinoceros mortality from necrotizing enteritis and septicemia following gastrointestinal infection with *Escherichia coli*, *Klebsiella*, and *Salmonella* spp. was prevalent in captive animals before advances in zoo husbandry (see Table 100.1).[3,4] No viral diseases have been described in the Sumatran rhinoceros, although West Nile virus and equine herpesvirus type 1 (EHV-1) infections have caused disease in captive greater Asian one-horned rhinoceros.[5,6] Fecal samples should be collected for salmonella culture and serotyping in cases of acute diarrhea. Skin tuberculin testing is recommended during quarantine and in high-risk environments. Vaccination against tetanus, rabies, West Nile virus, and leptospirosis are based on perceived risk and local veterinary practice.

Captive management of Sumatran rhinoceros requires routine health monitoring and basic animal husbandry practices, including physical exam with measurement of body weight, condition scores, screening for endoparasites and ectoparasites, and serial hematology and biochemistry (Table 100.2).[7] Blood may be collected readily in small volumes from the auricular vein located on the outside of the rhinoceros's pinna, from the tail or coccygeal vein, or from the radial vein.[8] It is relatively easy to collect blood from standing nonsedated rhinoceros in a chute while hand-feeding fruit (jackfruit, durian, melon, and bananas are favorite treats).

TABLE 100.1 Records of Disease Outbreaks With Associated Parasites and Pathogens in Captive and Wild Sumatran *(Dicerorhinus sumatrensis)* and Javan Rhinoceros *(Rhinoceros sondaicus)*

Species	Disease	Dates	No. Affected	Evidence	References
Sumatran Rhinoceros	*Trypanosoma evansi*	2003, 2006	6 (C)	• 5 died, *T. evansi* in blood smears, histopathology confirmed; poor hygiene proposed by critics—*E. coli* overgrowth without pathology • Trypanosomes found in rhinoceros blood (disease survey)	6, 14
	Klebsiella pneumoniae	1986	1 (C)	• Died after showing gastrointestinal signs • *Klebsiella* cultured from GI tract, necropsy indicated septicemia	2
	Ectoparasites	2006, 2007	- (C)	• *Haemaphysalis hystricis, Amblyomma testudinarium, Aponomma* spp.	4, 9
	Necrotizing Enteritis	1989	1 (C)	• Death after GI signs, necropsy suggests *Salmonella* enteritis	3
	Hemoparasites	2006, 2007	- (C)	• Microscopy: *Anaplasma marginale, Anaplasma centrale*, *Theileria* spp., and *Babesia* spp; Molecular techniques: *Theileria bicornis*	6
	Helminths	1986, 2006, 2007	- (C)	• *Fasciolopsis buski* infestation identified • *Fasciolidae*, *Oxyuris, Paramphistomidae*	5*, 6, 8
	Protozoa	2006, 2007	- (C)	• *Cryptosporidium, Entamoeba, Balantidium, Ophryoscolecidae, Spirodinium* spp.; families Buetschliidae, Cycloposthidae	6
	Iron Overload Disorder (Hemochromatosis, Iron Storage Disease)	1992–2017	9	• Biochemical & necropsy histopathology (6), biochemical (transferrin saturation & ferritin) (3)	Don Paglia, personal communication, 2018
	Ocular Disease	1990, 2004–2005	6 (C)	• Corneal opacity and ulceration, loss of vision in eye(s) • *Staphylococcus* and *Flavobacterium* spp. cultured	4, 16
	Lacerations	1990	- (W, C)	• Lacerations from snares, wood floors	4
	Hyperkeratosis	1990	- (C)	• Hyperkeratosis, *E. coli* pyoderma a common sequela	4
	Abscesses	1990	- (C)	• Injection sites, puncture wounds, horn abscesses	4
	Hoof Cracks	1990	- (C)	• Husbandry related	4
	Myiasis	1990	2 (C)	• No details given	4
	Phimosis	1990	1 (C)	• Attachment of penis to prepuce, approximately 5 cm of penis free • Improved markedly over 3–4 months	4
	Uterine Pathology	1986, 2001	- (C)	• Histology—Uterine leiomyoma, cystic endometrial hyperplasia • Ultrasound—tumors, uterine cysts, multiple corpora lutea	2, 25
Javan Rhinoceros	**Helminths**	1980, 2006	- (W)	• *Strongyloides, Bunostomum, Trichostrongylus, Fasciola, Schistosoma, Anaplocephalidae, Oesophagostomoma, Plagiotaenia*	31, 29
	Protozoa	2006	- (W)	• *Balantidium, Entamoeba, Cryptosporidium, Eimeria, Cycloposthium, Lavierella* spp.	31
	Ectoparasites	1980	10 (W)	• *Amblyomma* spp.	30
	T. evansi	2011	1 (W)	• Circumstantial—tabanids had positive *T. evansi* on PCR • Soil also positive for *C. difficile* (normal soil contaminant)	32
	Hemorrhagic Septicemia (HS) or Anthrax	1982	5 (W) 33* (W)	• Circumstantial-350 goats & 50 • buffalo died of HS in adjacent villages • History of anthrax 3 decades before	

(C) and (W) indicate captive and wild rhinoceros, respectively. References marked with an asterisk (*) are not peer reviewed.
Table prepared by Virgina Mule, DVM.
PCR, Polymerase chain reaction.

TABLE 100.2 Hematology and Biochemistry Values for Adult Captive Sumatran Rhinoceros *(Dicerorhinus sumatrensis)[*]**

Analyte	Mean ± SD	Median	IQR	Min	Max	CV_g (%)	C_{Vti} (%)	Index of Individuality[†]
Hemoglobin (g/dL)	13.2 ± 1.1	—	—	10.5	16.1	3	8	2.53
White blood cell count ($\times10^3/\mu L$)	7.1 ± 1.6	—	—	3.2	12.2	17	16	0.89
Red blood cell count ($\times10^6/\mu L$)	5.1 ± 0.5	—	—	4.1	6.5	4	8	2.33
Platelets ($\times10^3/\mu L$)	133 ± 59	—	—	18	280	24	40	1.68
Hematocrits (%)	39 ± 3	—	—	32	48	2	8	3.53
Mean corpuscular hemoglobin (pg)	26 ± 1	—	—	23	28	4	3	0.67
Mean corpuscular hemoglobin concentration (g/dL)	34 ± 1	—	—	32	37	2	2	0.98
Mean corpuscular volume (fl)	76.5	—	—	62.7	94.1	—	—	—
Protein (g/dL)	8.1 ± 1.1	—	—	5.2	10.8	3	13	4.66
Albumin[‡] (g/dL)	–	3.9	3.6–4.5	1.9	7.3	1	17	24.70
Globulin (g/dL)	3.9 ± 1.3	—	—	0.5	6.6	3	33	12.39
Aspartate aminotransferase (U/L)	72 ± 23	—	—	40	140	27	22	0.79
ALT[‡] (U/L)	–	21	16–31	7	69	5	14	3.05
LDH[‡,§] (U/L)	–	884	684–1217	212	3583	<1	8	16.64
Bilirubin_Total (mg/dL)	0.6 ± 0.2	–	–	0.3	1.2	14	31	2.25
Bilirubin_Direct[§] (mg/dL)	0.3 ± 0.1	–	–	0.01	0.6	2	52	23.68
Bilirubin_Indirect (mg/dL)	0.4 ± 0.2	–	–	0.1	0.9	17	48	2.85
Serum urea (mg/dL)	21 ± 7	–	–	6	42	20	31	1.55
Creatinine[‡] (mg/dL)	–	1.1	1.0–1.5	0.6	2.9	146	130	0.89

*Hematology and biochemistry values with estimates of central tendency (mean or median), variability (standard deviation [SD] or interquartile range [IQR]), minimum and maximum reference interval values, between-animal coefficient of variation (CV_g), within-animal coefficient of variation (CV_{ti}), and index of individuality for adult captive Sumatran rhinoceros *(Dicerorhinus sumatrensis)* at the Sumatran Rhino Sanctuary, Lampung, Indonesia.
[†]Estimated with the use of the equation CV_{ti}/CV_g.
[‡]The median and IQR are reported rather than the mean and SD because of the skewed distribution of these analytes; however, log-transformed variables were used in mixed ANOVA models to calculate CVs and the index of individuality.
[§]Ratu is excluded because of low sample numbers in order to estimate the between-individual animal variance.
Modified from Andriansyah, Candra D, Riyanto MA, et al. Hematology and serum biochemistry of Sumatran rhinoceros *(Dicerorhinus sumatrensis)* in a rainforest sanctuary in Way Kambas National Park, Indonesia. *J Zoo Wildl Med* 44(2):280–284, 2013.

Endoparasites

Internal parasites of the Sumatran rhinoceros include roundworms, flatworms, and protozoa of the genera typical of large ungulates with *Fasciolidae*, *Paramphistomidae*, *Strongyloidae*, *Oxyuridae*, *Cryptosporidium*, *Entamoeba*, *Balantidium*, *Ophryoscolecidae*, and *Spirodinium* spp. identified in captive animals (see Table 100.1).[9,10] Routine screening of animals at the Sumatran Rhino Sanctuary in Lampung Province, Indonesia was conducted using direct smears, magnesium sulfate, and acid techniques, which proved ideal for identifying fluke eggs because sugar flotations collected too much debris. Acid sedimentation techniques were superior to use of the McMaster chamber for quantification of egg counts.[11]

Ova of *Fasciola* sp. (liver fluke) was detected in feces of four of five sanctuary rhinoceros, whereas adults and ova of a previously identified *Paramphistome* sp. (stomach fluke) were found in feces of one of five rhinoceros with ova measuring 125 × 60–65 µm and 150 × 60–65 µm, respectively. Even though no apparent disease was attributable to these fluke infections, treatment was initiated with praziquantel at 3 mg/kg orally, with a slight decrease in egg

counts observed following a single trial. Because reinfection was likely, control of flukes in a wet rainforest environment must also target the snail intermediate host through environmental interventions, such as clearing of vegetation around day stalls where rhinoceros feed. A *Lymnea* sp. snail was identified in a wallow frequented by the rhinoceros at the sanctuary.[10–12]

Ticks and Tick-borne Disease

External parasites, including ticks, flies, and leeches, are common in the warm humid environments where Sumatran rhinoceros live. In addition to taking a blood meal (which may offer a natural mechanism for iron reduction), these parasites are vectors for important diseases. Of note are the hemoparasite infections carried by ticks in the family Ixodidae. In a survey of four captive Sumatran rhinoceros living in native rainforest habitat in Way Kambas National Park, two species of ticks were identified, *Haemaphysalis hystricis* (81%) and *Amblyomma testudinarium* (19%), with predilection for the neck and shoulder skinfolds of the animals. At the time of the 2008 tick survey, simultaneous microscopic hematoparasite examination of Giemsa-stained blood smears collected from the captive rhinoceros revealed tick-borne diseases, including *Anaplasma marginale* (27%), *A. centrale* (10%), *Babesia* sp., and *Theileria* sp. (15%).[10,13] Further molecular analysis using reverse line blot hybridization (RLB) and nested polymerase chain reaction (PCR) revealed *Theileria bicornis* in a single Sumatran rhinoceros. *T. bicornis* was first described as a novel blood parasite in free-ranging black rhinoceros *(Diceros bicornis)* in South Africa and, although fatal babesiosis from infection with *Babesia bicornis* was described in three black rhinoceros, there was no evidence that *T. bicornis* was associated with disease in African rhinoceros.[14]

In an effort to boost immunity against tick-borne pathogens, one captive-born Sumatran rhinoceros destined for repatriation back to Indonesia from an American zoo received three doses of a babesia-anaplasma vaccine (lyophilized protein of *Babesia bigemina*, *B. bovis,* and *A. marginale* of bovine origin) prior to the translocation.[15] Although no postvaccine titers were measured, the rhinoceros made a smooth transition into the tick-endemic environment of Way Kambas, with mild subclinical hemoparasite infections documented in serial blood smears.

Tabanids and Trypanosomes

The emergence of animal trypanosomiasis (surra) in Sumatran rhinoceros highlights the growing threat of pathogens transferred to novel hosts that have not adapted (or poorly adapted) to the agent.[16] Trypanosomes evolved on the African continent, and African rhinoceros have evolved a relatively stable host-parasite relationship, with disease observed primarily during periods of stress or following translocation of naïve animals into tsetse fly zones.[17] However, Asian rhinoceros are particularly susceptible and suffer high mortality.

A 2003 outbreak in a captive population of Sumatran rhinoceros housed in peninsular Malaysia at the Sungai Dusun Conservation Center was attributed to infection with *Trypanosoma evansi*.[18] The epidemic was characterized by a biphasic die-off of animals with clinical signs that varied from anorexia and depression to incoordination, rear limb paralysis, and recumbency. Pathology at the time of the outbreak showed overgrowth of *E. coli* and *Klebsiella* bacteria from multiple organ systems, generating significant debate about the level of hygiene and husbandry at the sanctuary.[19] However, subsequent histopathology revealed that the bacteria were not associated with disease but rather consistent with overgrowth. Furthermore, trypanosomes invaded tissues and were found in various organs (including the brain), together with unique lesions in the spleen consisting of enlarged periarteriolar sheaths with lymphoid depletion, pathologic lesions characteristic of surra in other mammals.[16,18]

Noninfectious Disease

Eye Disorders

The Sumatran rhinoceros has a propensity for ocular disorders that is greater than that observed in other captive rhinoceros species. Excessive exposure to ultraviolet light (UV) is the primary factor implicated in the ocular syndrome, although a multifactorial etiology is suspected given the broad presentation of clinical signs in a variety of environments, including confinement within range countries. Eye conditions progress from mild corneal edema to severe opacity, uveitis, and blindness, with secondary bacterial and fungal infections common sequelae. A case summary of a breeding pair of Sumatran rhinoceros in Sabah Malaysia compared development of clinical eye disease with indoor and outdoor locations in an attempt to elucidate causation from light intensity or other environmental factors. A seasonal pattern was noted, with all eye disorders appearing in the months of July and August, although no correlation could be found to excessive UV exposure or dry conditions.[20]

The difference in light intensity in captive environments compared with natural tropical forest architectures is likely significant, given that chronic recurrent eye syndromes are also prevalent in the captive Malayan tapir *(Tapirus indicus)* coming from the same region. The rainforest environments where these rhinoceros live consist of a complex forest structure in four layers, namely the emergent, canopy, understory, and forest floor. The extensive canopy and emergent layers filter direct sunlight before it reaches the forest floor. In a study of forest structure in Costa Rica, canopy architecture and light transmittance in both secondary and old growth rainforests were compared—diffuse transmittance of light at 1–2 m above the forest floor (the understory level where Sumatran rhinoceros live) was less than 3%.[21] Although shade structures in captive environments appear to help reduce eye disease in this species, it is not always sufficient. One captive Sumatran rhinoceros housed in an environment with extensive shade structures and high humidity

developed eye disease during the winter months when cloudy conditions predominated.

The integument of the black rhinoceros has been implicated as the primary organ in which allergic or disease conditions manifest under a variety of circumstances, suggesting that their epidermis is highly sensitive to disruption of metabolic homeostasis.[22] The corneal epithelium of the Sumatran rhinoceros eye may respond to disruptions in a similar manner with nutritional deficiency, UV exposure, and dry conditions leading to increased disease states and reduced healing—all of which are compounded with life in captive environments. The first sign of eye disease presents as corneal edema; then, if not treated with aggressive topical medication, peripheral vessel ingrowth occurs and pigmentation follows. Some animals progress to recurrent anterior uveitis similar to moon blindness in horses (S. Citino, personal communication, March 8, 2017). A vicious inflammatory cycle of reinjury drives pathogenesis of ocular disease, with the initial insult causing inflammation that augments further injury to the ocular surface—invasion of leukocytes and release of immune mediators from damaged cells leads to cyclic damage and reinjury. Therefore the best response to treatment may be achieved using topical cyclosporine ointment, an immunomodulator that inhibits T-lymphocyte proliferation (S. Citino, personal communication, March 8, 2017).

Iron Overload Disorder (Hemachomratosis, Iron Storage Disease)

The induction of toxic overburdens of elemental iron in captive black rhinoceros was first noted by Smith et al. (1995).[23] Subsequent evidence extended these findings to include Sumatran rhinoceros, the only other browser rhinoceros currently managed in captivity.[24,27] Sumatran rhinoceros develop progressive iron overloads even more rapidly than do African black rhinoceros, reaching tenfold elevations in body burdens within as little as 3 years of captive birth or transfer into captive conditions and increasing in direct relation to time in captivity.[24,27]

Measurements of serum *ferritin* concentrations and *transferrin saturation* (the ratio of serum iron to total iron-binding capacity [TIBC]) provide the least invasive means to assess iron status. It is widely acknowledged that serum ferritin concentrations reflect total body iron stores with an accuracy exceeded only by direct quantitative analyses of tissue samples.[25] Most rhinoceros studies have relied on the assay developed by Smith et al. (1984),[26] which is available through the Kansas State University Veterinary Diagnostic Laboratory. In separate studies, serum ferritin values measured by this assay in African black and white rhinoceros *(Ceratotherium simum)* free-ranging in their natural habitats were less than 100–200 ng/mL.[27] By contrast, specimens from 14 captive Sumatran rhinoceros averaged greater than 850 ng/mL, with individual values ranging as high as 2000–4000 ng/mL.

Despite its widespread use and verified correlation with quantitative tissue assays and necropsy histopathology, the Smith et al. ferritin assay has been criticized by some because it requires species-specific reagents with variable cross-reactivity among diverse species and because results sometimes seem highly variable in individual animals. The latter is likely due to serial dilutions of plasma that are required for exceptionally high ferritin concentrations in species with captivity-induced ISD. In addition, results may be confounding because ferritin is an acute-phase reactant that elevates secondarily in a number of inflammatory, neoplastic, or other conditions.

Transferrin saturation, the amount of iron bound to the plasma transport-protein transferrin, provides a simple, qualitatively reliable, supplement or alternative if ferritin assays are equivocal or unavailable. Transferrin saturation correlates well with ferritin concentrations, with quantitative tissue analyses, and with histopathology using ferric-specific stains such as Prussian blue.[24,27] Transferrin saturation in most vertebrates is approximately 35%. US captive Sumatran rhinoceros measure 90%–100%, clearly indicating iron in sufficient excess to overwhelm carrying capacity of protective proteins.[24,27] Alternative systems for measuring ferritin and assessing ISD status have been proposed, but these have not yet been validated by studies directly comparing both assay systems or their relation to demonstrable histopathology.[28,29]

Nutrition is fundamental to the health of the browsing rhinoceros, whether they are managed in captive or semicaptive environments. A comparison of browse diversity in Sumatran rhinoceros housed in three diverse settings (North American zoo, Malaysian center, Indonesian sanctuary) demonstrated marked differences in nutritional management and predicted that these disparities relate directly to differences in iron loading.[30] Browse diversity was measured across five areas: number of locally available plant species, number of plant species fed daily, access to a free-range browse environment (i.e., native rainforest), transit time from plant cutting to feeding, and percentage of nonbrowse items in diet (i.e., hay or pelleted ration). When comparing traditional zoo rhinoceros with sanctuary animals, zoo rhinoceros were fed fewer species of browse (8 vs. 100 species); fed fewer species on a daily basis (2–3 vs. 8–10 species); spent fewer hours browsing (0 vs. 6 hours); ate browse that had been in transit longer (>72 vs. <12 hours); and fed significantly more nonbrowse items as percentage of diet (20%–38% vs. 0%). These same groups differed in iron stores, with zoo rhinoceros having higher mean ferritin than sanctuary rhinoceros managed in range countries (2835 ± 295 ng/mL vs. 680 ± 168 ng/mL, respectively) (see also Chapter 99).

The inevitable morbidity and mortality of chronic progressive iron toxicity can be prevented by induction of negative iron balance through periodic phlebotomies, as validated by extensive experience with an equivalent human disorder, hereditary hemochromatosis. The clinical effectiveness of this procedure has been validated in African black rhinoceroses at multiple institutions, but has not yet been applied appropriately to Sumatran rhinoceroses (D. Paglia, personal communication, 2018).

Chronic Renal Disease

A 30-year-old male Sumatran rhinoceros named *Torgamba* housed at the Sumatran Rhino Sanctuary in Lampung, Indonesia developed renal disease characterized by progressive azotemia, hypercalcemia, and hypophosphatemia (data summarized over a 4-year period showed progressive deterioration in renal function as measured by BUN 30.3 ([10–52 µg/dL]; creatinine 3.2 [0.87–20.7 µg/dL]; Ca 15.8 [8.9–28.3 mg/dL]; Ph 3.3 [1.3–9.7 mg/dL]; and Ca to Ph ratio 5.6:1 [1.2:1–11.8:1]. Radcliffe, RW and Candra D: unpublished data, 2009). The disease was monitored with serial measurement of body weight and serum biochemistry analysis on a weekly basis. Nutritional management of the disease focused on feeding a highly palatable selection of browse that included hand-feeding during periods of inappetence together with supplementation of elemental phosphorus (Equi-phos; Uckele Health & Nutrition, Blissfield MI 49228: guaranteed analysis of Ph 19%; Na 4.5%–5.5%, and Ca 0.1%–0.2%, with each ounce supplying 5.4 g of elemental Ph), electrolyte water, and a salt lick. Phosphorus supplementation ranged from 1–4 oz per day, with dosing changes based on the most recent biochemistry panel; in general, the dose was increased by 1–2 oz per day when the Ca to Ph ratio exceeded 5:1. The condition was managed successfully for half a decade before the rhinoceros finally deteriorated and died from complications of the disease.

Reproductive Pathology and Allee Effect

The development of reproductive pathologies in female Sumatran rhinoceros impacts both captive and wild conservation programs. Uterine tumors, such as leiomyomas and cystic endometrial hyperplasia, have been visualized on ultrasound and confirmed on histopathology.[31] These diseases are more common in older animals and may be related to physiologic states related to long-term estrogen influence from cycling without pregnancy, a condition observed in other captive rhinoceros. Uterine and ovarian masses have also been observed in recently captured wild female rhinoceros, inferring that the same pathologies may be developing in wild animals. With their slow breeding rate (long gestation and intercalving interval), small populations of rhinoceros are particularly susceptible to stochastic factors and the Allee effect (i.e., solitary nature and reduced mating opportunity, reproductive pathologies from prolonged periods of nonparity, skews in sex ratios, and inbreeding depression), with the end result being fewer births than deaths.[19]

Javan Rhinoceros *(R. sondaicus*, Desmarest 1822*)*

Taxonomy

The Javan rhinoceros or one-horned lesser rhinoceros *(R. sondaicus)* is one of the most critically endangered terrestrial mammals in the world. There are three distinct subspecies, of which only one is extant; the Indonesian Javan rhinoceros *(R. sondaicus sondaicus)* solely inhabiting Ujung Kulon National Park (UKNP), with 67 individuals recorded in 2016; the Indian Javan rhinoceros *(R. sondaicus inermis)* now extinct but once common throughout Bengal, Bangladesh, and Burma; and the Vietnamese Javan rhinoceros *(R. sondaicus annamiticus)* recently declared extinct in Cat Tien National Park, Vietnam and formerly also found in Cambodia, Laos, Thailand, and Malaysia. Currently, many challenges threaten the last population of Javan rhinoceros, including infectious disease at the wildlife–domestic animal interface and noninfectious disease (toxic plants, invasive arenga palm, parasitism, and feeding competition with sympatric ungulates), all compounded by the significant demographic risk of natural disaster and inbreeding depression inherent in a single small population. If not addressed, these challenges may create an irreversible extinction vortex.

Javan Rhinoceros Mortality Events and Infectious Disease

The first population census of UKNP was conducted in 1955 by IUCN Director-General, Dr. Lee Talbot, and repeated a dozen years later by WWF researcher, Professor Schenkel; both recorded fewer than 30 Javan rhinoceros.[32,33] Since then, the population has fluctuated between 58 and 69 individuals. In 1982 the first of several mortality events was reported, with five carcasses discovered with horns intact, representing 8.9% of the population (see Table 100.1).[33] The investigation focused on infectious disease because the park lies adjacent to local agricultural communities with a significant population of water buffalo *(Bubalus bubalis)*. The sequence leading to death was deduced from traces at the site—walking and feeding, diarrhea, lying down, convulsive leg movements, and death. One comparatively fresh carcass showed prolapse of rectum and foamy mucus at the mouth and nostrils. Hemorrhagic septicemia (HS) is an infectious disease caused by the gram-negative bacteria *Pasturella multocida.* HS is a fatal disease of cattle, yak, camel, and water buffalo. In 1981 an HS outbreak was responsible for the death of 350 domestic goats and 50 buffaloes in the region around the park. Anthrax outbreaks were also recorded locally several decades previously. Despite inconclusive laboratory findings from the soil samples collected at the site, Schenkel concluded that anthrax was most likely the causative agent because the spores are long lived and clinical signs were typical of acute outbreaks.

Following the 1982 die-off, the local government sponsored an HS vaccination program to districts that were affected by the outbreak, although the implementation remains intermittent and irregular. For a 1-year period from June 2012 to July 2013, a disease surveillance study was conducted to investigate the prevalence of HS.[34] The study was conducted in 19 buffer villages surrounding the national park due to a high risk of cross-infection between the villagers' water buffalo and ungulates in the park, including the Javan rhinoceros. Blood samples for serology ($n = 770$) and nasal swabs for culture ($n = 85$) were collected

from water buffalo and compared with perceived risk factors for buffalo herd management. A low seroprevalence of 1.8% (14 of 770 animals) was observed, suggesting that carrier animals could contribute to ongoing outbreaks in the park. Husbandry practices associated with a positive serologic response in water buffalo were: lack of a permanent area to house buffalo at night; low body condition score (BCS = 2); high body temperature (fever ≥40°C); a history of clinical signs or sudden death in the previous year; and a grazing system that accessed significant forage inside the park. Serologic response was not associated with sex, age, vaccination status, or season.[34]

Historic surveillance for endoparasites in Javan rhinoceros fecal samples has demonstrated cestode, nematode, and trematode infections with *Strongyloides*, *Bunostomum*, *Trichostrongylus*, *Fasciola*, *Schistosoma*, *Anaplocephalidae*, *Oesophagostomoma*, and *Plagiotaenia*.[35–37] Protozoans isolated include *Balantidium*, *Entamoeba*, *Eimeria*, *Cryptosporidium*, *Cycloposthium*, *Lavierella*, and *Ophryoscolecidae*, some of which are known pathogens in other species.[37]

Vector-borne disease is emerging as a significant potential threat to the UKNP Javan rhinoceros population, given the disease reservoir of buffalo that are grazed inside the park. Tabanid flies of the genus *Tabanus* are common hemoparasites known to transmit animal trypanosomiasis or surra. Trypanosomiasis infects cattle, water buffalo, horses, elephants, camels, and rhinoceros, with Asian species being highly susceptible.[16,18] Surveillance for *T. evansi* in 2014 demonstrated a high prevalence (90%) of trypanosomiasis in the livestock of two villages intersecting directly with the park boundary. A comprehensive study of tabanid vector biology, including trypanosome infection rate and host blood meal analysis, is underway to better understand host-parasite-vector dynamics in the UKNP ecosystem.[38,39]

Noninfectious Disease

One of the most challenging aspects of conserving the Javan rhinoceros is the insufficient data regarding the species and the habitat that shelters it. The Javan rhinoceros population has been on the rise since 1937; however, for the past 2 decades, population growth has been stagnant. A variety of extrinsic and intrinsic factors may contribute to this population plateau.

Because vegetation analysis data in UKNP is limited, it is possible that toxic plants have contributed to rhinoceros mortalities. An invasive palm *(Arenga obtusifolia)* known locally as "langkap" may lower the carrying capacity for browsers in the park. The invasive palm crowds out sunlight and reduces secondary growth that provide the natural food plants for the Javan rhinoceros. With the disappearance of open grasslands in the park, banteng *(Bos javanicus)* may be competing with Javan rhinoceros for available browse. The UKNP banteng population has been increasing from 200 in 1983 to more than 800 individuals in 2000.[40]

Finally, the Allee effect may further limit Javan rhinoceros population growth rates. The idea that population size may impact fitness is significant for both species of Indonesian rhinoceros.[19] Cooperative rhinoceros behaviors such as feeding, breeding, territorial defense, and communication through dung middens are less effective at low population size, leading to decreased survivorship. Likewise, the per capita risk from predation and disease are heightened in small populations; a recent camera trap recording documents predation of a banteng juvenile and a Javan rhinoceros bull followed closely by a pack of Javan dhole *(Cuonalpinus javanicus)*.

Demographic Risks to a Single Population

The loss of the Vietnamese subspecies of Javan rhinoceros in 2011 leaves UKNP as the last habitat for Javan rhinoceros in the world. UKNP lies at the western most tip of Java Island in the heart of the Sunda Arc, an area of converging tectonic plates that commonly produces earthquakes and triggers tsunamis.[41] In 1883 the eruption of Krakatoa devastated Ujung Kulon and its surrounding area, making way for the Javan rhinoceros to colonize the region. Ironically, the same threat that gave the Javan rhinoceros its last refuge is looming with Strombolian eruptions of Anak Krakatau (Child of Krakatoa) actively spewing lava into the sea.[42]

The creation of a second population of Javan rhinoceros, remote from Ujung Kulon in Cikepuh Wildlife Reserve, has been proposed to the government of Indonesia.[43] Ecologic, biological, and socioeconomic viability assessments are underway to evaluate the readiness of Cikepuh to host a founder population of four select Javan rhinoceros based on distinct mean kinship. The second population strategy is planned for execution in 2023.

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