



# Demography and health of “village dogs” in rural Western Uganda



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## ABSTRACT

“Village dogs” in developing economies are assumed to be heavily burdened by infectious disease. We followed a cohort of 61 village dogs in rural western Uganda prospectively for fifteen months to measure changes in health and demographic outcomes, and to examine risk factors for morbidity and mortality. The mean ( $\pm$ standard deviation) number of dogs per household was 2.4 ( $\pm$ 2.0), of which 56.0% were male and 44.0% female. For females, average age at first estrus was 1.7 ( $\pm$ 0.6) years with a mean litter size of 3.8 ( $\pm$ 1.5). In the first, second and third parities, average puppy mortality per litter was 3.2 ( $\pm$ 2.5), 2.4 ( $\pm$ 2.1) and 3.4 ( $\pm$ 2.9), respectively. The main causes of morbidity and mortality were infectious disease (46.1%), culling (euthanasia) by owners (30.8%), and attacks by baboons, *Papio anubis* (23.1%). Cox proportional hazard regression showed that a clinical diagnosis of anemia significantly predicted morbidity (HR = 4.3 (95% CI: 1.1–17.8);  $p < 0.05$ ), and younger age significantly predicted mortality (HR = 3.6 (95% CI: 1.2–10.6);  $p < 0.05$ ). Our results indicate that infectious disease is indeed important to the health and survival in village dogs in this setting, but that cultural practices related to ownership and interactions with wildlife also contribute substantially to morbidity and mortality.

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## 1. Introduction

“Village dogs” (dogs in rural communities in developing economies) are generally believed to suffer a high burden of infectious disease. Dogs clearly play a role in the transmission of diseases important to human and wildlife health (e.g. MacPherson et al., 2000; Lembo et al., 2010; Day, 2011). For example, a long-term study in the Serengeti ecosystem in Tanzania identified domestic dogs as helping to maintain rabies in local human, livestock and wild carnivore populations, as well as distemper in wild carnivores (Cleaveland et al., 2000, 2007; Fitzpatrick et al., 2012).

Despite a growing body of important research, the basic causes of morbidity and mortality in village dogs have remained understudied. Village dogs are integral parts of human communities (Cleaveland and Dye, 1995; Butler and Bingham, 2000; Windyaningsih et al., 2004; Gsell et al., 2012). Consequently, human activities and behaviors related to dog ownership contribute significantly to the demography and health of village dogs (Beran and Frith, 1988; Denduangboripant et al., 2005; Zinsstag

et al., 2009; Talbi et al., 2010; Townsend et al., 2013). In addition, most studies of dog populations in developing countries have been cross-sectional (Chomel et al., 1987; Kitala et al., 2001; Pal, 2001; Hampson et al., 2009). Fewer studies have followed cohorts of dogs to understand drivers of health and demographic outcomes (Morters et al., 2014).

We followed a cohort of 61 dogs in rural Western Uganda prospectively for fifteen months to measure changes in demographic outcomes and health status. Our objectives were to describe the demographic characteristics of the canine population and to identify infectious and other factors associated with morbidity and mortality.

## 2. Materials and methods

### 2.1. Study area

The study was conducted in two communities (Bugembe and Isunga) near Kibale National Park, Western Uganda (0° 13'–0° 41' N, 30° 19'–30° 32' E; Chapman et al., 2005). This region is known for its high human population growth rate and consequently high frequency and intensity of interaction among people, wildlife and livestock (Paige et al., 2014). Cross-species transmission of infectious agents has been documented in this area, including novel and

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potentially zoonotic viruses such as Hepatitis A virus, arteriviruses, pegiviruses, and lentiviruses (e.g. Goldberg et al., 2009; Lauck et al., 2013; Thurber et al., 2013; Ghai et al., 2014a,b; Lauck et al., 2014; Sibley et al., 2014; Bennett et al., 2016).

## 2.2. Study population

Households were selected at random from among those enrolled in a long-term study of human health. Within selected households, all adult and subadult dogs were enrolled. Dogs were implanted dorsally between the shoulder blades with subcutaneous microchips (HomeAgain, Merck Animal Health, Kenilworth, NJ, USA), which were used to confirm the identities of dogs throughout the study.

Following informed consent, an owner questionnaire was completed for every dog in the cohort by a trained, licensed Ugandan veterinarian (DH). Information was obtained on age, acquisition, nutrition, vaccination and deworming history, prior disease events, reproductive history, and frequency of interaction with conspecifics, other domestic animal species, and wildlife. For females, reproductive data were collected, including the age at first estrus, parities, average litter sizes, and puppy mortality per litter.

Households were visited subsequently every 2–4 months over a period of approximately 15 months. During follow up visits, owners were re-interviewed using an abbreviated survey instrument that captured any health or demographic events that had transpired since the last visit (e.g. illness, immigration, emigration, births, and deaths).

## 2.3. Health evaluation

At the time of enrollment, each dog was given a physical examination by a licensed Ugandan veterinarian (DH). During subsequent household visits, dogs were examined for physical abnormalities and to score body condition (on an ordinal scale of 1–5). Any dogs showing signs of illness or reported to be ill underwent a full physical examination.

Venous blood (1–2 ml obtained via needle and syringe from the cephalic vein), feces (obtained per rectum using a gloved finger), and ectoparasites (obtained using a comb and forceps) were collected from all dogs that underwent physical examination. Blood was used to make thin smears on glass slides for microscopic examination of hemoparasites, and the remainder was placed in evacuated EDTA tubes (BD Vacutainer, Becton, Dickinson and Company, Franklin Lakes, NJ, USA) for measurement of packed cell volumes and manual hemocytometry. Feces obtained *per rectum* were placed in 10.0% formalin and examined by microscopy for parasites (identified to the genus level) using direct smears and floatation/sedimentation.

All protocols were approved by the University of Wisconsin-Madison and the Uganda National Council for Science and Technology prior to collection of data.

## 2.4. Data analysis

Data from both communities were aggregated because of small sample sizes and similar ranges and distributions of demographic and health-related variables. Risk factors for time to death and time to onset of clinical signs were investigated using Cox proportional hazard regression. Backwards elimination of predictor variables was used to select models that retained only significant covariates (at the  $\alpha < 0.05$  level), and final models were chosen when they explained significantly more variance than the null model (Likelihood-ratio test, at the  $\alpha = 0.05$  level), as well as satisfying proportional hazards assumptions (residuals were not significantly correlated with time: Pearson's rho,  $p > 0.05$ ). All

**Table 1**  
Reproductive parameters for females.

Parameter	Mean $\pm$ SD
Age at first estrus (years)	1.69 $\pm$ 0.59
Average litter size (puppies)	3.81 $\pm$ 1.45
Average inter-birth interval (months)	11.13 $\pm$ 4.36
Puppy mortality/litter, parity 1 (%)	84.3 $\pm$ 64.3
Puppy mortality/litter, parity 2 (%)	63.0 $\pm$ 55.2
Puppy mortality/litter, parity 3 (%)	89.2 $\pm$ 76.7
Puppy mortality/litter, parity 4+ (%)	65.6 $\pm$ 48.6

analyses were performed with the *surv*, *coxph*, and *cox.zph* functions in the Survival package of R version 3.3.0 (R Core Team, 2014).

## 3. Results

We enrolled 61 dogs in 26 households (mean of  $2.4 \pm 2.0$  standard deviations dogs per household), of which 34 were male and 27 were female, 42 were adults (sexually active) and 19 were subadults (not yet sexually active). Females produced approximately four puppies per litter, with puppy mortality rates between approximately 60.0% and 90.0%, depending on parity (Table 1). Based on owner interviews and physical examinations, the main causes of mortality were infectious disease (46.2%), culling (euthanasia) by owners (30.8%), and attacks by olive baboons, *Papio anubis* (23.1%).

Owner interviews indicated that 98.4% of dogs were fed human food, including potatoes, cassava, maize, millet, pumpkins, beans, fish and meat. Thirty percent of dogs were fed once per day, and the remaining 70.0% were fed multiple times per day. Owners reported that 80.3% of dogs ate dead livestock at least once per year, and 60.7% of dogs ate wildlife that they hunted or scavenged. All dogs interacted daily with other dogs, and 80.3% of dogs had contact with wildlife every week. Physical examinations and subsequent testing of clinical samples revealed a diversity of illnesses, with parasitism and hematological parameters indicative of infection being the most common (Table 2).

During the 15-month study period, 13 dogs died, 8 dogs emigrated and/or were lost to follow-up, and 9 dogs developed signs of disease. The crude mortality rate was 1.4 deaths per 100 dogs per month, and the overall incidence of disease was 1.8 incident cases per 100 dogs per month. Median survival time was

**Table 2**  
Frequency distribution of clinical findings on veterinary evaluation.

Finding	Percent of dogs
Gastrointestinal parasites <sup>a</sup>	97.1
Ectoparasites (ticks and fleas)	95.0
Lymphadenopathy (enlarged palpable lymph nodes)	86.7
Coarse fur	77.1
Congested mucosa	60.7
Poor body condition (body condition score $\leq 2/5$ )	54.1
Hyperproteinemia (Total Plasma Protein $> 7.5$ g/dl)	46.3
Abnormal leukogram <sup>b</sup>	44.6
Anemia (hematocrit $< 37\%$ )	38.6
Hemoparasites <sup>c</sup>	31.6
Conjunctivitis	19.7
Fever (rectal temperature $> 102.5$ °F)	18.0
Open wounds	18.0
Splenomegaly and/or hepatomegaly	14.8
Urinary tract disorders	9.8
Rhinitis	4.9

<sup>a</sup> Gastrointestinal parasites included hookworm (82.2%), ascarids (42.2%), *Cryptosporidium* (17.8%), *Isoospora* (13.3%), *Giardia* (13.3%) and cestodes (13.3%).

<sup>b</sup> Abnormal leukograms included eosinophilia (85.2%), leukocytopenia/leukocytosis (42.6%), neutropenia/neutrophilia (36.1%), basophilia (34.4%), monocytopenia/monocytosis (31.1%), and lymphocytosis/lymphocytopenia (27.9%).

<sup>c</sup> Hemoparasites included *Ehrlichia* (75.0%), *Anaplasma* (31.3%), and *Hepatozoon* (12.6%).

**Table 3**  
Cox proportional hazard regression models for time to death (mortality) and time to development of clinical signs (morbidity).

Model <sup>a</sup>	Significant risk factors	$p >  z $	HR <sup>b</sup> (95% CI)
Morbidity <sup>c</sup>	Anemia (reference = normal PCV)	$p < 0.05$	4.33 (1.05–17.84)
Mortality <sup>d</sup>	Age (reference = adult)	$p < 0.05$	3.56 (1.19–10.62)

<sup>a</sup> For both models, the ZPH proportional hazard assumption  $p > 0.05$ , indicating that residuals were not significantly correlated with time.

<sup>b</sup> HR: hazard ratio.

<sup>c</sup>  $N < 5$  for dogs with conjunctivitis, fever, splenomegaly and/or hepatomegaly, urinary tract disorder, rhinitis or without gastrointestinal parasites, ectoparasites, lymphadenopathy or wildlife interactions. These variables were not included as potential covariates in the morbidity model.

<sup>d</sup>  $N < 5$  for dogs with rhinitis, and without gastrointestinal parasites and ectoparasites. These variables were not included as potential covariates in the mortality model.

175 days, and median time to development of clinical signs was 231 days. Cox proportional hazard regression showed that a clinical finding of anemia (packed cell volume  $< 37\%$ ) was a significant predictor of morbidity (HR = 4.3;  $p < 0.05$ ). Age (being younger than adult), was a significant predictor of mortality (HR = 3.6;  $p < 0.05$ ) (Table 3). Interacting with wildlife ( $\chi^2$  (1,  $N = 54$ ) = 3.5,  $p = 0.06$ ) and hyperproteinemia ( $\chi^2$  (1,  $N = 35$ ) = 3.0,  $p = 0.07$ ) were of marginal significance in log rank-tests but did not explain much variation in the overall model. Similarly, receiving veterinary medical care ( $\chi^2$  (1,  $N = 33$ ) = 3.0,  $p = 0.09$ ) was of marginal significance but did not contribute meaningfully to the overall explanation of variance.

#### 4. Discussion

Our data demonstrate that village dogs in rural western Uganda lead “Hobbesian” lives (“poor, nasty, brutish, and short”) (Hobbes, 1996). Only 10–30% of puppies survived, and mortality rates of older age classes were over 1% per month. Clinical condition (e.g. body score) of dogs was overall poor, and prevalence of parasitism approached 100%. Over the course of the 15-month study, we documented a suite of clinical and hematological findings that would be considered well outside the normal range for dogs in the developed world.

A surprising finding was that culling by owners was the second most common cause of mortality after infectious disease. Culling by owners was in response to predation by dogs of household livestock, mainly chickens. This observation underscores the tensions of dog ownership in this setting, in that owners often balance the benefits of owning a dog (principally defense of the household against intruders and wild animals) with the associated costs (economic investment and risk to livestock). Clearly, this balance is precarious from the dogs’ perspective.

Also surprisingly, the third most common cause of mortality was aggressive interactions with baboons. We have previously described antagonistic interactions between dogs and wild primates in this area, but these former observations were of packs of dogs hunting wild arboreal monkeys (Goldberg et al., 2008; Paige et al., 2014). Baboons, being large-bodied and terrestrial, are considered “vermin” by the Ugandan government, due to their crop-raiding propensities (Naughton, 1998), and this behavior clearly leads to antagonism with domestic dogs, with the outcomes not necessarily favoring the dogs. Although our data cannot attribute the open wounds we documented on 18% of dogs (Table 2) to particular causes, we suspect that the majority resulted from baboon attacks. These observations are noteworthy from the perspective of cross-species disease transmission, in that our long-term research shows baboons in the region to be hosts of novel blood-borne viruses (Sibley et al., 2014; Bennett et al., 2016).

Because our study was conducted entirely under field conditions, we were unable to test dogs for specific diseases, such as

rabies or distemper. Dogs clearly play important roles in maintaining such diseases in wildlife and humans elsewhere in Sub-Saharan Africa (Hampson et al., 2007), and we suspect the situation to be no different in Uganda, where human rabies is endemic and dog bites are common (Fevre et al., 2005). Our study differs from previous investigations in that we studied dogs in a forest ecosystem; most previous studies have focused on grassland ecosystems, such as in the Serengeti ecosystem of Tanzania (Cleaveland et al., 2007; Fitzpatrick et al., 2012).

The generality of our study is unclear, and our sample sizes were too small to construct formal demographic projection models. Nevertheless, our results reveal unexpected drivers of canine survival and health, revealing conditions that may apply to village dogs in similar ecological settings across sub-Saharan Africa. Overall, our data portray dogs in Western Uganda as unsurprisingly unhealthy but surprisingly pressured by antagonistic interactions with their owners and wildlife.

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