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# Soil Correlates and Mortality from Giraffe Skin Disease in Tanzania

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ABSTRACT: Giraffe skin disease (GSD) is a disorder of undetermined etiology that causes lesions on the forelimbs of Masai giraffe (*Giraffa camelopardalis tippelskirchi*) in Tanzania, East Africa. We examined soil correlates of prevalence of GSD from 951 giraffe in 14 sites in Tanzania, and estimated mortality using 3 yr of longitudinal mark–recapture data from 382 giraffe with and without GSD lesions, in Tarangire National Park (TNP). Spatial variation in GSD prevalence was best explained by soil fertility, measured as cation exchange capacity. We found no mortality effect of GSD on adult giraffe in TNP. Based on our findings, GSD is unlikely to warrant immediate veterinary intervention, but continued monitoring is recommended to ensure early detection if GSD-afflicted animals begin to show signs of increased mortality or other adverse effects.

*Key words*: Disease ecology, emerging infectious disease, giraffe skin disease, mortality, prevalence, wildlife.

Emerging diseases can be a significant threat to wildlife, especially for populations experiencing other stressors such as habitat loss and hunting (Daszak et al. 2001; Langwig et al. 2015). However, medical treatment of free-living animals can be logistically difficult and expensive; therefore, understanding mortality effects of emerging diseases can provide a basis for determining whether expensive interventions are necessary.

Giraffe skin disease (GSD) is a disorder of the skin grossly characterized by proliferative, crusty lesions on the posterior forelimbs of adult Masai giraffe (*Giraffa camelopardalis tippelskirchi*), the only subspecies that occurs in Tanzania (Epaphras et al. 2012; Lee and Bond 2016). First reported in 2000 in Ruaha National Park (RNP), central Tanzania, the disease has since spread and has been observed in northern Tanzania >300 km away (Lee and Bond 2016). Etiology of GSD remains undetermined, but unpublished data from tissue samples of 12 immobilized giraffe from RNP suggested a spirurid nematode worm as a potential causative agent, with possible secondary infection of fungi and bacteria (Mpanduji et al. 2011). Further assessments of etiology are needed to establish epidemiology, pathogenesis, and transmission of the disease.

Identifying ecologic correlates of a pathogen can help wildlife veterinarians and managers understand risk factors, and longitudinal data from individually identified animals can reveal mortality effects of disease and inform disease-management strategies. If GSD increases mortality rates among Masai giraffe, veterinarians might consider implementing measures to treat the disease. If no mortality effect is associated with GSD, then immediate intervention may not be necessary, although the disease might have adverse effects on mobility or reproduction.

Pathogens can be harbored in soil (Belden and Harris 2007), and a previous study on GSD posited soil characteristics as a possible ecologic correlate of prevalence (Lee and Bond 2016). We hypothesized that prevalence of GSD would be associated with soil properties such as fertility, pH, and salinity, all of which vary considerably across the geographic range of Masai giraffe. We also hypothesized that severity of GSD would be correlated with mortality rates of affected giraffe.

Sampling sites were located in Serengeti National Park (SNP; 4 sites), Ngorongoro Conservation Area (NCA; 2 sites), Lake Manyara National Park (LMNP), Manyara Ranch Conservancy, Mtowambu Game Controlled Area, Lolkisale Game Controlled Area, Lake Natron Game Controlled Area, Mugumu village, and TNP (Fig. 1). The center of our study areas is Tarangire Ecosystem at 3°44′S, 36°0′E.

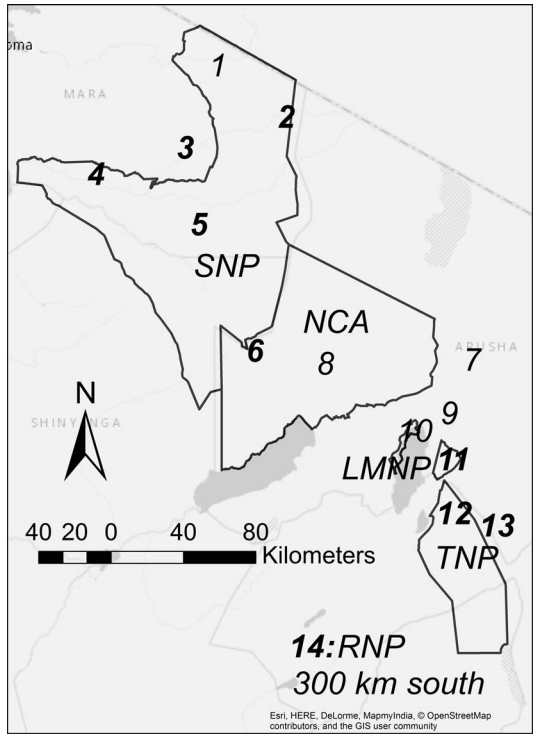


FIGURE 1. The prevalence of giraffe skin disease (GSD) in adult, free-ranging Masai giraffe (*Giraffa camelopardalis tippelskirchi*) during 2008–10 and 2015–16 in northern Tanzania, East Africa. Numbers 1–14 correspond to sites in Table 2. Bolded numbers are locations where GSD was present. SNP=Serengeti National Park; NCA=Ngorongoro Conservation Area; LMNP=Lake Manyara National Park; TNP=Tarangire National Park; RNP=Ruaha National Park.

We sampled for GSD prevalence by driving available roads searching for live adult giraffe between November 2015 and February 2016. For each adult giraffe encountered, we noted sex and, if the posterior sides of the front legs were observable with binoculars, we noted whether GSD lesions were visible (positive) or not (negative) and assigned a severity score according to estimated total diameter of all lesions present (no lesions=no symptoms, 1–30 cm=mild, 31–60 cm=moderate, >60 cm or with cracked or sloughing skin=severe). We measured prevalence as number of animals visually determined to be affected divided by total number of animals for which the posterior side of front legs was visible. In two areas of SNP (Kirawira and Bologonja), we collected photographs of the posterior forelimbs each dry season (August–October) from 2008 to 2010 and used these photographs to assess presence and severity of GSD. We also included estimates of GSD prevalence in RNP in central Tanzania as reported by Epaphras et al. (2012) for our analysis of soil correlates.

We examined how soil qualities of fertility (cation exchange capacity [CEC]=CEC as Cmol/kg of the clay fraction of the soil), soil pH, and salinity (ECe as dS/m) correlated with our observed spatial pattern of GSD prevalence. We assigned site-specific values from the Harmonized World Soil Database (Food and Agriculture Organization of the United Nations 2012) and regressed prevalence on soil properties using generalized linear models with a logistic distribution where symptomatic adults were the number of successes and total number of assessed adults was the number of trials. We conducted all statistical analyses of prevalence in R (R Core Development Team 2013).

For survival analysis, we collected longitudinal data on live giraffe during systematic fixed-route road transect sampling for photographic capture–mark–recapture (PCMR) in TNP. We conducted 18 surveys collecting PCMR data between January 2012 and October 2014. We sampled on three occasions per year by driving 246 km of road transects (Lee et al. 2016). We surveyed with a robust design (Pollock 1982; Kendall et al. 1995). Each occasion included two sampling events during which we surveyed all road transects (3 occasions/year × 2 events/occasion × 3 yr = 18 events). By the second year nearly all resident adult animals available for capture had been detected.

During PCMR sampling, individuals we encountered were "marked" or "recaptured" by photographing the animal's right side. We attempted to photograph every giraffe encountered and recorded sex, GPS location, age class, and GSD status. If the posterior side of the front legs was observable (by eye with binoculars), we noted whether GSD lesions were visible or not and assigned a severity score as described earlier. We identified individual giraffe using their unique and unchanging coat patterns (Foster 1966). We matched giraffe images using WildID (Bolger et al. 2012) and created individual encounter histories for adult male and female giraffe for survival analysis in program MARK 7.1 (White and Burnham 1999).

We used encounter histories to model and estimate survival and nuisance parameters with Pollock's (1982) robust design statistical models. We estimated adult apparent survival probabilities (S) and nuisance parameters of capture (p), recapture (c), and temporary emigration probabilities ($\gamma'$ and $\gamma''$). The model is described in detail by Kendall et al. (1995, 1997). All giraffe were assigned to one of four disease groups (no symptoms, mild, moderate, or severe) based on median severity score of all lesions on the individual over all observations. We performed survival model selection using Akaike's Information Criterion following Burnham and Anderson (2002). We began with the most-fully parameterized model in our set with constraints on survival (constant survival) and with both temporal and group effects in capture, recapture, and temporary emigration rates. We first ranked competing models with reduced temporal complexity of temporary emigration, then detectability parameters. Once a parsimonious form of nuisance parameters was obtained, we ranked three models of survival: a null model with no effects of disease on survival; a presence–absence model where all symptomatic giraffe were compared to all nonsymptomatic; and a group model where each severity group had distinct survival probabilities.

We assessed disease prevalence for 842 individual giraffe in 13 sites in northern Tanzania and included previously reported data from 109 giraffe in RNP (Table 1). Disease prevalence was negatively correlated with soil fertility measured as CEC (Fig. 2; $t$=–3.9, $P$=0.002, $r^2$=0.56).

We estimated survival from encounter histories for 382 giraffe in TNP of which 78 were nonsymptomatic and 127 had mild, 91 had moderate, and 86 had severe lesions. Model selection indicated that the null model of no effects of GSD on survival best fit the data (Table 2). In models that included GSD effects, survival estimates were similar or slightly higher in GSD groups relative to nonsymptomatic groups.

Spatial variation in GSD prevalence was best explained by soil fertility, measured as CEC. If parasites such as nematodes are involved in the pathogenesis of GSD, differences in soil fertility may influence ground-dwelling life stages. Alternatively, soil characteristics may impact nutritional status of giraffe through vegetation quality, thereby altering their susceptibility to GSD. As this disease was first detected and reported only 15 yr ago, it is possible climactic or anthropogenic factors may have led to the spread of a native pathogen or, alternatively, to the introduction and establishment of an invasive pathogen.

We found no mortality effect of GSD on adult giraffe in TNP, indicating that currently GSD is unlikely to warrant immediate veterinary intervention in this park. We did not examine effects on reproduction or movement from the presence of GSD lesions. However,

TABLE 1. Prevalence (proportion adult animals visually symptomatic) of giraffe skin disease in free-ranging Masai giraffe (*Giraffa camelopardalis tippelskirchi*) at 14 sites in northern and central Tanzania, East Africa, and severity of infection at eight sites. Sites are ordered by latitude, north to south; na indicates data not available.

| Site[a] | Site | *n* | Prevalence | Mild | Moderate | Severe |
|---|---|---|---|---|---|---|
| Kogatende (SNP) | 1 | 27 | 0.00 | 0.00 | 0.00 | 0.00 |
| Bologonja (SNP) | 2 | 34 | 0.15 | 0.15 | 0.00 | 0.00 |
| Mugumu (MV) | 3 | 4 | 0.50 | 0.50 | 0.00 | 0.00 |
| Kirawira (SNP) | 4 | 48 | 0.42 | 0.33 | 0.08 | 0.00 |
| Central Serengeti (SNP) | 5 | 72 | 0.49 | 0.40 | 0.08 | 0.00 |
| Ndutu (NCA) | 6 | 21 | 0.19 | 0.19 | 0.00 | 0.00 |
| Engaruka (LNGCA) | 7 | 6 | 0.00 | 0.00 | 0.00 | 0.00 |
| Crater slope (NCA) | 8 | 23 | 0.00 | 0.00 | 0.00 | 0.00 |
| Selela (MGCA) | 9 | 11 | 0.00 | 0.00 | 0.00 | 0.00 |
| Lake Manyara (LMNP) | 10 | 50 | 0.00 | 0.00 | 0.00 | 0.00 |
| Manyara Ranch (MRC) | 11 | 122 | 0.12 | 0.09 | 0.03 | 0.00 |
| Tarangire (TNP) | 12 | 382 | 0.79 | 0.33 | 0.24 | 0.23 |
| Lolkisale (LGCA) | 13 | 42 | 0.69 | 0.12 | 0.38 | 0.19 |
| Ruaha (RNP) | 14 | 109 | 0.92 | na | na | na |
| Total | | 951 | | | | |

[a] SNP = Serengeti National Park; MV = Mugumu Village; NCA = Ngorongoro Conservation Area; LNGCA = Lake Natron Game Controlled Area; MGCA = Mtowambu Game Controlled Area; LMNP = Lake Manyara National Park; MRC = Manyara Ranch Conservancy; TNP = Tarangire National Park; LGCA = Lolkisale Game Controlled Area; RNP = Ruaha National Park.

reproductive rate in TNP was similar to LMNP where GSD has not been documented (Lee et al. 2016), so it is unlikely that GSD causes a large effect on reproduction. Movement of infected giraffe remains an unexplored aspect of GSD effects, but limited mobility could lead to lower survival or reproduction if climate, habitat, or predation factors change from current conditions.

Giraffe are a preferred prey for lions (*Panthera leo*; Hayward and Kerley 2005). In SNP, predation by lions was a minor source of adult giraffe mortality (Schaller 1972; Strauss and Packer 2013) and lions rarely kill adult giraffe in TNP (B. Kissui unpubl. data), but anecdotal observations in RNP indicate lions regularly kill and consume adult giraffe there (M. Ryen pers. comm.). Giraffe in both RNP

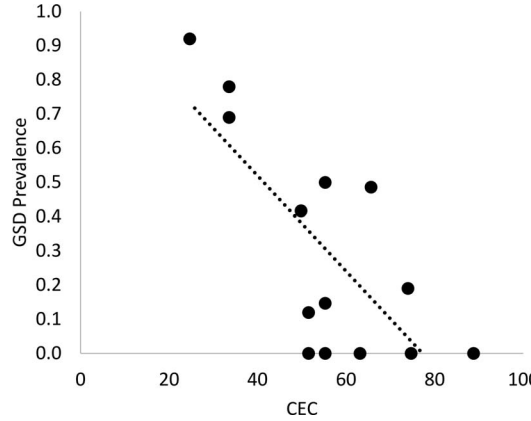


FIGURE 2. The correlation between giraffe skin disease (GSD) in free-ranging Masai giraffe (*Giraffa camelopardalis tippelskirchi*) and soil fertility as measured by cation exchange capacity (CEC) of the clay fraction of the soil in Tanzania 2008–16.

TABLE 2. Model selection results for generalized linear model of giraffe skin disease effects on survival of free-ranging Masai giraffe (*Giraffa camelopardalis tippelskirchi*) in Tarangire National Park, Tanzania.

| Model | ΔAICc[a] | W[b] | k[c] | Deviance |
|---|---|---|---|---|
| Null – no effect | 0 | 0.68 | 81 | 6761.2 |
| Present/absent | 1.69 | 0.29 | 82 | 6760.7 |
| Severity-specific | 5.99 | 0.03 | 84 | 6760.6 |

[a] Corrected Akaike's Information Criterion.
[b] Model weight.
[c] Number of parameters in model.

and TNP suffer high rates of GSD (Table 2); it is possible that differences in pride size and predatory behavior of lions between the two parks have enabled lions to exploit GSD-related limitations in mobility of giraffe in RNP. However, demographic data are not available from RNP to determine effects of GSD on survival rates. Continued monitoring in TNP is recommended to ensure early detection if GSD-afflicted animals begin to show signs of increased mortality or other negative effects, and individual-based demographic research in RNP would provide information about vulnerability of afflicted giraffe to predation.

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