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The Importance of Wildlife Harvest to Human Health and Livelihoods  
in Northeastern Madagascar

By

Christopher DeWeir Golden

A dissertation submitted in partial satisfaction of the requirements for the degree of

Doctor of Philosophy

in

Environmental Science, Policy and Management

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

Professor Claire Kremen, Chair

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Madagascar

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## ABSTRACT

### The Importance of Wildlife Harvest to Human Health and Livelihoods in Northeastern Madagascar

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Professor Claire Kremen, Chair

Global environmental transformation has triggered well-studied impacts on ecological interactions and biodiversity but the effects of damaged ecosystems and diminished access to natural resources on human health is less understood. Mammalian wildlife not only influences ecological systems but also serves as a primary source of meat to hundreds of millions of people throughout the developing world. The consumption of animal-source foods is important in providing essential micronutrients to humans in bioavailable forms. Thus, loss of access to wildlife may have powerful and far-reaching effects on the health of human populations that rely on bushmeat. In this dissertation, I evaluated the provisioning service value of wildlife populations to human health and livelihoods, using methods from ecology, economics and epidemiology.

Ecosystem service valuations are useful to conservation and development practitioners for calculating the total value of benefits from ecosystems, choosing between alternative land use or management scenarios, ascertaining the distribution of the costs and benefits of services to users, and identifying or developing financing mechanisms for ecosystem services. I use the case of wildlife harvest and consumption in northeastern Madagascar to identify the distribution of these benefits to further understanding of local incentives for conservation promotion and conservation rule-breaking. Through my analysis of the disproportionate benefits of these services to local people, conservation practitioners may better understand the heterogeneity of local people's incentives for conservation. These fine-scale differences between communities and households can also be used to create frameworks for policy linkages so that public health and development initiatives can best allocate sparse funds to support regions, households or individuals most vulnerable to changes in access to wildlife. This valuation framework highlights the local importance of this service, but still ignores the cultural, ecological and health value of

wildlife and wild meat to local people, and the existence value of this biodiversity to the global community.

Despite widespread human reliance on wildlife for food, the impact of wildlife depletion on human health remains poorly understood. I studied a longitudinal cohort of 77 pre-adolescent children in Madagascar and show that consuming more wildlife was associated with significantly higher hemoglobin concentrations. My empirical models demonstrate that removing access to wildlife would induce a 29% increase in the numbers of children suffering from anemia and a tripling of anemia cases among children in the poorest households. This research quantifies costs of reduced access to wildlife for rural communities in Madagascar and provides evidence linking global trends in biodiversity loss to declines in childhood health.

This study linking anemia to wildlife availability and access seeks to provide an improved understanding of the ultimate determinants of anemia, a widespread and critically important nutritional disorder. Anemia is a disease characterized by a deficiency in red blood cells, hemoglobin (an iron-containing protein whose main function is oxygen transportation in humans) or both. Although there are multiple etiologies, the majority of anemia cases worldwide are a result of iron deficiency, and this is the most common nutritional disease globally affecting one-fourth of the world's population. Because of the critical role of iron in metabolic processes, reductions in hemoglobin on an individual and population level may induce a breadth of health sequelae from nearly all health domains. I use results from a systematic review of the literature to describe the potential short- and long-term effects of iron-deficiency anemia in two ways: 1) by linking shifts in hemoglobin to dose-response health effects using previously published studies and then 2) demonstrating the impact on health and economies of predicted increases in the future prevalence of anemia in my study population.

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"The world is as delicate and as complicated as a spider's web. If you touch one thread you send shudders running through all the other threads. We are not just touching the web, we are tearing great holes in it" –Gerald Durrell

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# CHAPTER 1

## General Introduction

There is significant global congruence between priority areas lacking basic human health conditions and hotspots for biodiversity. This overlap highlights the need to simultaneously address rapid declines in biodiversity and natural habitat and the persisting prevalence of human disease and food insecurity. Less well understood is that in many developing countries human health depends on the presence of intact environments that provide ecosystem services that benefit human health and well-being. These ecosystem services deliver benefits to humans in myriad ways but are broadly categorized into regulating (water purification, pollination, and the moderation of climate and disease), cultural (recreation, ecotourism, spiritual and religious attachment, and inspirational and innovational development), provisioning (water, fuelwood, food and medicines) and supporting services (the soil formation, nutrient cycling and primary production that underlie all other services, MEA 2005).

Although there are theoretical models of the ways in which the natural environment may affect human health, the mechanisms of these relationships have rarely been empirically studied. In this dissertation, we use the case of wildlife in the Makira Protected Area (MPA), Madagascar to better understand the value of natural resources to human health and livelihoods. We do this by estimating the ecosystem service value of wildlife as a harvested meat, analyzing the linkages between wildlife consumption and human nutrition, and projecting the anticipated health effects of removing access to wildlife. In the first chapter, we use an ecosystem services framework to estimate the variation and distribution of the provisioning service value of harvested wildlife to better understand local attitudes toward conservation and to identify those groups who would be most vulnerable to perturbations in this service. In the second chapter, we use methods from nutritional epidemiology and a prospective cohort study design to determine the association between individual wildlife consumption and hemoglobin concentrations (a measure of anemia) in a group of rural, Malagasy pre-adolescents. The third chapter is divided into two cohesive components. First, we conduct a review of the literature that links anemia as a risk factor to consequential health outcomes. This literature tends to show this relationship as either a dose-response effect between hemoglobin concentrations and degree of illness/impairment or as a difference between groups (healthy vs. anemic). We then merge our empirical results on the relationship between bushmeat consumption and hemoglobin concentrations to the models presented in the review to develop projections of future population health in the MPA.

This dissertation is situated at the confluence of global environmental change, food security and human health- subjects that are inextricably interrelated. This relationship is particularly worrisome because the past century has witnessed stark increases in pressures on biodiversity such as resource consumption, invasive alien species, nitrogen pollution,

overexploitation, and climate change impacts (Butchart et al. 2010), accompanied by unprecedented rates of biodiversity decline (MEA 2005). Recently, much emphasis has focused on the impacts of climate change on biodiversity declines (Thomas et al. 2004, Araujo and Rahbek 2006, Heino et al. 2009); however, these impacts are less pressing than the more direct effects of deforestation and overexploitation.

Madagascar is the fourth largest island in the world and the largest oceanic island. The island boasts high rates of diversity and endemism. Approximately 10,000 to 12,000 plant species are found in Madagascar, 85% of which are endemic, and 209 bird species reside in Madagascar, with a striking 52% endemism (Goodman and Benstead 2005). Of Madagascar's 199 amphibian species (only frogs), 99% are endemic to the island while of the 340 reptile species in Madagascar, 92% are endemic (Goodman and Benstead 2005). Ninety percent of the 131 Malagasy mammals are endemic to the island and if one focuses solely on non-volant mammals, endemism approaches 100% (Goodman and Benstead 2005). Although no new endemism estimates have been formally published, incredible numbers of species have been discovered in Madagascar since 2005 (Mittermeier et al. 2010). In Madagascar generally, the high levels of highly isolated and fragmented endemism in Madagascar may predict significant global rather than strictly local extinction rates.

In Madagascar, Dewar (1984) considered the two predominant views of extinctions, which wiped out 7 of 17 genera of lemuroids (prosimian primates), both genera of ratites (flightless birds), three species of non-primate mammals, and two species of large land tortoises. He first presented the theory of widespread desiccation that he believed was not supported by climatic data which he found improbable because so many different ecological zones were affected and only the megafauna was targeted (Dewar 1984). The extinction of the strictly *diurnal* megafauna seems to be most likely explained by the arrival of human hunters (Dewar 1984). According to Wright and Rakotoarisoa (2003), the history of hunting animals to extinction in Madagascar dates back to A.D. 100-300, evidenced by the discovery of pygmy hippopotamus and other subfossil bones that had been cut with iron tools. However, it is unlikely that all of the extinctions were exclusively caused by hunting. Dewar (1984) suggested that the widespread extinctions were likely induced by a combination of hunting, competition, and habitat destruction and fragmentation, with hunting as the major causal factor.

Historically, Malagasy faunal extinctions have not been attributed to a unicausal source, whether it is hunting, habitat loss, or climate change (Godfrey & Irwin 2007). However, Godfrey and Irwin (2007) prioritized threats and considered the relative importance of hunting as a threat to species viability to have declined in the present atmosphere in Madagascar, even though it may have been the greatest factor in driving megafaunal extinctions during the Holocene. More recently in Madagascar, deforestation has emerged as the critical issue that encapsulated debates over land protection and local populations' land use, demonstrating that approximately 10% of original forest extent remains (Sussman et al. 1994, Harper et al. 2007). It has been suggested that deforestation is the major factor in species extinction risk, even when focusing on primates

(Ganzhorn et al. 2001), which are typically more sensitive to hunting than other mammals. Approximately 9% of species in Madagascar have been “committed” to extinction because of the deforestation that occurred between 1950-2000 (Allnutt et al. 2008) and the temporal lag (n.b. extinction debt literature, Tilman et al. 1994, Hanski and Ovaskainen 2002, Helm et al. 2006) that delays extinction until the effects of resource competition and reduced breeding lead to extinction. Other estimates point to a 50% reduction in species even though 10% of remaining habitat remains (Hanski et al. 2007).

Although an understudied issue in Madagascar currently, my recent work, among others, has highlighted the incredibly damaging effect of overexploitation to mammalian species persistence (Perez et al. 2002, Garcia and Goodman 2003, Goodman 2006, Cardiff et al. 2009, Golden 2009, Randrianandrianina et al. 2010). Since the political crisis in early 2009 when a military-supported coup d'état ousted former President Marc Ravalomanana, localized monitoring and enforcement of environmental legislation degenerated and there were sharp increases in commercial hunting and other forms of consumer-driven resource extraction in particular areas (Barrett and Ratsimbazafy 2009). This state of dismantled protected areas and lack of local monitoring and enforcement continues into the present. However, the increased commercially-driven extraction does not equal the magnitude of the large-scale subsistence extraction that is driven by necessity to provide food to local inhabitants.

The connections that we make between biodiversity, resource conservation and human nutrition are novel. In fact, the general framework for the field of environmental health science traditionally focuses on the aspects of the environment that produce negative health outcomes (i.e. toxins, pollution and occupational health). In this same vein, many studies have investigated the ways in which exposure to biodiversity is a risk factor for the incidence of infectious diseases like zoonoses and other vector-borne illnesses (Hahn et al. 2000, Tenter et al. 2000, Levett 2001, Wolfe et al. 2004, Wolfe et al. 2005, Reperant 2010). On the other hand, there has been other research that has empirically illuminated relationships between environmental change and infectious diseases (Allan et al. 2003, LoGiudice et al. 2003, Brownstein et al. 2005, Ezenwa et al. 2006, Vittor et al. 2006, Pattanayak and Wendland 2007, Allan et al. 2009, Disney and Ruedas 2009, Pongsiri et al. 2009, Salkeld and Lane 2010), implying that a protected environment is beneficial to human health.

A healthy environment can not only protect humans against the incidence of infectious diseases but environments with a full suite of pollinators can also prevent declines in crop yields (Allen-Wardell et al. 1998, Kremen 2008). Both agriculture (Ellis 1998, Welch and Graham 1999, Rosegrant and Cline 2003) and wild plant foods (Grivetti and Ogle 2000, Shackleton 2003) in developing countries are mainstays of local diets and hugely reliant on native pollinators (Bawa 1990). Without fully functioning environments, agriculture and wild plant foods would not be able to provision local residents with both macro- and micronutrient rich foods. In fact, crop plants that depend fully or partially on animal pollinators contain more than 90% of the Vitamin C, all of the Lycopene, and almost all of the vitamin A and calcium (Eilers et al. 2011).

In this dissertation, we provide empirical evidence of the link between wildlife populations and human health, and the threat of either biodiversity loss and wildlife depletion or strictly enforced conservation regimes that do not provide feasible alternatives to freely accessed natural resources. Often, wildlife is viewed as a risk factor for human health via zoonoses (Wolfe et al. 2005); however, hundreds of millions of people in the developing world use wildlife as a primary source of meat (Brashares et al. in press). This creates a tension between biodiversity conservation and human health. Here we demonstrate the economic and health value of the provisioning service of wildlife to local people living adjacent to the Makira Protected Area, Madagascar. It is our hope that by highlighting the inextricable ties between biodiversity conservation and human health we may increase the constituency for both those with public health and environmental interests to jointly tackle these pressing issues.

## CHAPTER 2

### The Provisioning Service Value of Wildlife Populations in Madagascar

#### Introduction

Quantifying the services provided to humans by ecosystems has become a major area of research within ecology, economics, and conservation biology (Costanza et al. 1997, Daily et al. 1997). One of the primary uses for valuing ecosystem services is to identify who wins and who loses under different forms of land management, and how benefits and costs are distributed among stakeholders across scales (Kremen et al. 2000, Pagiola et al. 2004, Farley & Costanza 2010). Through fine-scale analyses of the distribution of costs and benefits to local users, the conservation community can better understand incentives for conservation promotion and rule-breaking behavior. Further, through empirical demonstration of the effects of a given policy option, ecosystem service analyses can inform eligibility criteria for economic development and public health targeting to most efficiently support those most affected by disruptions or restrictions to the service.

The widespread harvest of wildlife for human consumption is a multi-billion dollar provisioning service that provides benefits to tens of millions of rural poor (Bodmer et al. 1994, Balmford et al. 2002, Milner-Gulland et al. 2003). In areas of Africa where the majority of harvested wildlife is sold (e.g., more than 90% of bushmeat in rural households in the Democratic Republic of Congo, de Merode et al. 2004), studies of the value of bushmeat commonly use market analysis rather than non-market valuation techniques. In other areas, researchers have used market reports in conjunction with wildlife utilization surveys to determine the national or regional value of bushmeat harvested each year (Afolayan 1980, Anadu et al. 1988, Antsey 1991, Steel 1994, Chapman & Peres 2001, Refisch & Kone 2005). Each of these approaches struggles to account for the significant proportion of harvested wildlife in developing countries that is consumed locally and never is integrated into either cash or non-cash markets (Robinson & Bennett 2000). Thus market-based studies of wildlife use evaluate only the partial flow (i.e. the harvest that actually arrives at market) and ignore the often large proportion of subsistence value due to local consumption.

As in many developing countries, wildlife in Madagascar is a major nutritional resource and significant to the livelihoods of rural communities (Golden 2009). Yet, because there is often no formal commercial market for this commodity, its monetary value and importance as an ecosystem service is often overlooked. In addition to its value as a provisioning service, mammalian wildlife in Madagascar also provides regulatory, cultural and supporting services (sensu MEA 2005). For example, frugivorous and nectarivorous bat and lemur species serve a regulatory function in maintaining forest floral diversity through their role as seed dispersers and pollinators (Dew & Wright 1998). Many of the insectivorous bat and carnivorous species also are natural predators of insects, snakes, and rodents that impact local agriculture and livestock. Mammalian wildlife also has aesthetic and economic value in its ability to attract foreign ecotourists and global intrinsic value as comprising members of endemic and often threatened biodiversity (Goodman & Benstead 2005).

The provisioning service of wildlife can be conceived as both a productive fund service and as a stock resource (sensu Farley & Costanza 2010). The productive fund service is the self-generating reproductive capacity or the maximum sustainable yield of the wildlife population. As a productive fund service, wildlife cannot be stockpiled, their rates of increase cannot be controlled and they are not physically converted into what they produce. The stock resource is the dynamic reservoir of wildlife available for human harvest. As a stock resource, the reproductive capacity can be converted into meat and humans control this rate of transformation with possibilities of stockpiling the resource by drying or smoking. In this study, we use basic valuation techniques to value the flow of the stock resource of mammalian wildlife to characterize the distribution of benefits between villages and households in Madagascar's Makira Protected Area (MPA, Fig. 1). Characterizing the flow of benefits is important for two primary reasons: 1) to better illuminate local people's incentives for resource conservation or rule-breaking behavior, and 2) to create eligibility criteria based on those most at risk from land-use changes (including protection) to efficiently target and allocate development and public health support. In this way, communities or households most vulnerable to changes in access to wildlife can be supported prior to anticipated changes in dietary nutrition and economic livelihoods.

## **Methods**

### *Study Site*

The Makira Protected Area (MPA) in north-eastern Madagascar covers 371,217 ha and is characterized by lowland and mid-altitude rainforest. It is one of the nation's largest remaining blocks of contiguous forest and contains high levels of biodiversity (Golden 2009). Two ethnic groups are common locally, with Betsimisaraka in the east and south, Tsimihety in the north and west, and a mixing where these regions overlap (Golden 2009). We studied 26 villages that had geographic access to the MPA, chosen to represent both geographic and cultural diversity. We selected villages regionally by following trade routes so that we could observationally track the flow of bushmeat harvest and consumption.

### *Bushmeat Consumption*

Semi-structured interviews were conducted in 481 households in 26 villages from January-December 2007 throughout the study area (Fig. 1). Villages were grouped into previously established travelling or trade routes and then randomly cluster-sampled. Households within selected villages were identified for surveys using systematic random sampling techniques and were recruited over a period of several years starting in 2004. We surveyed 10-100% (mean 22%, median 41%) of village households in each of the 26 villages included in our study (Appendix 2.1). There was no statistical trend between the proportion of households sampled within a village and larger standard deviations of village bushmeat consumption or village annual income (Appendix 2.1).

During each interview, we asked the head of household to quantify household characteristics and the dynamics of wildlife harvest in the study area. Household annual income was measured by adding the value of products sold, wages earned and items bartered but did not include the subsistence consumption of other provisioning services (e.g. tubers, honey, wood for construction, etc., see Kremen et al. 1999). We also obtained the total number of individuals of each of the 23 locally-occurring mammalian bushmeat species that were consumed by the household during the previous year (Golden 2009, see Brashares et al. in press for validation of method). Total harvest for each village was then determined by summing the harvest of each species for all surveyed households and extrapolating to the total number of households within sampled villages. A range of total biomass harvested for each species was calculated by multiplying the extrapolated total annual harvests of each species by the range of adult body mass (Garbutt 2007). Use of adult biomass may produce a slight overestimation of total biomass harvested as not all individuals within species that are harvested were adults.

### *Bushmeat prices*

Building on previous work that used proxies of livestock meat prices, urban market prices or a flat rate for bushmeat to estimate wildlife value (e.g. Bodmer et al. 1994, Naidoo & Ricketts 2006), we used purchasing prices specific to each species harvested to estimate the total value of harvested wildlife. Although there are no reports of formal commercial markets for bushmeat in Madagascar, likely because the majority of mammal species are illegal to hunt throughout the nation, animal carcasses are occasionally sold household to household, making it possible to develop an index for a local pricing structure. Because price information was collected locally in each village, these prices were not skewed by long-distance transportation costs to urban markets and the ratcheting effects of brokers. Although prices obtained were both locally and species-specific, prices for stock resources such as this are still affected by the imperfect nature of markets (see Discussion).

Local prices of bushmeat species were reported during household interviews when interviewees had purchased rather than hunted individual animals (Appendix 2.2). Although much of the harvest is illegal, we are confident that the prices and reports are accurate because of our long-term presence in these communities with assistants living on site throughout the year. The reported price per species was averaged across all villages in the sample and then divided by the minimum and maximum range of adult animal body mass to determine a regional price per kilogram. The bush pig, *Potamochoerus larvatus*, is typically sold by the kilogram so no conversion was necessary. We used a conversion rate of 1,700 ariary per dollar to convert the local prices to US dollars. Though currency exchange rates fluctuate widely, the price of bushmeat in local Malagasy currency remained stable during the study (unpublished data).

### *Bushmeat hunting effort*

The annual opportunity cost of hunting (C) was calculated as:

$$\text{Equation 1: } C = \sum_{i=s}^n M_s * (Y_s * T_s * W)$$

where  $M_s$  was the total number of households participating in each hunting activity,  $Y_s$  was the total number of days per year engaged in each hunting activity,  $T_s$  was the average number of hours devoted to each type of hunting activity and  $W$  was the wage labor rate per hour. The proportion of households participating in each hunting activity was then extrapolated to the total number of households across all 26 surveyed villages. The opportunity cost of time allocated to hunting activities was estimated through participant-observation during hunter follows conducted over several years (2004-09) to calculate the time investment required for particular hunting activities (Appendix 2.2). Twenty-seven hunters were followed on 76 hunting days (mean 2.8 follows per hunter, SD = 1.9). Subjects were selected for follows through the development of close relationships over four years to minimize the effect of an outside observer (i.e. the Hawthorne effect). Bushmeat hunting was categorized into the following nine activities: bat night hunting, bush pig snare construction, bush pig snare checking, dog hunting of bush pigs and tenrecs, lemur snare construction, lemur snare checking, carnivore snare construction, and carnivore snare checking. Handling time of captured animals was minimal and thus not included as a separate cost category. A 30% travel time discount was applied to lemur snare checking as hunters almost always passed by their agricultural fields for work before arriving at the snare locations. This 30% discount represents the approximate one-hour round trip (from the village to agricultural fields) which was excluded from hunting time allocation costs. Observations of these nine activities allowed us to determine the average number of hours ( $T_s$ ) devoted to each type of hunting activity per day (Appendix 2.2).

The total number of days per year spent participating in each hunting activity ( $Y_s$ ) was determined through self-report by participants estimating the number of days per week engaged in each activity and multiplying it by the number of weeks in each season for hunting a particular taxa. The frequency and seasonality of each hunting type was routinely collected during interviews. The total number of hours expended by all hunters across the 26 villages was calculated and multiplied by the minimum amount paid for local labor and then an average annual village time allocation cost was produced.

The labor wage per hour ( $W$ ) was based on men's daily agricultural labor ( $n = 293$  hired-people days). The minimum daily wage was \$0.59, and the median daily wage was \$1.18. An average of 10 hours was worked by these laborers each day, producing an estimated hourly wage of \$0.06-0.12. The lower range of this hourly labor wage was utilized because using agricultural labor rates overestimates the true cost of time allocation since wage labor is highly seasonal and thus overvalued. Annual opportunity costs of bushmeat hunting ( $C$ ) were then calculated by multiplying the number of individuals estimated to be involved in each hunting activity by the cost of allocating this time.

We deemed the material costs of hunting to be negligible, and thus, did not include them in calculating the total costs of bushmeat harvest. The natural materials used to construct snares, traps and nets are plentiful and essentially free. Machetes, slingshots and dogs are typically owned for other reasons than hunting and have minimal maintenance costs. Hunting with firearms, which would have significant costs, is extremely rare in the MPA.



### *Determining the value of wildlife per hectare*

Harvest area was calculated as a circle surrounding a village center, with a mean radius of 4.4 km and a standard deviation of 2.9 km, determined by hunter reports of either distance or time travelled to actively hunt or passively trap lemurs (see Golden 2009 for details). We used geographic information systems (ArcGIS v. 9.3) to obtain the total forested area within the mean radius, hereafter called the “harvest area” (Fig. 1). We assumed the maximum harvest area to be the forested area within the mean harvest radius plus one standard deviation. We did not include other habitat types besides forest since the majority of mammal species included in the analysis (i.e. lemurs, carnivore species, etc.) are heavily forest-dependent (Irwin et al. 2010).

The revenue accrued for all bushmeat biomass harvested ( $R$ ) was calculated by multiplying the total harvested bushmeat biomass for each species ( $H_s$ ) by their respective prices per kilogram ( $P_s$ ):

$$\text{Equation 2: } R = \sum_{i=s}^n H_s * P_s$$

After determining the revenue value of all bushmeat harvested within the 26 villages, we divided this value by the total harvest area across the sampled 26 villages. This provided a dollar value per hectare which is a common unit of comparison in ecosystem service analyses (e.g., Pearce & Moran 1994, Kremen et al. 2000). When the harvest areas of adjacent villages overlapped, the area was not double counted, but, instead, the combined value of bushmeat biomass of villages with overlapping areas was calculated and then divided by the union of the villages’ harvest areas. We extrapolated only to village harvest areas and not to the entire area of the MPA as the majority of central Makira is unharvested because of lack of settlements and traveling paths. We then conducted a cost-benefit analysis to calculate the net value of wildlife harvest ( $V$ ):

$$\text{Equation 3: } V = R - C$$

where  $R$  is the potential revenue accrued for bushmeat harvest (Equation 2) and  $C$  is the opportunity costs of time allocation (Equation 1). We present the revenue value rather than the net value as a proportion of total annual income (Results), however, because the total annual income measure used in this study does not subtract time allocation costs from harvesting other local products.

To model the effect of conservation monitoring and enforcement on the benefits received from wildlife harvest, we recalculated the revenue and flow values after removing the values (and time allocation costs) of species that are illegal to hunt (lemurs and endemic carnivores). This conservation enforcement scenario provides an estimate for the opportunity cost to local economies of biodiversity conservation. We assumed that the enforced hunting restrictions would not cause increases in hunting of legally-hunted species. Although unrealistic, hunting exclusively legally harvested mammals to compensate for lost biomass would likely cause local extinctions; thus our calculation of the opportunity cost of enforcement was in fact conservative because lost access to illegal bushmeat would intensify the hunting of legal bushmeat leading quickly to depletion.

## Results

Our survey of 481 households revealed that the vast majority of bushmeat is consumed by the hunter and his family, with only 7% of consumed bushmeat being purchased, demonstrating a near absence of a formal market for bushmeat in this area. Personal decisions to sell bushmeat are made on a per day basis and not made based on total harvest for a hunting season. Because of a lack of refrigeration and effective preservation methods, bushmeat tends to be sold only if it is too substantial to consume in a day. Therefore, only bushpigs and bats were sold frequently as they were either too large to consume in a day or were collected in great quantities on a given night, respectively.

The percentage of households hunting particular taxa ranged from 16-91% (Fig. 2), with the fewest number of households hunting bats and the greatest number of households hunting tenrecs. Harvest was extremely variable between villages (Fig. 3). The mean bushmeat harvest revenue (R) ranged from \$8.50-25.90 per household per year (Table 1). The common tenrec, *Tenrec ecaudatus*, a frequently hunted and extremely fecund species, contributed 5-57% to this annual value per household, more than any other species (Table 1). An estimated total opportunity cost (C) of \$8.30 per household per year was established based on time dedicated to hunting activities (Table 2, for derivations of these costs refer to Appendix 2.2).

Net values (V) of \$0.20 – 17.60 per household per year and average net values of \$16 – 1,480 per village per year were calculated from the cost benefit analysis (Table 2), demonstrating the large variability in benefits received among households and among villages (Fig. 3). Based on the lower and upper end estimates of harvest radius of 4.4-7.3 km (Fig. 1) surrounding each of 26 villages (total hunted area = 92,000-195,000 ha), the net value in one year of wildlife provisioning ranged from \$0.01 - 0.20/ha (Table 3). Incorporating the impact of strict conservation monitoring and enforcement, revenue and net values would be reduced to approximately one third of their current value (Tables 2 and 3). Effective conservation monitoring would not only impact local economies but would also incur a 45% reduction in the biomass of wildlife received by households, a major public health effect.

In the MPA, the median annual income was \$70. The revenue value of the annual bushmeat harvest as a fraction of median annual income across the 26 villages ranges from 12-37%. The replacement cost for the amount of bushmeat consumed, using beef as the substitute (\$2.35/kg), was \$34 - 63 per household per year. This replacement cost would represent 49 – 90% of median annual income, an amount inconceivable for local residents to pay. Using chicken as the substitute (\$1.87/kg), the replacement cost would be \$27 – 50 per household per year or 39 – 71% of median household annual income.

## Discussion

Our study of wildlife provisioning in Madagascar highlights variability in the flow of ecosystem services at the sub-regional, community and household scale, which could prove useful for development targeting and understanding local incentives for conservation or environmental rule-breaking. Such understanding is critical if conservationists wish to determine the impact of enforcement on wildlife provisioning or the potential cost of unsustainable use. We focused only

on the opportunity costs to local economies from potential decreases in utilization of direct-use services due to conservation enforcement (approximately two thirds reduction of current value) and did not examine the long-term cost to local livelihoods (both economies and human health) of unsustainable use leading to depletion. We do, however, pay attention to the distribution of net benefits among sub-regions, communities and households.

Our estimate of current net value, \$0.01 - 0.20 per hectare per year, is significantly less than other estimates elsewhere; e.g., approximately two orders of magnitude less than Cross River National Park, Nigeria (Ruitenbeek 1989) and Iquitos, Peru (Padoch & de Jong 1989). Even though we found a relatively low absolute monetary value for this service, markets are imperfect and market valuation weights all preferences by purchasing power (Scitovsky 1993). Further, economically poor, resource-dependent people heavily discount future benefits (Pearce et al. 2003). Thus, few commodities will have a high monetary value to the poor, even if they are essential to life. If they truly are essential and non-substitutable, it is possible that the value is infinite and inestimable (Gowdy 1997). While estimating per hectare monetary values may provide a basis for cross-regional comparisons (Godoy et al. 1993) or comparisons among alternative management scenarios (i.e. Table 3, Kremen et al 2000), measurements at this scale do not elucidate our understanding of differences in equity among communities and households. Thus, it is important to examine benefits at the community and household levels to estimate the proportional value of a service to local livelihoods and annual income.

Cost-benefit analysis often ignores the distribution of benefits and focuses on potential Pareto improvements, where those who benefit could compensate losers (Farley 2008). Yet, when dealing with such extreme poverty and low capacity to compensate those most affected, it is likely that the costs of conservation will disproportionately burden the poor and vulnerable - those most reliant on access to natural resources (Shyamsundar 1996, Shyamsundar & Kramer 1997, Ferraro 2002). Analyses that determine fine-scale dynamics of benefit flows facilitate the identification of groups (i.e. communities, households or individuals) most vulnerable to disruptions or restrictions in the flow of ecosystem services. Identifying these groups would allow for targeted support of sparse resources from development and public health organizations.

Recently, there has been an ideological shift in development policies from universalism (where people receive development support based on coarse-scale geography rather than localized need) to targeting of economic or other support to those perceived to be most in need (Mkandawire 2005). Targeting support is seen as particularly valuable in the context of developing countries as it is more cost-effective than universalist approaches (Mkandawire 2005), not unlike systematic conservation planning approaches that attempt to maximize conservation value obtained per dollar spent (Naidoo et al. 2006). Development targeting is broadly equivalent to the public health approach of eliminating health disparities (Satcher & Higginbotham 2008), designing interventions to ameliorate the health status of communities, households or individuals most in need. The public health approach is particularly relevant to this case study because interventions are often focused on the prevention of future health problems based on current or anticipated vulnerability. Our study helps to identify the portion of stakeholders most vulnerable to the loss of access to wildlife.

Results of this study could be used in specific ways to target development and public health support and to understand local attitudes toward conservation policies, a central focus of the emerging field of conservation psychology (Saunders 2003). Specifically, our results show

that household revenue from wildlife harvest contributed 12-37% of median annual income and that conservation enforcement scenarios could induce both a negative net value from hunting and an average 45% loss of wildlife biomass in households. The value of bushmeat when expressed as a proportion of annual income is less reliant on price elasticities and enhances our ability to understand motivations for resource extraction decisions and target those most vulnerable to ecosystem service perturbations or restrictions. Those who have the greatest proportional economic reliance on this service are likely to be the most resistant to policy changes that restrict access to the service. Additionally, negative net values of harvesting due to conservation enforcement may reveal local attitudes toward conservation where some local people may receive greater economic benefit by rule-breaking than following the regulations of community-based natural resource management regimes (Keane et al. 2008). The results of the conservation scenario (Table 2 & 3) provide an empirical framework for determining eligibility criteria to determine where to allocate targeted development and public health support. For example, those who disproportionately rely on the economic value of wildlife provisioning could be slotted for an alternative agriculture intervention and those who disproportionately rely on the biomass of wildlife could be targeted for a public health intervention designed to promote dietary diversity or sustainable poultry husbandry. If households were to switch to legal prey species to compensate for the loss in biomass, they would need to double the rates at which these species are currently harvested, likely leading to rapid local extinctions. If households switched to domesticated meats, their production would likely increase and prices would drop. However, this would be prohibitively expensive for local people without initial assistance.

There are several limitations to our study including the use of a basic valuation technique which ignores dynamic valuation models (for further information, see Bockstael et al. 2000). We do not project current ecosystem service values into the future because of limited data on possible trajectories of the service. If significant depletion occurs or if access is highly restricted, it is possible that there would be inelastic demand for wildlife and that prices would increase (Farley 2008). Taste preferences (unpublished data) indicate that domesticated meats are generally preferred in this system. If domesticated meats were to become more available (which might require significant development interventions), then bushmeat prices would not majorly increase and consumption would instead shift to alternatives. Wilkie and Godoy (2001) have shown in a similarly remote and subsistence setting that bushmeat shows elastic demand and that consumption would be reduced through price increases. If we were to model future flows, a high social discount rate would have been used because the recent inception of the protected area in 2005 may cause local people to value this resource more in the present than in the future when it is likely to be more restricted through monitoring and enforcement. In developing countries, there is always a lag between when a protected area becomes gazetted on paper and when the local infrastructure develops to mark boundaries, set up patrolling to monitor infractions and enforce conservation policies. Therefore, the option value and bequest value of this service may become negligible to local users because of either unsustainable use or policy regulations that could translate into a high social discount rate. Currently, in the MPA, monitoring and enforcement is minimal (Golden 2009) and we did not include the risk of being caught conducting an illegal activity into the costs of hunting in our analysis. This cost will likely increase as conservationists and local governing officials become more aware of hunting as a threat to the sustainability of mammal species in Madagascar.

In order for the provisioning service of wildlife to be maintained in the long-run, the harvest needs to be sustainable, which is unlikely for many species in the MPA (Golden 2009). In such an economic setting, issues of sustainability are unlikely to be considered by local people, particularly when the majority of hunting uses passive techniques at low cost. Furthermore, supply of bushmeat is not based on a given species' sustainability or population dynamics because snares and traps target communities (i.e., lemurs, carnivores, etc.) of several species. Because of the nature of passive hunting, even if a population crashes for a given species, hunting behavior is unlikely to change (Noss 1998, Wilkie & Carpenter 1999). Moreover, the declines may not be visible to local people because the population trajectories may not linearly decay. Instead, they may reach a threshold or tipping point after which local extinction of wildlife is inevitable. It is vital to maintain the reproductive capacity (i.e., the productive fund service, sensu Farley & Costanza 2010) in order to ensure future flows of benefits.

Although not quantified in this study, it is also necessary to understand non-monetary values of provisioning services, such as the role wildlife may play in enhancing human health and nutrition. Elsewhere, we assess potential effects on human health such as the loss of essential nutrients like iron, due to either enforcement or depletion (Golden et al. in review). Furthermore, these studies ignore the other important natural resources whose access may also be restricted through conservation enforcement. By uncovering the winners and losers from conservation management, community and household-level targeting for development and public health support is possible. Through a detailed understanding of the local value of wildlife and other natural resources, those working towards conservation and economic development can better recognize the true value of natural resource access to the local residents who strongly influence the fate of the forest and its inhabitants.

Table 1: Wildlife biomass harvest rates per annum and associated revenue

Species	Harvested biomass/ household/yr (kg) <sup>a</sup>	Mean Price/kg <sup>b</sup>	Revenue per household/yr (USD)
<b>LEMURS:</b>			
<i>Avahi laniger</i> /Eastern woolly lemur	0.54 – 0.99	1.04 – 1.89	0.57 – 1.87
<i>Cheirogaleus sp.</i> /Dwarf lemur sp.	0.29 – 0.92	1.21 – 3.82	0.35 – 3.52
<i>Daubentonia madagascariensis</i> / Aye-aye	0.08 – 0.08	0.94 – 0.98	0.07 – 0.08
<i>Eulemur albifrons</i> / White-fronted brown lemur	1.60 – 2.08	0.85 – 1.10	1.36 – 2.28
<i>Eulemur rubriventer</i> / Red-bellied lemur	0.28 – 0.41	0.91 - 1.36	0.25 – 0.56
<i>Hapalemur griseus</i> / Eastern bamboo lemur	0.42 – 0.59	1.03 – 1.44	0.43 – 0.85
<i>Indri indri</i> /Indri	0.80 – 1.17	0.48 – 0.70	0.38 – 0.81
<i>Lepilemur sp.</i> /Sportive lemur sp.	0.21 – 0.31	1.23 – 1.84	0.26 – 0.57
<i>Microcebus sp.</i> /Mouse lemur sp.	0.004 – 0.004	5.88	0.02 – 0.02
<i>Propithecus candidus</i> /Silky sifaka	0.03 – 0.04	0.45 – 0.59	0.01 – 0.02
<i>Varecia rubra</i> /Red ruffed lemur	0.01 – 0.02	0.68 – 1.08 <sup>c</sup>	0.01 – 0.02
<i>Varecia variegata</i> / Black and white ruffed lemur	0.61 - 0.96	0.68 – 1.08	0.42 – 1.04
<b>CARNIVORES:</b>			
<i>Cryptoprocta ferox</i> /Fosa	1.25 – 2.49	0.39 – 0.78	0.49 – 1.95
<i>Eupleres goudotii</i> /Falanouc	0.41 – 0.74	0.59 – 1.06	0.24 – 0.78
<i>Fossa fossana</i> /Fanaloka	0.07 – 0.13	0.31 – 0.59	0.02 – 0.07
<i>Galidia elegans</i> / Ringtailed mongoose	0.27 – 0.38	0.72 – 1.02	0.19 – 0.39
<i>Viverricula indica</i> / Lesser Indian civet	1.04 – 2.08	0.33 – 0.66	0.35 – 1.38
<b>BATS:</b>			
<i>Pteropus rufus</i> / Madagascar flying fox	0.02 – 0.03	1.23 – 1.85	0.02 – 0.06
<i>Rousettus madagascariensis</i> / Madagascar rousette	0.08 – 0.31	0.44 – 1.76	0.03 – 0.55
Insectivorous bats spp.	0.0002 – 0.0006	4.20 – 9.80	< 0.01
<b>TENRECS AND BUSH PIG:</b>			
<i>Potamochoerus larvatus</i> / Bush pig	1.29 – 2.59	0.97	1.25 – 2.51
<i>Setifer setosus</i> / Greater hedgehog tenrec	0.15 – 0.25	3.92 – 6.72	0.58 – 1.71
<i>Tenrec ecaudatus</i> / Common tenrec	5.05 – 10.10	0.24 – 0.48	1.21 – 4.85
<b>BUSHMEAT BIOMASS HARVEST</b>	14.50 – 26.69		8.53 – 25.91

<sup>a</sup> Mean number harvested multiplied by the range in adult body mass. Body mass values from Garbutt 2007.

<sup>b</sup> For all species except *P. larvatus*, values were derived from mean price of individual species divided by a range in adult body mass. At time of research, \$1 USD = 1,700 ariary.

<sup>c</sup> Price per kilogram proxied by *Varecia variegata*.

Table 2: Cost-benefit analysis of bushmeat harvest value and time allocation costs

	Status quo value (USD)	Value with conservation enforcement <sup>a</sup> (USD)
Bushmeat biomass revenue <sup>b</sup>	8.53 - 25.91	3.44 - 11.06
Average time allocated <sup>c</sup>	139 hours	87 hours
Average time allocation costs <sup>d</sup>	8.34	5.22
Flow value per household <sup>e</sup>	0.19 - 17.57	-1.78 - 5.84

<sup>a</sup>The conservation enforcement scenario removes all illegally-harvested species (i.e. lemurs and endemic carnivores) from the revenue and removes all illegal-hunting activities from the allocation costs.

<sup>b</sup> Minimum and maximum revenue per household per year (see Table 1).

<sup>c</sup> The number of households involved in each hunting activity multiplied by the average number of hours per year expended in each hunting activity.

<sup>d</sup> The average number of hours allocated multiplied by the minimum hourly wage labor rate. Time allocation costs per household per year are shown.

<sup>e</sup> Household bushmeat biomass revenue subtracted by the average time allocation costs.

Table 3: Bushmeat harvest value scenarios per hectare

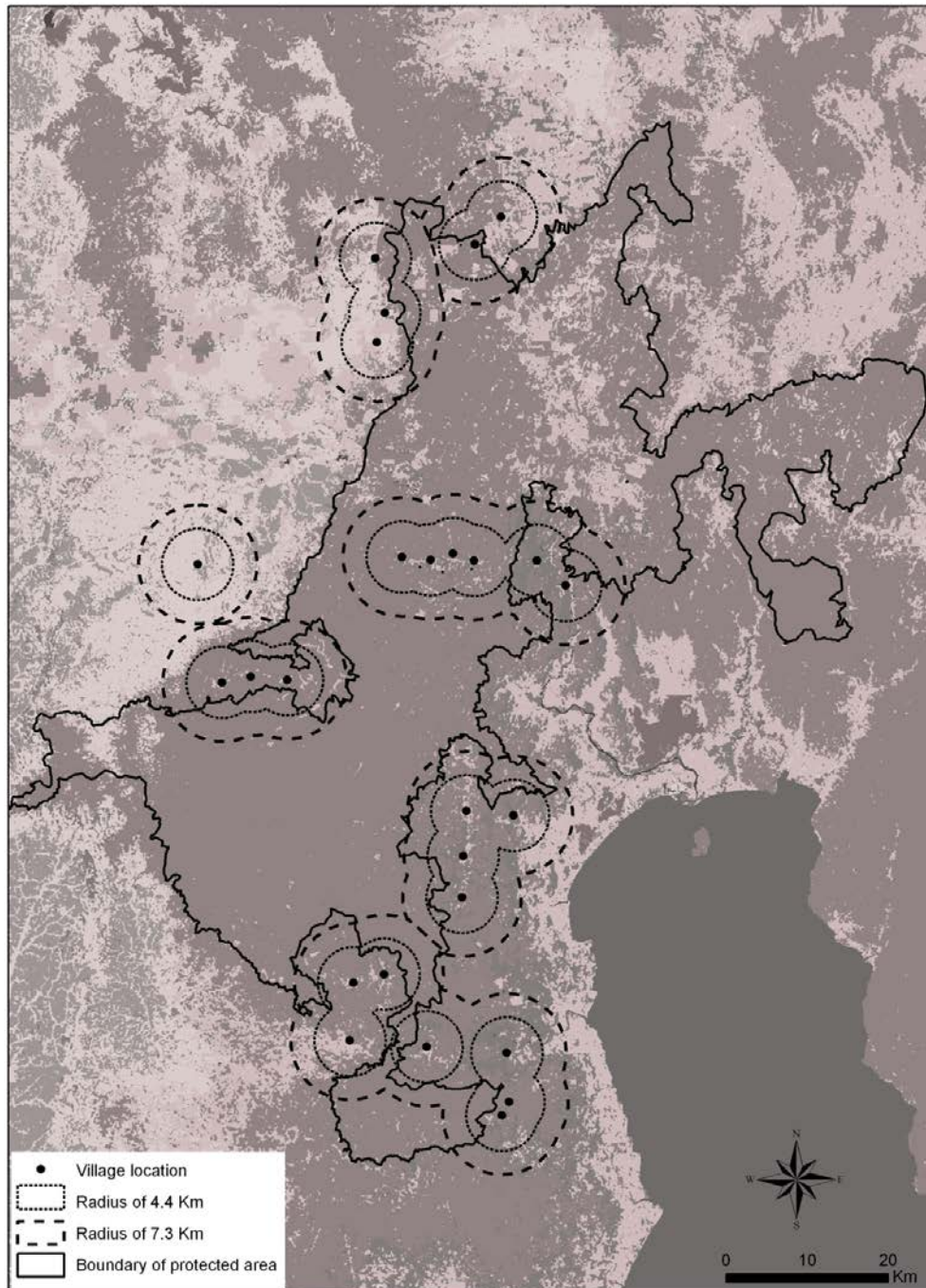
Value scenario ( per hectare)	Status quo value (USD)	Value with conservation enforcement (USD)
Revenue value (R) <sup>a</sup>	0.21-0.29	0.08-0.12
Flow value (V) <sup>b</sup>	0.01-0.20	-0.04-0.07

<sup>a</sup> The range in annual offtake per year of all wildlife harvest unadjusted for costs incurred.

<sup>b</sup> The range in mean flow value per household extrapolated to the total number of households and divided by the minimum and maximum harvest areas for villages.

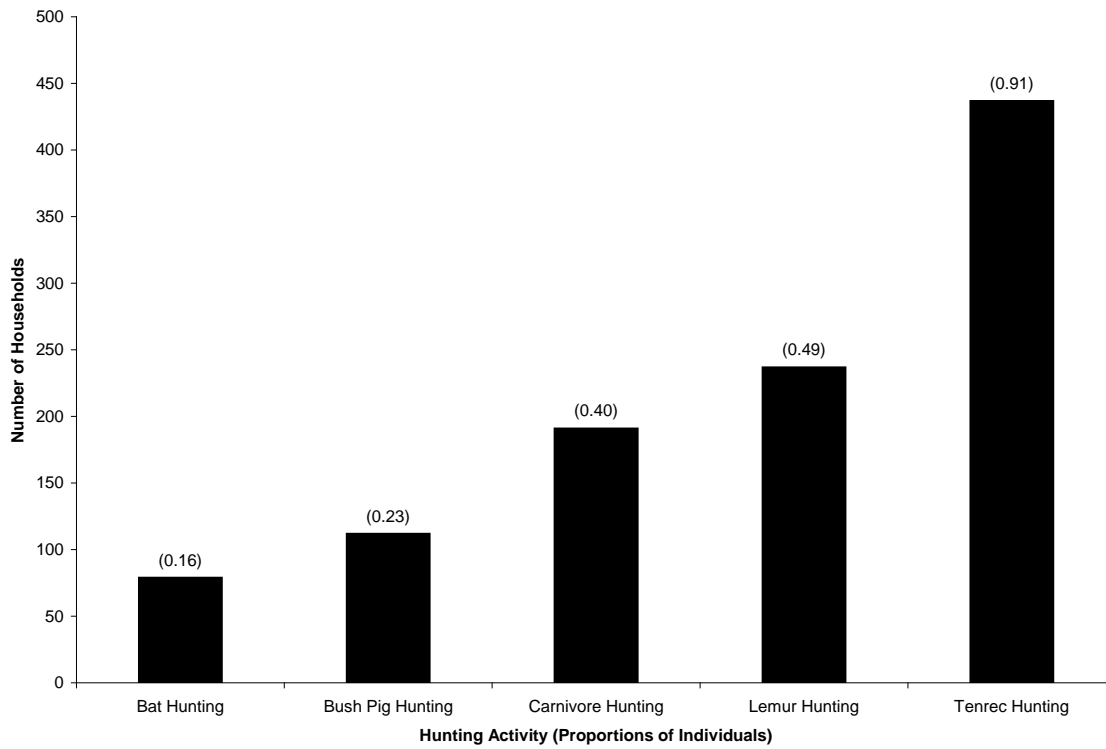


Figure 1: Map of the Makira Protected Area and Hunting Harvest Areas



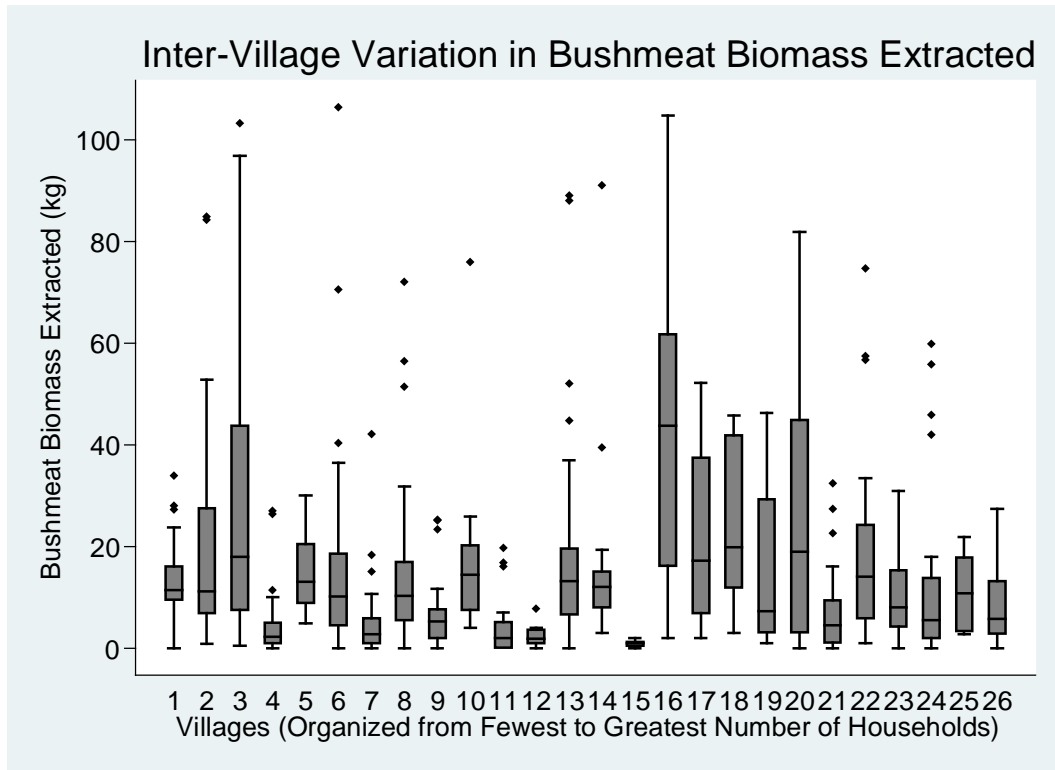
The Makira Protected Area is pictured with two circles surrounding each village: the first is the mean harvest area surrounding a village while the second is the mean harvest area plus one standard deviation (Golden 2009).

Figure 2: Household Involvement in Hunting Activities



Across the Makira Protected Area, the percentage of households hunting particular taxa ranged from 16-91% with the fewest number of households hunting bats and the greatest number hunting tenrecs.

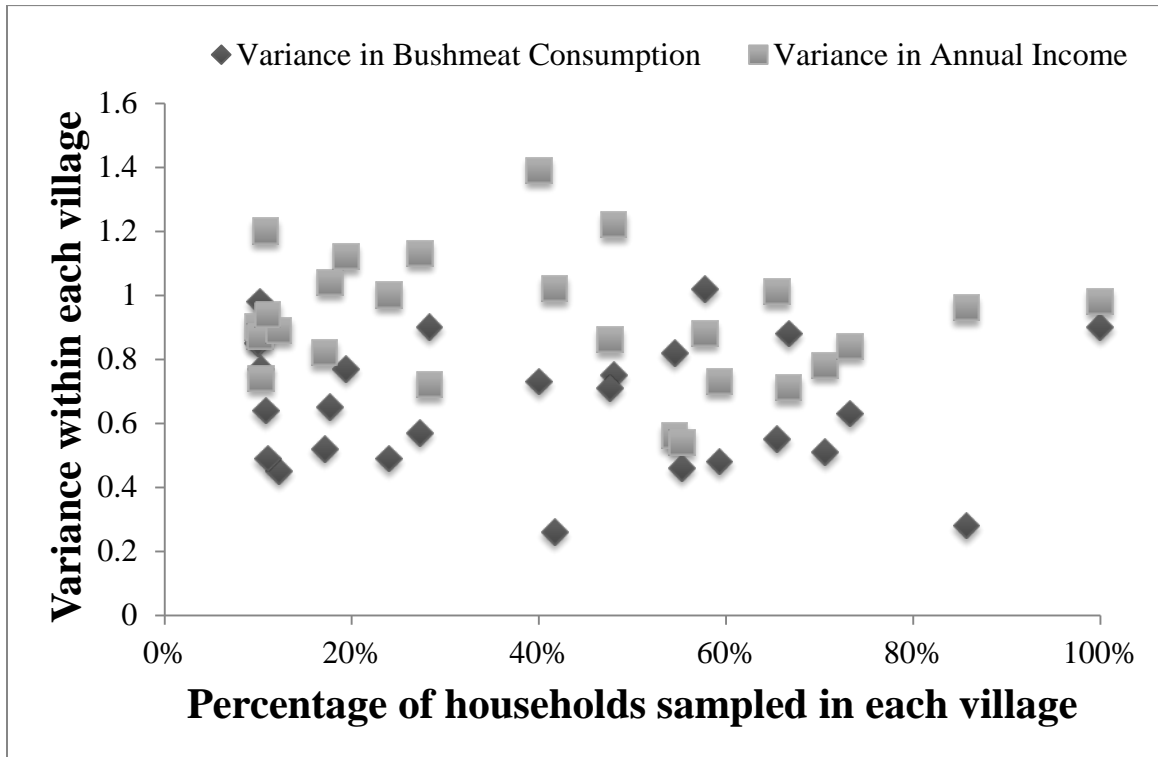
Figure 3: Variability in the distribution of the flow of harvested wildlife among villages



Each boxplot exhibits the variation within and between villages in the amount of wildlife biomass being harvested. Longer boxes denote greater inter-household variation while shifts in scale and height between villages denote inter-village variation.

Supporting Information

Appendix 2.1: The variation in bushmeat consumption and annual income among sampled households



There does not appear to be a statistical pattern between the percentage of households sampled in each village and the amount of intra-village variance in both the reported rates of bushmeat consumption or annual income.

Appendix 2.2: The time allocation of hunting behavior

Hunting Activity	Time Allocated <sup>a</sup> (Hours)	# of Days	Average # Days/Year <sup>b</sup>	Average # Hours/Year <sup>c</sup>	Total Time Allocated (Hours) <sup>d</sup>
<b>BATS:</b>					
Average Bat night hunting	3.52	7	24.00	84.57	
<i>Average Time Hunting Bats</i>				84.57	6,509
<b>BUSH PIGS:</b>					
Average Bush Pig Snare Construction	2.88	3	1.00	2.89	
Average Bush Pig Snare Checking	2.78	7	33.80	94.16	
Bush Pig Snare Construction Total				97.05	
Average dog hunting of bush pig	5.50	3	12.00	66.00	
<i>Average Time Hunting Bush Pigs</i>				81.52	9,019
<b>LEMURS:</b>					
Average Constructing lemur snares	5.80	4	1.00	5.79	
Average Checking lemur snares	3.97	24	30.61	121.59	
<i>Average Time Hunting Lemurs</i>				127.38	30,022
30% Travel Discount Incorporated <sup>e</sup>					21,016
<b>TENRECS:</b>					
Average Dog Hunting of Tenrecs	1.47	11	40.75	59.89	
<i>Average Time Hunting Tenrecs</i>				59.89	26,214
<b>CARNIVORES:</b>					
Average Carnivore Snare Construction	1.20	5	1.00	1.20	
Average Carnivore Snare Checking	0.65	12	31.69	20.69	
<i>Average Time Hunting Carnivores</i>				21.89	4,212
<b>Total Across Taxa</b>					66,969
<b>Average Time Allocated per Household</b>					139

<sup>a</sup> Measured as a mean number of hours expended per excursion.

<sup>b</sup> Calculated from routine interviews by multiplying the number of days per week engaged in each hunting activity by the length in weeks of each type of hunting season.

<sup>c</sup> Calculated by multiplying the number of minutes allocated to each activity by the number of days each year engaged in each hunting activity.

<sup>d</sup> Calculated by multiplying the average number of hours per year engaged in hunting each taxa (italicized) by the number of households hunting each taxa in the surveyed villages.

<sup>e</sup> Lemur hunting was typically conducted after having traveled to the hunter's agricultural fields. Because the time traveling to agricultural fields should be subtracted from the allocation of hunting time, a discount of 30% was applied to these time allocation statistics. This accounts for the approximate 1hr round trip travel time from the village to the fields.

## CHAPTER 3

### Benefits of Wildlife Consumption to Child Nutrition in a Biodiversity Hotspot

#### Introduction

Biodiversity loss and large-scale wildlife declines are now globally pervasive and well-documented (Pauly *et al.* 2003, Brashares *et al.* 2004, Butchart *et al.* 2010). These losses have triggered severe ecological ramifications such as trophic meltdown (Terborgh *et al.* 2001), loss of critical ecological interactions (Şekercioğlu *et al.* 2004, Peres & Palacios 2007) and extinctions of fish and game species (Brashares *et al.* 2004, Roberts & Hawkins 1999). Surprisingly few studies have quantified the effects of wildlife declines on human economies and health outcomes (Milner-Gulland *et al.* 2003, Worm *et al.* 2006) despite the essential role of wildlife consumption in shaping human evolution (Hill 1982, Kuhnlein & Receveur 1996) and in the diet of hundreds of millions of rural people across the globe (Robinson & Bennett 2000, Milner-Gulland *et al.* 2003, Brashares *et al.* in press). Wildlife declines are likely to have direct and powerful effects on human health and nutrition, particularly via lost access to critical micronutrients (Neumann *et al.* 2003). While many studies have suggested that wildlife can provide a food security safety net (Brashares *et al.* in press), our study illuminates quantitative links between micronutrients derived from wildlife and critical human health outcomes. These results provide a clear example of how rapid global declines of access to wildlife for consumption, due either to conservation measures (Agrawal *et al.* 2008) or wildlife depletion, could significantly affect the health of local human populations. Such linkages between biodiversity loss and human health highlight the need for research and mitigation approaches that integrate the disciplines of public health and conservation biology.

The widespread harvest of wildlife for human consumption is a multi-billion dollar provisioning service (*sensu* Millennium Ecosystem Assessment 2005) worth tens of millions of dollars to the rural poor (Milner-Gulland *et al.* 2003, Pimentel *et al.* 1997), but to date research has documented the economic value of wildlife with little regard for the importance of wildlife consumption to human health. Animal source foods such as wildlife are rich in energy, protein, and micronutrients that have greater bioavailability than vegetable sources (Neumann *et al.* 2003). Micronutrient deficiencies, described as “hidden hunger” because of their often asymptomatic nature, are the most prevalent form of malnutrition globally and have a range of health sequelae (Black *et al.* 2008). Iron deficiency is the most prevalent nutrient deficiency worldwide and results in negative consequences for brain metabolism, myelination, neurotransmitter function, motor development, physical activity, and emotional regulation (Pollitt 2001, Lozoff *et al.* 2006). Its most severe form is Iron-deficiency anemia (IDA), which is characterized by a deficiency of red blood cells, and affects more than 2 billion people worldwide including 46-66% of children under age four in developing countries (Stozfus *et al.* 2004). IDA is caused by the inadequate intake of iron-rich foods and/or excessive blood loss due to bleeding or infectious diseases such as malaria or parasitic infections. Our research examined how access to wildlife as a food source affected the risk of anemia for a longitudinal cohort of 77 children (Table 1) living in a remote area of eastern rainforest in Madagascar (Fig. 1), who were measured monthly from March 2008 to February 2009. The rural community where the study

was conducted relies heavily on local wildlife resources (Fig. 2) as do more than 300 million people globally who are supported nutritionally by forest products (Pimentel *et al.* 1997).

Unlike protein which can be acquired at adequate levels from multiple dietary sources bioavailable iron is almost exclusively derived from animal-source foods (Beaton *et al.* 1992, Neumann *et al.* 2003). We hypothesized that increased wildlife consumption would be associated with a reduced incidence of anemia based on clinical evidence linking animal source foods with improved human nutritional status (Neumann *et al.* 2003).

## Methods

### *Site Description*

The Makira Protected Area (Fig. 1) in north-eastern Madagascar covers 3,712 km<sup>2</sup> of lowland and mid-altitude rainforest (Golden 2009). It is one of the most biologically diverse ecosystems in Madagascar and represents one of the nation's largest remaining blocks of contiguous forest. Two ethnic groups dominate the area, with Betsimisaraka predominating in the east and south, Tsimihety predominating in the north and west, and a mixing where these regions overlap. This forest supports 18 species of lemurs, all of which are endemic to Madagascar (Golden 2009). There are also several unique species of carnivores, bats, and micro-mammals, all of which are hunted in this ecosystem (Golden 2009). Although the majority of hunting was already illegal before the recent formation of the Makira Forest as a protected area in 2005, the dawning of conservation attention through its new protection status and its co-management with the Wildlife Conservation Society will engender increased monitoring and enforcement over a geographical area that was previously habituated to customary access rights (Golden 2009). In this region, we have focused this research in one village adjacent to the demarcation of the protected area.

### *Randomization and Adherence*

The village selected for this research had a total of 105 households. We had previously utilized systematic random sampling from a census list of households to select 48 households for an environmental resource use study examining rates of wildlife and other non-timber forest product extraction. Of the 48 households already enrolled in the environmental cohort, 28 had children 12 years of age or younger and were asked to join the current study. We restricted the study base to children 12 years of age and younger to exclude girls who had reached menarche (a factor known to impact hemoglobin concentrations). After screening, we individually spoke to the females in the study group to assure that they had not yet reached menarche. All 28 households joined the study in March 2008, enrolling 77 children. Before the end of the study in February 2009, four children withdrew from the study (one after 6 months of follow-up and three after nine months of follow-up).

### *Health and Diet Measurements and Protocols*

Every household was visited each month for anthropometric measurements<sup>1</sup>, illness recalls and hemoglobin sampling using a HemoCue Hb 201+ Analyzer (Bäck *et al.* 2004). For children who were too young to answer their own health questions (typically under age five), surrogate

responses from mothers were accepted. Outcome assessors were blinded to the exposure status of children and children were not aware of the purpose of the study. On a daily basis, the female head of household maintained a diet calendar throughout the duration of the study, recording the type (i.e. chicken, duck, fish, beef, pork, or species of wildlife) and weight of every meat consumed by the household. Meat weights were measured through the use of scales and recorded daily in diet calendars over the course of one year. Dressed meat weight (after hair removal, feather plucking, etc.) was used.

### *Intra-Household Food Allocation*

Fourteen of the 28 households in the study base, also randomly selected at the beginning of the study, were visited without forewarning once a month and observed during dinner to determine patterns of intra-household food allocation. Because the daily diet calendars measure the amount of food consumed at the level of the household, it is also necessary to develop estimates of how this food is then partitioned to individuals within households. The 14 households were visited each month during the study and 40 of the 77 children in the study sample were observed. A research assistant counted the number of spoonfuls of stew consumed by each household member from a communal stew bowl. These stews were occasionally comprised of meat, vegetables, or a mixture of both. These observations permitted the calculation of a mean proportion of stew typically consumed by individuals by summing all spoonfuls and then calculating an individual's allotment. Determining the proportion of stew consumed by individuals based on age and sex permitted modeling of individual level wildlife consumption.

### *Statistical Analysis*

All statistical analyses were conducted using STATA Version 10.0. To test the hypothesis that wildlife consumption was associated with hemoglobin concentrations, we used a generalized linear mixed-model (GLMM) regression. The use of a GLMM permitted multi-level modeling and we clustered observations at the repeated measures for individuals and by the households in the study population. Thus, the model treated individuals and households as latent random effects. The model was bootstrapped to produce robust standard errors. We generated two models: one with the full set of covariates and the second included the statistically significant ( $p < 0.05$ ) variables from the first model (Table 2). In model 1, we included household-level variables, including domesticated meat consumption and annual income. We also included temporally varying individual-level variables, such as body mass index for age z-score, malaria incidence in the previous month, and the number of months since the consumption of deworming medication. The best fit equation for predicting the hemoglobin concentration of an individual from Model 2 was:

$$\text{Equation 1: } Y_{ijk} = \beta_0 + \beta_{0i} + \beta_{0ij} + \beta_1 x_{ijk} + \beta_2 x_{ijk} + \beta_3 x_{ijk} + e_{ijk}$$

where  $Y_{ijk}$  is the concentration of hemoglobin (g/dL) for the  $i^{\text{th}}$  individual in the  $j^{\text{th}}$  household at the  $k^{\text{th}}$  visit,  $\beta_0$  (10.48) is a constant,  $\beta_{0i}$  is the random effect for the individual,  $\beta_{0ij}$  is the random effect for the household,  $\beta_1$  (0.20) is the annual amount of individual wildlife consumption,  $\beta_2$  (0.55) is the log-transformed annual household income centered around the mean,  $\beta_3$  (0.17) is the age in years of the individual, and  $e_{ijk}$  is the error term (Table 2).



### *Modeling the Effect of Wildlife Loss on Hemoglobin*

To understand the potential health impact of the loss of access to wildlife, we modeled the response of hemoglobin concentrations of individual children to changes in wildlife consumption. Using the best fit equation from the Model 2 GLMM (Equation 1), we estimated the change in hemoglobin concentrations from wildlife consumption by subtracting the expected hemoglobin concentrations with zero wildlife consumption from the hemoglobin concentrations under observed levels of wildlife consumption. The difference in hemoglobin concentrations for an individual is the effect of the loss of access to wildlife. By comparing the prevalence of anemia under observed conditions to the prevalence following a scenario where wildlife access is lost, we estimated the predicted increase in childhood anemia. Anemia was determined based on a threshold of 11.0 g/dL for children less than five years of age and 12.0 g/dL for children five to twelve years of age.

### *Calculating Harvest Required to Eliminate Anemia*

In our study village, we determined the amount of additional hunting required to eliminate anemia from the population of children. We calculated the additional amount of harvest required by solving the GLMM (Equation 1) for the amount of wildlife consumption required to raise all individuals above the thresholds for anemia (see above), finding that harvest would need to be increased 11-fold. For the purposes of this analysis, we assumed that the study village was representative of other villages in the Makira region in hemoglobin levels and wildlife consumption, and then utilized a larger data set on bushmeat consumption (481 households distributed among 26 villages, median village size is 57.5 households- see Chapter 2) to assess the effects of increasing hunting 11 fold on wildlife. From this larger dataset, we calculated the average number of individuals per animal species consumed by each household over the course of a year. We then multiplied the number of individual animals harvested per household per year (determined from oral recall) by the lower and upper range in species body mass from the literature (Garbutt 2007) to determine the average biomass consumed by each household per year. Following this, we extrapolated the biomass consumed by households to the level of each village and multiplied this biomass by 11. The expanded harvest rate required to eliminate anemia from the 26 villages would equal approximately 1,280.8 kg/km (using a median village size of 57.5 households and a harvest area of 7.3 km<sup>2</sup>- see Chapter 2). We took the difference between the harvest needed for anemia elimination and the current rates of harvest to determine the number of additional animals required. We divided the difference between required and current harvest by the maximum body weight of each species assuming that the relative proportion of each species in the diet remained unchanged. An average additional 142 individual animals would be needed by each household (range 1-50 individual animals per species). To determine the potential sustainability of this required harvest, we used the range in observed household wildlife biomass consumption produced from the lower and upper body masses of each species and compared it to the required amount of biomass needed for harvest per square kilometer. The wildlife biomass harvest required (1,280.8 kg/km<sup>2</sup>) would be 6-11 times more than the observed rates of current wildlife harvest and 8 times more than the expected sustainable rates of maximum sustainable yield (Robinson & Bennett 2000). This means that current rates of biomass harvest are right at the fringe of sustainability, although this is not indicative of species-specific sustainability.

## Results

### *Meat Consumption Patterns*

Household wildlife consumption ranged from 0 to over 53 kilograms of wildlife per year in the study households. The proportional composition of wildlife in the diet over total meat consumption ranged from 0-76%, with a mean of 16% and a median of 14% (Fig. 2). There was a negative correlation ( $-0.40$ ,  $p$ -value $<0.0005$ ) between household income and the proportional mass by weight of wildlife over total meat consumption in the diet. Thus, there was a greater relative reliance on wildlife as an animal source food in lower income households. Wildlife consumption was positively skewed with median household consumption at approximately 8.75 kilograms per year. In terms of dressed weight, wildlife accounted for approximately 16% of annual meat consumption.

### *The relationship between bushmeat and hemoglobin*

A high prevalence of stunting is often a marker of a chronically malnourished population whereas a high prevalence of wasting (low Weight-for-Height z-score) is a marker of acute malnutrition. The population of children appears to be chronically undernourished, evidenced by high rates of stunting and very low rates of wasting (Table 1). Hemoglobin concentrations were normally distributed in our study population (mean 11.7, SD: 1.3 g/dL) and we categorized 42% of children as anaemic. The mean hemoglobin concentrations show that this population is on the fringe of health.

We found strong support for our primary hypothesis in our year-long monitoring of hemoglobin levels and wildlife consumption in children. Children who consumed a greater quantity of wildlife had higher hemoglobin concentrations ( $\beta$  (95%CI) = 0.20 (0.0078, 0.39),  $p = 0.041$ ), when controlling for domesticated meat consumption, household income, gender, age, and nutritional and disease status (Table 2). Of the confounding variables, only household income ( $\beta = 0.55$ ,  $p < 0.0005$ ) and age ( $\beta = 0.17$ ,  $p < 0.0005$ ) were significantly positively associated with hemoglobin concentration.

To examine wildlife's relative nutritional importance, we used an empirical model of hemoglobin levels in response to wildlife consumption. The maximum benefit of wildlife consumption for a child, when controlling for age and household income, is a mean increase in hemoglobin concentration of 0.69 g/dL (95% CI: 0.36, 1.02 g/dL, Fig. 3). Using our empirically-derived estimates of the contribution of wildlife to hemoglobin levels, we modelled the health impact of removing household access to wildlife, such as would occur if current laws in Madagascar against wildlife hunting were strictly enforced or if wildlife populations collapsed from overharvest or other factors (e.g., habitat conversion, climate change, and drought). Under this scenario, the prevalence of anemia would increase from 42% to 54% in our study group, a nearly 1.3 fold increased risk of anemia at the population level (Fig. 3).

By comparing the modelled population with no access to wildlife to the observed population that frequently consumed wildlife, we estimated that the odds of transitioning into anemia following wildlife loss would be four times higher (95% CI: 1.90, 8.40) in households with greater dependency on wildlife than in households with less dependency (dependency

determined through a median split of total mass of wildlife consumed over total mass of meat consumed, Fig. 4). Because households at the highest income levels showed the lowest dependency on wildlife consumption (correlation: -0.254,  $p < 0.001$ ) and were most able to afford domesticated meat as an alternative to wildlife, children in the economically poorest households would be three times (odds ratio=3.05, 95% CI: 1.29, 7.45; Fig. 4) more likely to transition into anemia than either middle income or high income households if access to wildlife was restricted. Based on our calculation of the association between wildlife consumption and hemoglobin levels, approximately 1280.8 kg/km<sup>2</sup>/yr of wildlife would need to be harvested from local hunting grounds before anemia would be eliminated in our study community. This biomass would be equivalent to an increased annual harvest of 142 animals per household in total (range, 1–51 animals per species per household).

## Discussion

Anemia has significant and life-long impacts on human health outcomes (Lozoff *et al.* 2006). Thus, the increased prevalence of childhood anemia that would occur with decreased access to wildlife may have significant and lasting effects at the individual and population level. In our population, we showed that access to wildlife could increase hemoglobin levels by almost 0.7 g/dL, providing approximately 61-81% of the expected effect of iron supplementation on hemoglobin as determined in efficacy trials (Stolzfus *et al.* 2004). Decreasing hemoglobin concentrations by as little as 1.0 g/dL has been associated with a decline of 1.73 IQ points (Stolzfus *et al.* 2004) at a population level and a 1.28 fold increased risk of mild to moderate cognitive delay (Hurtado *et al.* 1999). Cognitive deficits driven by IDA in infancy have been shown to persist until adulthood (Lukowski *et al.* 2010) and are likely to negatively affect the functioning of a healthy work force in developing countries where iron-deficiency anemia is pervasive (Horton & Ross 2003). We assumed that anemia in this system was mainly IDA as hemoglobin was tightly linked to meat consumption and we controlled for malaria incidence and deworming of intestinal parasites. Studies relating anemia to numerous domains of human health and well-being suggest the tremendous importance of wildlife to local people in Madagascar and highlight the potential for health reductions as a result of wildlife loss in the absence of meat alternatives.

Wildlife hunting is illegal for the majority of mammalian species in Madagascar, but, local human populations continue to exploit them for local consumption due to lack of enforcement of national conservation policies (Golden 2009). Of the 23 mammalian species that are exploited for consumption in our study area, available data on five species suggests that 80% are hunted unsustainably (Golden 2009). Using even the most conservative estimates of current wildlife harvest, wildlife populations in this region of Madagascar will not be sufficient to sustain local human health into the future (Golden 2009). The approximate 1280.8 kg/km<sup>2</sup>/yr of wildlife needed to be harvested from local hunting grounds before anemia would be eliminated in our study community would exceed current rates of exploitation by 6-11 times (see Chapter 2) and maximum sustainable levels of estimated wildlife production by 8 times (Robinson & Bennett 2000).

The enforcement of national wildlife conservation policies that heavily restrict hunting could have a negative effect on the nutritional status of local people, therefore, that is similar to the effects of unsustainable harvest. Seasonal coping strategies like hunting, foraging, and off-farm income are necessary to compensate for seasonal health stresses and reductions in crop productivity (Barrett *et al.* 2001). In 2003, Madagascar's former President Ravalomanana pledged to triple the system of nature reserves to approximately 10% of the nation's surface area (Kremen *et al.* 2008). This bold commitment was justified with ecological and environmental considerations and it was universally applauded by the conservation community (Duffy 2006). Our results suggest that there may be unanticipated health costs and consequences of this rapid change in environmental policy. In fact, both unsustainable hunting and conservation enforcement can lead to the same livelihood outcome on different scales. Conservation enforcement would enact a more rapid restriction of resources, but self-depletion would potentially lead, albeit more slowly, both to irrevocable local population extinctions and loss of the resource. Thus, conservation policy makers and health practitioners must implement integrated conservation and development solutions to mitigate both the effects of wildlife loss on human health and livelihoods, and the potentially severe consequences to biodiversity.

Table 1: Household and Individual Level Characteristics of the Study Population

<b>Household Variables</b>	<b>Mean (SD)/ Percentage</b>
Household Size (# of people) <sup>a</sup>	5.36 (1.73)
Annual Wildlife Consumption (kg) (Median(IQR)) <sup>b</sup>	8.75 (13.35)
Annual Income (Median(IQR)) <sup>c</sup>	\$153.33 (\$185.64)
Maternal Education	
No Education	25%
Primary Education	61%
Secondary Education	14%
Ethnicity <sup>d</sup>	
Betsimisaraka	89%
Tsimihety	11%
<b>Individual Variables</b>	<b>Mean (SD)/ Percentage</b>
Female	55%
Annual Wildlife Consumption (kg) (Median(IQR)) <sup>e</sup>	0.97 (1.98)
Hemoglobin Concentration (g/dL) <sup>f</sup>	
Average Hemoglobin Concentration (g/dL)	11.7 (1.3)
Hemoglobin Concentration (g/dL) for Children<5 years old	11.2 (1.3)
Hemoglobin Concentration (g/dL) for Children>=5 years old	12.0 (1.2)
Anemia <sup>g</sup>	
Average Anemia	42%
Anemia for Children<5 years old	39%
Anemia for Children>5 years old	44%
HFA z-score <sup>h</sup>	-1.18 (1.88)
BMI for age z-score <sup>i</sup>	-0.3 (0.69)
WFH z-score <sup>j</sup>	-0.32 (0.72)
Time Since Deworming (mo.)	2.84 (2.56)

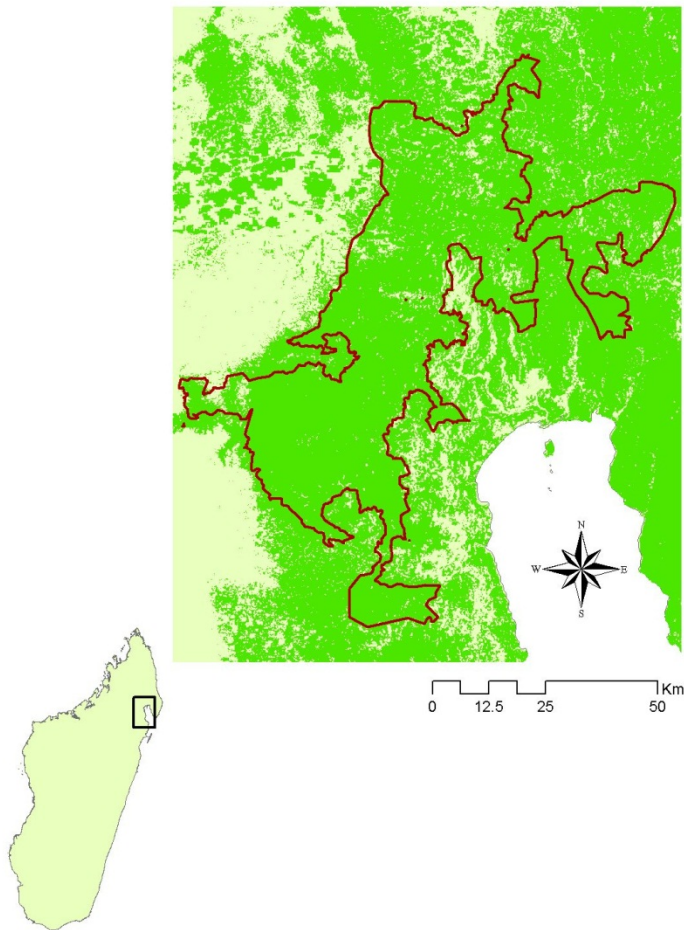
<sup>a</sup>Household size is the unweighted number of individuals in a household. <sup>b</sup>Annual wildlife consumption was measured as the number of kilograms of meat consumed by the household. <sup>c</sup>Annual income was measured as the total value of wages earned, products sold, and items bartered. A conversion rate of \$1=1,800 Malagasy ariary was used. <sup>d</sup>Betsimisaraka and Tsimihety represent the two primary ethnic groups in this region. <sup>e</sup>Individual wildlife consumption is calculated by multiplying household consumption by a proportion allocated to an individual as derived from observation in the intra-household allocation study. <sup>f</sup>Hemoglobin concentrations were measured using a HemoCue Hb 201+<sup>2</sup>. <sup>g</sup>Anemia was defined as hemoglobin concentrations less than 11.0 g/dL in children 0-5 years of age and 12.0 g/dL in children 5-12 years of age<sup>6</sup>. <sup>h</sup>HFA z-score is a standardized value of vertical height by age where children below two standard deviations are considered stunted, or abnormally short for their age (a condition secondary to malnutrition). <sup>i</sup>BMI (Body mass index) for age z-score is a standardized measure which is the ratio of a child's weight to his height squared. <sup>j</sup>WFH (Weight-for-Height) z-score is a standardized measure of acute malnutrition where a value two standard deviations below the mean of zero is considered wasting (a form of severe undernutrition).

Table 2: Results of a generalized linear mixed-model examining the response of hemoglobin concentrations (g/dL) to levels of wildlife consumption

Model 1 73 children			Model 2 77 children		
Variables:	Coefficient (95% CI)	p-value	Variables:	Coefficient (95% CI)	p-value
Wildlife Consumption	0.20 (0.0078, 0.39)	0.041	Wildlife Consumption	0.20 (0.014, 0.38)	0.035
Age (yrs)	0.14 (0.094, 0.19)	<0.0005	Age (yrs)	0.17 (0.12, 0.22)	<0.0005
Sex	0.30 (-0.014, 0.61)	0.061	Annual Income	0.55 (0.30, 0.79)	<0.0005
Annual Income	0.69 (0.39, 1.00)	<0.0005			
Domesticated Meat Consumption	-0.022 (-0.39, 0.34)	0.908			
BMI for age z-score	-0.084 (-0.24, 0.075)	0.300			
Malaria	0.11 (-0.20, 0.43)	0.483			
Deworming	-0.020 (-0.047, 0.0065)	0.139			
Constant	10.66 ( 9.23, 12.09)	<0.0005	Constant	10.48 (10.10, 10.87)	<0.0005

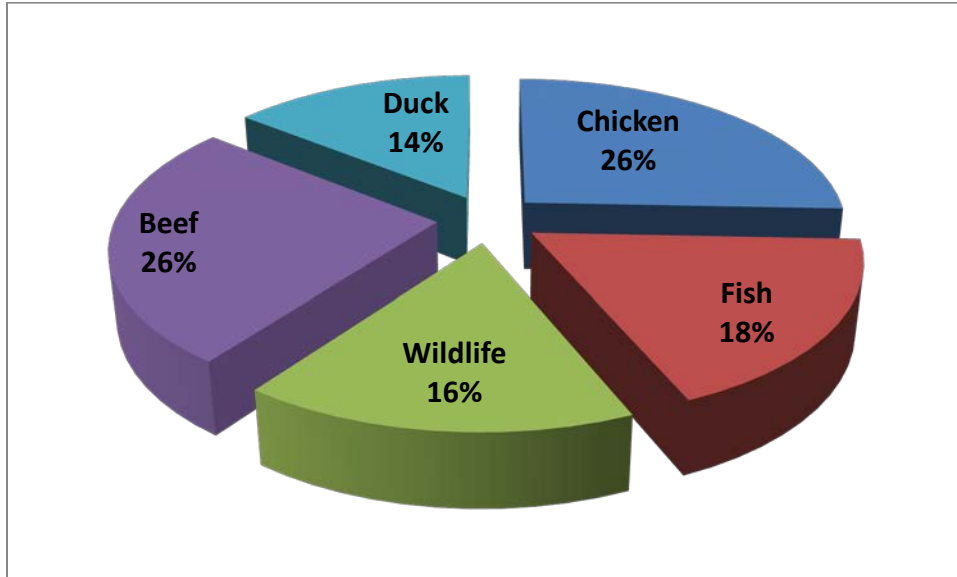
Model 1 has fewer children because of incomplete deworming data for four children. Model 2 includes the significant ( $p < 0.05$ ) variables from Model 1. <sup>a</sup>Wildlife Consumption is the amount of annual wildlife consumption (kg) and was log-transformed due to positive skew. <sup>b</sup>Household size is the unweighted number of individuals in a household. <sup>c</sup>Annual Income is the mean centered log-transformed annual income data. Income was measured as the total value of wages earned, products sold, and items bartered. <sup>d</sup>Domesticated meat consumption is the amount of annual domesticated meat consumption (kg) and was log-transformed due to positive skew. <sup>e</sup>Malaria is a binary variable of whether or not the individual had malaria in the previous month. <sup>f</sup>Deworming is the number of months since the last deworming medication has been taken.

Figure 1: Map of the Makira Protected Area, Madagascar



Featured is a map of the Makira Protected Area (outlined in red) in northeastern Madagascar. This protected area is located in the Maroantsetra region west of Masoala National Park.

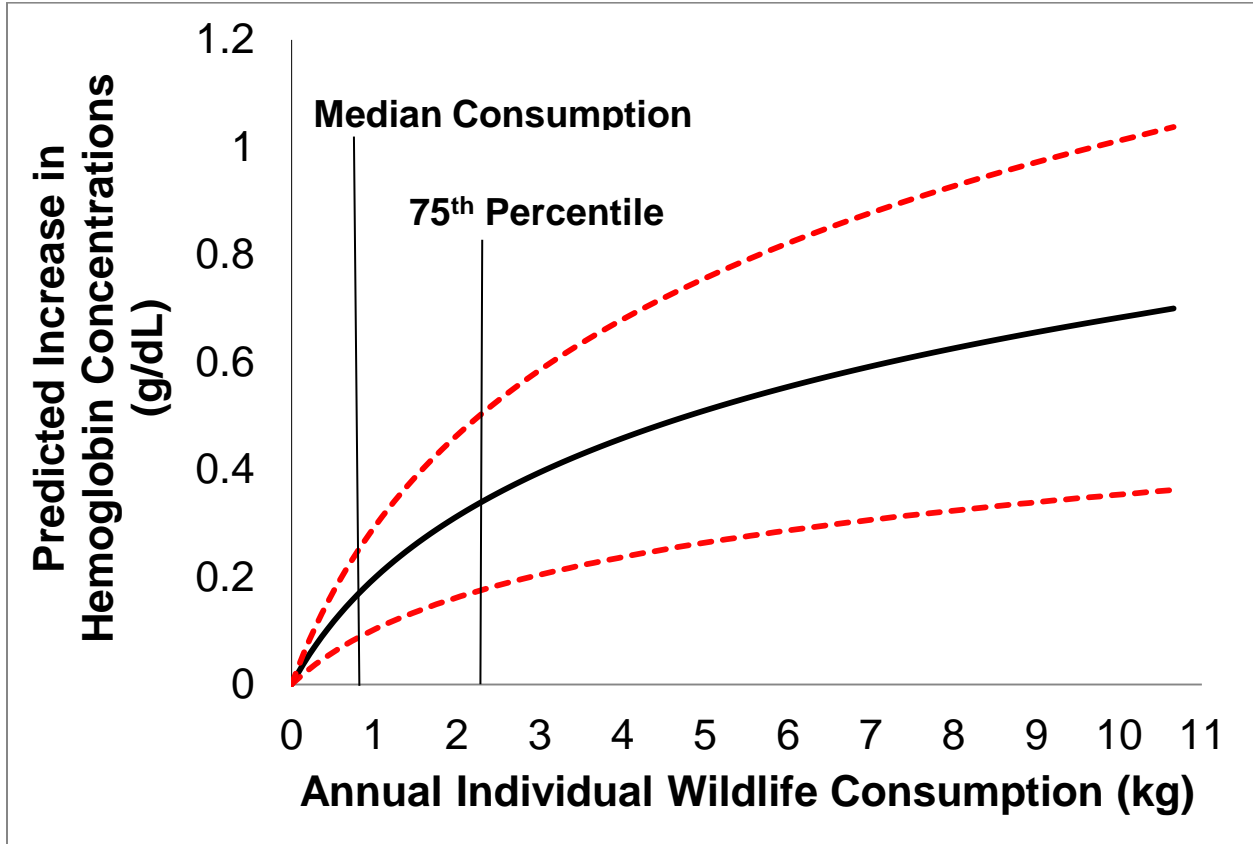
Figure 2: Diet Composition of an Average Malagasy Diet in the Study Population



Twenty-eight households recorded the weight and types of meats consumed on a daily basis for a duration of one year. Kitchen scales were used to measure the weight of dressed meat. On average across households, 16% of animal source foods consumed by mass were derived from wildlife.

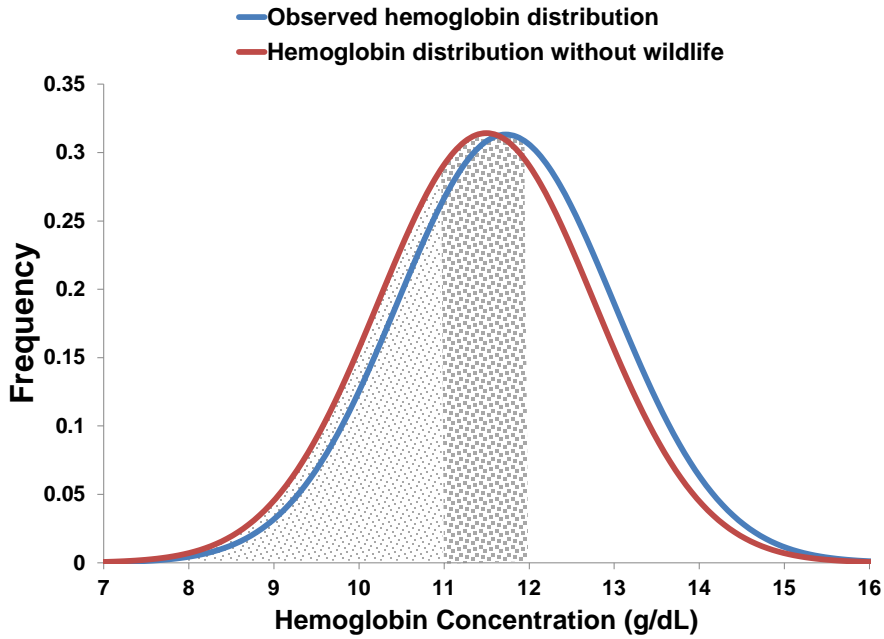


Figure 3: The impact of wildlife consumption on children's hemoglobin concentrations



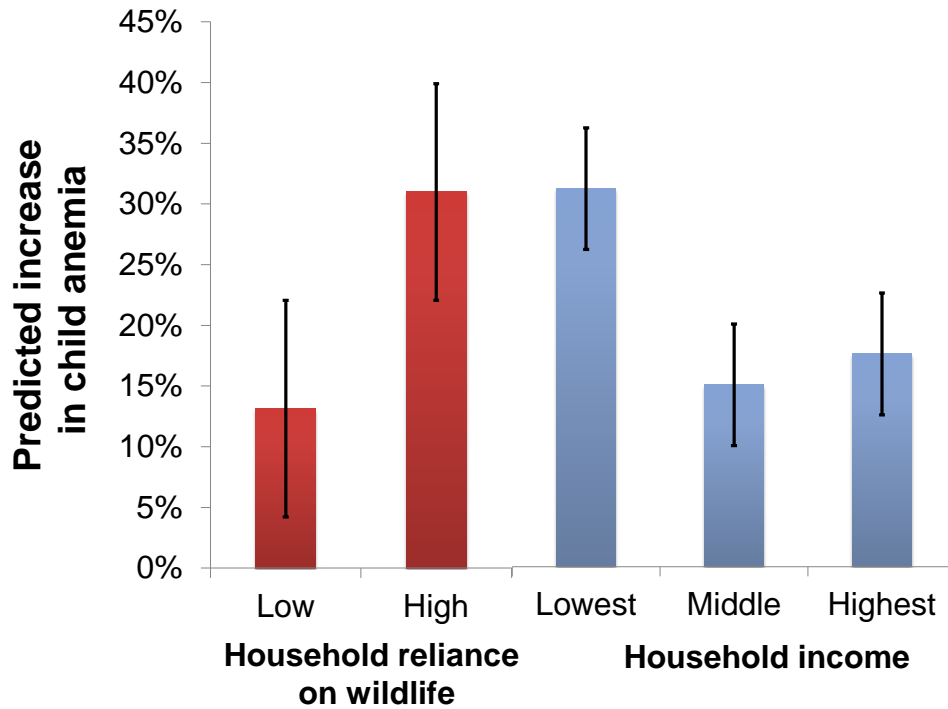
Bootstrapped estimates of the impact of wildlife consumption on hemoglobin concentrations in Malagasy children show a log-linear relationship (mean=black line, 95% CI=red). Household wildlife consumption was measured with daily diet calendars and individual-level consumption was calculated from direct observation of intra-household allocation. As a point of reference, increases in hemoglobin concentration between 0.85 to 1.13 g/dL are expected from iron supplementation efficacy trials (Stolzfus *et al.* 2004).

Figure 4: Removing access to wildlife causes a downward shift in population-level hemoglobin concentrations



A lack of wildlife for consumption would cause a downward shift in the observed population hemoglobin distribution (blue) to a predicted future population (red). Anemia was defined as below 11.0 g/dL (small dots) for children less than 5 years of age and below 12.0 g/dL (large dots) for children 5-12 years old. This downward shift in hemoglobin would cause an estimated 30% increase in the number of children suffering from anemia.

Figure 5: Predicted increase of anaemic children from wildlife loss by household-level characteristics



Wildlife loss induces major increases in childhood anemia that is modified by household-level characteristics. Predictive models of the association between wildlife consumption and children’s hemoglobin concentrations demonstrate that removing wildlife from the diet engenders a disproportionate risk of developing anemia in households with a high reliance on wildlife (odds ratio = 4.00, 95% CI: 1.90, 8.40) and in low-income households (odds ratio = 3.05, 95% CI: 1.29, 7.45). Thresholds for anemia were defined as hemoglobin concentrations below 11.0 g/dL for children under age five and below 12.0 g/dL for children 5-12 years old. Income is a composite of products sold, wages earned and items bartered and was split into tertiles according to natural breaks in the income variable.

## CHAPTER 4

### The Predicted Health Consequences of Lost Access to Wildlife in Madagascar

#### Introduction

Global environmental transformation has triggered well-studied impacts on ecological interactions and biodiversity (Terborgh et al. 2001, Pauly et al. 2003, Butchart et al. 2010) but the effects of damaged ecosystems and diminished access to natural resources on human health is less understood (Chivian & Bernstein 2004). Mammalian wildlife not only influences ecological systems but also serves as a primary source of meat to hundreds of millions of people throughout the developing world (Brashares et al. in press). The consumption of animal-source foods is important in providing essential micronutrients to humans in bioavailable forms (Neumann et al. 2003). In a cohort of pre-adolescent children in Madagascar, Golden et al. (in review) demonstrated that removing access to wildlife could induce a downward shift in population-level hemoglobin of 0.7 g/dL and a 30% increase in the numbers of children suffering from anemia, the most globally-prominent nutritional disorder (Beard 2008). Past research has demonstrated the relationship between wildlife consumption and hemoglobin concentrations, a nutritional biomarker (Chapter 3). In this chapter, I consider the long-term health consequences of removing local access to wildlife by examining the published literature that links shifts in hemoglobin or changes in anemia to subsequent health impairment.

Throughout the developing world, over 300 million people rely on forest products for nutritional support (Pimentel et al. 1997). The people most at risk for nutritional deficiencies are those who are economically and geographically marginalized and thus are heavily reliant on free forest products. These same people are at greatest risk of micronutrient deficiencies because most animal-source foods, such as domesticated meats, are often prohibitively expensive (Steinfeld 2003). Wildlife plays an important role in human nutrition as a low-cost forest product that buffers vulnerable populations at the fringe of health from becoming further nutritionally deficient. Animal-source foods are the primary source for bioavailable iron in diets (Murphy & Allen 2003) and iron is generally the most limiting factor in developing country diets (Gibson et al. 1998, Dewey 2007).

This study linking anemia to wildlife availability and access seeks to provide an improved understanding of the ultimate determinants of anemia, a widespread and critically important nutritional disorder. Anemia is a disease characterized by a deficiency in red blood cells, hemoglobin (an iron-containing protein whose main function is oxygen transportation in humans) or both (Fararouei et al. 2010). Iron is critical to many physiological processes including electron transportation and synthesis of DNA, neurotransmitter and myelin (Beard 2001, 2003). Anemia as a general disorder has multiple causal pathways that may be genetic (i.e. hemoglobinopathies such as sickle-cell anemia), infectious (i.e. malaria, intestinal parasites, etc.) or nutritional (i.e. deficiencies in iron, folate, copper, and vitamins A and B<sub>12</sub>; Beard 2001). Although there are multiple etiologies, the majority of anemia cases worldwide are a result of iron deficiency, and this is the most common nutritional disease globally affecting one-fourth of the world's population (WHO 2008). In a previous study linking wildlife consumption to rates of

anemia (see Chapter 3), Golden et al. (in review) was unable to measure biomarkers in addition to hemoglobin; however, they controlled for the incidence of malaria and the time passed since last deworming to remove potential non-nutritional factors likely to induce anemia. Further, their analysis demonstrated that hemoglobin co-varied with wildlife consumption, suggesting that the majority of anemia cases observed could be characterized as iron-deficiency anemia.

Iron-deficiency anemia (IDA) is inequitable in its distribution, with individuals experiencing rapid growth most likely to be iron-deficient (e.g. young children, adolescents and pregnant women; WHO 2008). Estimated global anemia prevalence is 47.4% in preschool-aged children, 41.8% in pregnant women and 30.2% in non-pregnant women (McLean 2008). Because of the critical role of iron in metabolic processes, reductions in hemoglobin on an individual and population level may induce a breadth of health sequelae from nearly all health domains. In this chapter, I use results from a review of the literature to describe the potential short- and long-term effects of iron-deficiency anemia in two ways: 1) by linking shifts in hemoglobin to dose-response health effects using previously published studies and then 2) demonstrating the impact on health and economies of predicted increases in the future prevalence of anemia in our study population (see Chapter 3).

## **Methods**

There are several components to this chapter that I merged together to develop a model of the health consequences of losing access to wildlife for pre-adolescent children and reproductive-aged women in the Makira Protected Area. First, I conducted a review of the literature to determine previously established associations between either changes in hemoglobin or changes in anemia status and subsequential health conditions. Second, I developed estimates of population prevalences of anemia in our study area based on cross-sectional health data from the Makira Protected Area. Then, I used the model from Chapter 3 linking removal of wildlife to changes in hemoglobin concentrations to the population-level hemoglobin data in this chapter to determine the changes in the prevalence of anemia by sub-population (i.e. children <5 years old, children 5-12, pregnant and breastfeeding women, non-pregnant women and men). Finally, I merged the model of changes in hemoglobin and changes in anemia to anticipated future health outcomes that have been witnessed in human populations elsewhere in the published literature.

### *Literature Review*

I conducted a review of the English-language literature since 1970 in ISI Web of Knowledge linking iron-deficiency anemia (or its associated biomarkers) to consequential health outcomes (Fig. 1; see Appendix 4.1 for search criteria). After my initial search results, I removed studies that used animal models and ones that investigated the effectiveness of iron supplementation but did not use biomarkers to look at health effects. Additionally, I removed studies that looked solely at iron deficiency and not at iron-deficiency anemia, although many of these omitted studies demonstrated that anemia is only the tip of the iceberg with regard to iron-deficiency and that lesser health effects can even be seen in iron deficiency without anemia. This literature

review resulted in 76 studies ultimately linking anemia and/or hemoglobin to health consequences from at least one health domain other than nutrition.

### *Quality Assessment*

The past research of Golden et al. (in review) focused on children less than twelve years of age in whom the prevalence of anemia was 42%. As the study population is a group of pre-adolescent children situated in a remote rainforest area in northeastern Madagascar, it is difficult to utilize studies from elsewhere to inform exact predictions of what will occur in our study site. I conducted quality assessments (McMaster 2011) for each paper in this review to evaluate the epidemiological rigor with which the studies were conducted, analyzed and reported (See Appendix 4.2). The final scores for each paper included three categories: strong (1), moderate (2) and weak (3) where the strongest papers are likely to be the most applicable to this study system. Anemia is a disease precipitated by several causal pathways and mediated by a number of confounding variables that vary across the globe. However, the purpose of this study is to investigate the possible health consequences of increases in anemia due to lost access to wildlife arising from either conservation enforcement or unsustainable use (See Chapter 3 for discussion). Therefore, we focused on studies linking changes in hemoglobin or anemia to consequential health effects in pre-adolescent children or reproductive-aged women.

### *Health Assessment and Projected Health Changes*

In 2009, my research team and I surveyed 165 households in 16 villages adjacent to the Makira Protected Area, Madagascar, and collected hemoglobin data on 876 individuals (Table 1). Households were surveyed following a process of systematic random sampling from self-generated census lists (Chapter 2). All individuals available within selected households were sampled. For further information regarding clinical sampling, hemoglobin testing, and other data collected, please refer to Chapter 3. We also present new data from the study by Golden et al. (in review) concerning the duration of anemia in the prospective cohort study and the predicted future duration of anemia if access to wildlife were to be lost. We assumed that pre-adolescent children and women of reproductive age in the larger human population in these 16 villages would show a similar response to the model restricting wildlife from the diet as individuals from a small, prospective cohort study (Chapter 3). There are no major reasons why we would anticipate the biological response of removing iron-rich foods from an individual's diet to be different across sub-populations. In fact, women of reproductive age require more iron daily than pre-adolescent children (Stoltzfus et al. 2004, Stoltzfus 2008) and thus the estimates of projected health effects may be conservative.

### *Model Synthesis*

I used the cross-sectional health data described above, in conjunction with the longitudinal data collected during the prospective cohort study (Chapter 3) and the results from the review, to

model the projected future health effects of lost access to wildlife. I merged the model predicting changes in the prevalence of anemia and the distribution of population-level hemoglobin concentrations with the literature presented here in the review to predict possible future health outcomes. Additionally, I followed Horton and Ross (2003) to calculate the economic costs due to reduced productivity from both IDA in adults (loss of physical capacity) and in children (cognitive deficits that cause future economic loss). To calculate the economic costs of reduced productivity in adulthood, I used an adapted version of Horton and Ross' equation (2003):

$$\text{Equation 1: Physical loss} = \text{WT} * \text{D} * \text{WS} * \text{GDP/cap(k)} \times \text{Pr(adult)}$$

where WT was the work type (percentage of the adult population engaged in either heavy manual labor or physically easier work), D was the degree to which anemia caused a reduction in that labor (12% for heavy manual labor and 5% for easier work), WS was the wage share for Madagascar, and Pr(adult) was the prevalence of adulthood anemia. A wage share is an indicator of the distribution of income between capital and labor and was not available for Madagascar. It was thus calculated as the average of all available wage shares for sub-Saharan Africa (29%, International Labour Office 2009). GDP per capita in Madagascar was \$488 (UNdata 2008). Similarly, to calculate the future economic costs lost from reduced labor productivity due to IDA experienced in childhood, I again used an adapted version of Horton and Ross' equation (2003):

$$\text{Equation 2: Cognitive loss} = 2.5\% * \text{WS} * \text{GDP/cap} * \text{Pr(child)}$$

where 2.5% was the anticipated reduction on future productivity due to childhood IDA, WS was the wage share for Madagascar and Pr(child) was the prevalence of childhood anemia.

## RESULTS

### *Literature Review*

Through the literature review, I found that iron-deficiency anemia (and its relevant biomarkers) seems to be associated with the following health domains (Fig. 2): physiology, neurologic function, behavior, cognition, motor function, physical capacity, growth and morbidity/mortality. Below, I organize the results of my literature search into these domains.

### *Physiology (9 Studies, average rigor rating: 2.2):*

The physiological health consequences of anemia affect the cardiac, endocrine, neural, renal and hematological systems. Cardiac system effects included a shorter time taken to revert to the basal pulse rate (recovery time) by non-anemic girls compared to anemic girls (Sen & Kanani 2006). Endocrine effects included an elevated serum prolactin pattern in response to stress (Mahajan et al. 2008) and could persist more than ten years after iron-deficiency in infancy, demonstrating

the long-lasting effects to dopaminergic inhibition from iron deficiency in infancy (Felt et al. 2006).

Physiological effects in renal function showed that children with iron deficiency anemia have impaired renal tubular function as demonstrated by a significantly higher ( $p < 0.05$ ) mean fractional excretion of sodium (Ozcay et al. 2003, Sadeghi-Bojd et al. 2009) and mean urinary N-acetyl- $\beta$ -D glucosaminidase/creatinine in IDA children (Ozcay et al. 2003). Further, this association has been demonstrated at the biomarker level showing hemoglobin levels negatively correlated ( $r = -0.44$ ,  $P = 0.015$ ) with urinary N-acetyl- $\beta$ -D-glucosaminidase/creatinine (Ozcay et al. 2003).

Hematological effects of IDA were also observed in epidemiological research. Anemia was associated with a more rapid decline in CD4 counts in HIV-positive pregnant Tanzanian women, measured as time to a 50% drop in CD4 cell count from baseline (O'Brien et al. 2005). This has important implications in how HIV is managed and the risk of nutritional deficiencies in accelerating pre-mature death. IDA is also linked to significant increases in both maternal and fetal insulin-like growth factor 1 which is important in promoting childhood growth (Mahajan et al. 2008), a potential benefit of experiencing IDA. In contrast, increases in the severity of anemia in mothers were associated with decreased  $T_3$  (a thyroid hormone) in maternal blood (Mahajan et al. 2008), leading to growth restriction or hypothyroidism/cretinism in infants. Iron-deficiency without anemia was also shown to affect thyroid concentrations in young girls in Iran (Eftekhari et al. 2006).

Neurological physiology is also affected by IDA; anemic infants had significantly higher central conduction time, indicating a slower nerve conduction velocity (Roncagliolo et al. 1998). Also, iron-deficient anemic infants were shown to have sleep spindles with reduced density and lower frequency (Peirano et al. 2007), illuminating the mechanism by which sleep patterns and duration were affected. IDA infants had a lower initial eye-blink rate as compared to non-anemic infants (Lozoff et al. 2010). All of these prior results relevant to neurologic function indicate an association between iron-deficiency anemia and reduced dopamine function. Although these highly specific results may seem unimportant, the greater clinical importance of a reduced dopaminergic response likely affects movement, motivation, cognition, and hormone release (Lozoff et al. 2010).

#### *Neurologic Function (6 studies, average rigor rating: 2.2):*

Iron-deficiency anemia (IDA) has been hypothesized to be associated with neurotransmitter and myelin synthesis (Beard 2001). These impacts on neurotransmission can be seen where IDA infants exhibited lower neural transmission in the auditory system despite one year of iron therapy (Peirano et al. 2009). Delays due to IDA were also found in both auditory and visual systems with a large (1-1.2 SD) magnitude of effect in four-year olds (Algarin et al. 2003, Walter 2003), and similar systems affected in adults (Khedr et al. 2008). When looking at nearly all brain wave latencies in the temporal, parietal and occipital regions, appropriate iron therapy was shown to improve brain neural conduction in post-treatment IDA individuals (Kececi & Degirmenci 2008).



IDA is also clearly linked to sleep behavior and memory. IDA preschool-aged children showed altered REM sleep patterns, longer inter-spindle intervals in both NREM (non-rapid-eye-movement) sleep stage 2 and slow-wave-sleep, and delayed transmission in both auditory and visual systems (Peirano et al. 2007, Peirano et al. 2009). These impacts on altered REM sleep may represent more severe effects like the maladjustment of the internal temporal order which may interrupt optimal neurologic function during periods individuals are both asleep and awake (Peirano et al. 2009). Non-anemic infants showed a greater attentive response to the mother and a greater updating of memory in recognition of strangers at nine months (Burden et al. 2007). Furthermore, this pattern of responses was not evident in the IDA group until one year of age, indicating delayed development (Burden et al. 2007).

*Behavior (11 studies, average rigor rating: 2.2):*

Iron-deficiency anemia (IDA) has significant and potentially long-lasting behavioral effects during wakefulness and sleep. Chronic IDA infants exhibited less positive affect, less frustration tolerance, more passive behavior, and more physical self-soothing when stressed (Chang et al. 2011). If the anemia was corrected before two years of age, these behaviors were no different than non-anemic infants (Chang et al. 2011). Neonatal hemoglobin and serum iron were positively associated with negative emotionality and negatively associated with alertness and soothability (Wachs et al. 2005). IDA infants were two times more likely than iron-sufficient infants (48 vs. 24%) to never show positive affect and showed significantly more shyness and engagement delay, and significantly less orientation-engagement and soothability (Lozoff et al. 2008). Correcting iron-deficiency anemia caused improvements in mental development in Chilean infants which was associated with improvements in attention span and cooperative behavior (Walter 2003). IDA was associated with differences in reported sleep characterized by shorter night sleep duration and higher frequency of night waking in 6-18 month old infants from Zanzibar and Nepal (Kordas et al. 2008). Iron-deficiency anemic students' showed significantly improved concentration and scholastic achievement when provided iron supplementation than IDA students who received placebo and whose anemia was not corrected (Soemantri et al. 1985). Anemic students performed significantly worse in learning and positive task orientation (Palti et al. 1985).

IDA also influences mother-child relationships. In a group of five-year old children, mother-child reciprocity during a structured task (e.g., eye contact, shared positive affect, turn taking) was more likely to be lower in the chronic iron deficiency group compared to the iron-sufficient group and the lack of positive affect persisted from infancy despite the correction of IDA (Corapci et al. 2006). Mother ratings of externalizing and internalizing problems from age 5 to 11–14 years were higher for the chronic iron deficiency groups but these problems did not persist beyond adolescence (Corapci et al. 2010). During feeding behavior, mothers of IDA infants responded with significantly less sensitivity to infant cues and less socio-emotional fostering behavior (effect size 0.8-1.0 SD) than mothers of non-anemic infants (Armony-Sivan et al. 2010). IDA in mothers of children with breath-holding spells was found to be significantly higher as compared to controls, suggesting that iron deficiency anemia rather than behavioral/psychosocial problems of mothers affects the development of breath-holding spells (Hudaoglu et al. 2006).

*Cognition (24 studies, average rigor rating: 2.1):*

The relationship between IDA and cognition is perhaps the best studied health domain. Mechanistically, iron is prioritized to red cells at the expense of other tissues, most importantly the brain (Lozoff & Georgieff 2006), and several studies have documented the effects of anemia and shifts in hemoglobin on deficits in IQ, memory, language and scholastic achievement. Inner-city American infants with IDA exhibited poorer recognition memory (Carter et al. 2010). Older Egyptian children (mean age 9.5 years) treated for anemia showed higher efficiency in matching familiar figures than untreated peers (Pollitt et al. 1985) and anemic adolescent Indian girls scored lower on visual memory tests (Sen & Kanani 2006). Academic and communication delays were observed in anemic children (Gupta et al. 2010) and anemic infants failed in language capabilities specifically when the nutritional disorder lasted for longer than three months (Walter et al. 1989). Correcting for anemia through iron supplementation improved language abilities (DeAndraca et al. 1990), specifically by 0.8 points (range 0.2-1.4) on a 20 point scale (Stoltzfus et al. 2001). These linguistic deficits are shown to persist from infancy until at least five years of age (Corapci et al. 2006), demonstrating the importance of detecting and treating anemia early.

IDA also has documented links to intellectual deficits as measured by IQ. Both before and after treating IDA, there was a significant correlation between hemoglobin levels and mental state examination scores, and total verbal and performance IQ scores (Khedr et al. 2008). Hemoglobin at 6 and 36 months was associated with IQ at four years of age (Wasserman et al. 1994). Further, anemia in infancy predicted an average six point deficit in IQ at age seven exhibiting the long-lasting damage of anemia in infancy (Cantwell 1974). A prospective cohort study in Israel showed that hemoglobin concentration at nine months was correlated with IQ at 5 years with each 1.0g/dL incremental increase in hemoglobin associated with a 1.75 point higher IQ score (Palti et al. 1983). Similarly, IDA infants showed 5 point deficits in IQ as compared to non-anemic infants at 5 years of age with these cognitive deficits persisting until age 10 (Walter 2003), and even to 19 years of age with effects heightened by low socio-economic status (Lukowski et al. 2010). Further substantiating the invariability of IQ trajectories, there was a strong correlation (0.62 - 0.65) between IQ scores at age 6-8 with those at age 17 (Jensen 1980). Anemia in adulthood significantly undermines cognitive functions and hemoglobin concentration shows a dose-response for better cognitive ability even for non-anemic individuals (Petranovic et al. 2008).

Aside from IQ, IDA also negatively affected cognitive achievement in infants (Soemantri et al. 1985) and scholastic achievement in school-aged children and adolescents (Li 2009). Further, IDA infants tested significantly lower on cognitive tests as compared to iron-sufficient infants (0.5 - 1.5 standard deviations, Lozoff 1988; 0.5 - 1 standard deviations, Pollitt 1993; and 0.3 - 0.5 standard deviations, Sungthong et al. 2002) and 0.1 - 0.7 standard deviations lower on a variety of audio-visual, motor and intelligence tests (Lozoff et al. 1991). Iron-deficient American children regardless of anemia status had twice the risk (OR 2.3, 95% CI 1.1-4.4) of scoring below average in mathematics (Halterman et al. 2001), an effect which would likely be heightened if IDA children were compared to iron-sufficient children. Correction for anemia showed vast improvements in cognitive deficits (Akman et al. 2004), and mental scores increased by 18.8 points (over 1 S.D.,  $p < 0.001$ ) among IDA-treated Indonesian infants (Idjradinata & Pollitt 1993) and by 6 - 15 points on average among studies scoring post-treatment cognitive improvements in IDA-treated infants (Lozoff et al. 2006).

After adjusting for the child's confounding factors (sex, birth weight and birth month) and maternal confounding factors (parity, smoking, mental status, whether pregnancy was wanted or not, education, social class and marital status), only a mother's hemoglobin concentration measured at the ninth month was significantly associated with her child's scholastic performance in a cohort of almost 12,000 mother-child dyads in Finland (Fararouei et al. 2010). If a mother's hemoglobin concentrations were  $\geq 11.0$  g/L at three measurement points (representing maternal health throughout the course of pregnancy), her child performed better in school at age 14 and 16 and also had an increased odds (OR = 1.14,  $p=0.04$ ) of attaining higher education at age 31 (Fararouei et al. 2010).

In Costa Rica, declines in infant mental development were found at hemoglobin concentrations less than 10.0 g/dL (Lozoff et al. 1987, Lozoff 1989); similarly, these children performed significantly worse on mental exams at school entry if hemoglobin concentrations were still below that threshold (Lozoff et al. 1991). Hemoglobin has also been linked to the speed at which individuals can complete cognitive tests (Petranovic et al. 2008). In a cross-sectional study in the US, each 1g/dL drop in hemoglobin was associated with a 1.28 (95% CI: 1.05, 1.60) increased odds of "mild to moderate mental retardation" in school-aged children (Hurtado et al. 1999). Similarly, in Costa Rica, 48 children who had been treated for severe chronic iron deficiency in infancy were 2-3 times more likely to repeat a grade or be referred for tutoring or special education and still scored lower in mathematics and writing achievement at ages 11-14 as compared to iron-sufficient children (Lozoff et al. 2000). Not only has anemia been shown to be negatively associated with individual and population-level economic attainment (Horton & Ross 2003), but the extra care required for students suffering chronic IDA places an additional economic burden on public education.

*Motor Function (18 studies, average rigor rating: 1.9):*

IDA during infancy has been linked to delayed and/or impaired motor development with long-lasting effects. IDA infants had significantly lower motor scores (Shafir et al. 2006) and there were linear effects of iron status on developmental milestones (Shafir et al. 2008). Mean corpuscular hemoglobin concentration was significantly associated with development, motor and social quotients at 24 months (Dommergues et al. 1989). Controlling for urban culture and geography (China, Ghana and African-Americans in the US), there were linear trends between infants' iron status and gross motor milestones and fine motor skills with small objects (Angulo-Barroso et al. 2011). Motor function impairment impacts other health domains as well where motor scores in infancy predict cognitive test performance not only later in childhood but also at 18 years of age (Pollitt & Gorman 1990).

IDA's effect on motor function includes locomotion impairment. IDA infants showed significantly poorer upper extremity control (Shafir et al. 2009) and only 19% of iron-deficient and IDA infants could stand alone, while 34% of iron-sufficient infants could stand alone and 19% were already walking alone (Shafir et al. 2008). In walking Zanzibari young children, hemoglobin concentration was positively associated with activity and locomotion (Olney et al. 2007). In a large population study of British children, hemoglobin concentrations under 9.5g/dL at 8 months of age were associated with impaired locomotion at 18 months (Sherriff et al. 2001).

Walking skills are important because children become more sociable and affectionate after the acquisition of walking skills (Birengen et al. 1995; Campos et al. 1992). Thus, delays in locomotion and motor activity delay access to external sources of emotional regulation and constrain the development of self-sufficiency and independence. It follows that anemic children are at risk of delaying the acquisition of developmentally appropriate systems of emotional regulation (Pollitt 2001).

There is mixed evidence on whether the effects of IDA on motor skills can be reversed through treating anemia. In one study, one year of iron therapy was unable to reverse the motor function effects of iron-deficiency in infancy (Angulo-Kinzler et al. 2002). In a cohort of American neonates given iron treatment and then followed to seven years, motor deficits were still observed in the formerly anemic group, exhibiting a higher incidence of "soft signs" (i.e., balancing clumsiness and difficulty with tandem walking and repetitive hand and foot movements, Cantwell 1974). Epidemiological research has shown that one to two-year old infants showed a decline in motor development when hemoglobin concentrations fell below 10.5 g/dL, with a mean motor score 10 points lower (Lozoff et al. 1987, Lozoff 1989). These same IDA children at 5 years of age showed significantly lower scores on the Bruininks-Oseretsky Test of Motor Proficiency, demonstrating deficits in both gross and fine motor skills (Lozoff et al. 1991). After 10-12 years of follow-up, and an average increase of 3.7 g/dL (very large change), no reversal in development was observed (Lozoff et al. 2000).

Other evidence showed that iron deficient children under five showed significantly lower psychomotor developmental test scores compared to iron-sufficient children but these deficits were corrected with iron treatment (Akman et al. 2004, Lind et al. 2004). Iron supplementation of anemic children led to greater improvement in motor development in IDA infants than non-anemic infants in Indonesia (Harahap et al. 2000) and had similar effects for Chilean children (DeAndraca 1990) and Zanzibari children with hemoglobin less than 9.0 g/dL (Stoltzfus et al. 2001). Duration of the anemia and degree of the treatment effect also clearly matter because infants that have hemoglobin under 10.5 g/dL for more than three months had significantly lower motor scores as compared to other infants that were either non-anemic or did not have prolonged anemia (Walter et al. 1989). Also, children who experienced an increase in hemoglobin concentration  $>2$  g/dL were more than two times as likely (37% vs. 16%) as children below this threshold to achieve the average psychomotor development rate (Aukett et al. 1986). Although the evidence for reversing the effect of early IDA is mixed, it is clear that there is a strong link between IDA and motor function and infancy appears to be a critical period.

*Physical Capacity (6 studies, average rigor rating: 1.8):*

In a systematic review of the evidence for reduced work capacity due to iron-deficiency anemia, 29 research reports were evaluated of both human and animal evidence. This survey highlighted a strong causal effect of IDA on aerobic capacity (Haas & Brownlie 2001, see this paper for more detailed information on studies not captured in this review). The mechanism for this causal pathway is likely through reduced oxygen transport associated with reduced hemoglobin from anemia (Haas & Brownlie 2001) where IDA restricts the oxygen supply and muscle respiratory capacity (Zhu & Haas 1998).

The effects of anemia on physical capacity are not limited to particular ages or windows of vulnerability. The most classic example is Viteri and Torun's (1974) pioneering study that showed evidence of a linear dose-response relationship between hemoglobin concentrations and performance on the Harvard step test (Viteri & Torun 1974). Looking at a spectrum of cardiac output, the maximal expendable workload (kcal/min) is 0.8 – 1.3 times an individual's hemoglobin concentrations (Viteri & Torun 1974), providing concrete evidence of the importance of anemia to deficits in physical capacity. Significantly reduced maximal exercise tolerance and labor was observed in anemic subjects, who transported 15% less oxygen per pulse than did non-anemic subjects (Edgerton et al. 1979). Five year old Costa Rican children who had experienced chronic iron deficiency in infancy exhibited impaired physical performance (Corapci et al. 2006). There is no specific window of vulnerability for anemia's effects on physical capacity and this health domain may be the most likely to be reversible. During a randomized control trial trying to correct anemia in adolescent Indian schoolgirls, the mean number of steps climbed was significantly higher in the group whose hemoglobin increased by greater than 1 g/dL as compared to the group whose hemoglobin concentration increased by less than 1g/dL (Sen & Kanani 2006, 2009).

Anemia not only impacts exercise capacity but also impacts manual labor and non-manual work capacity. Physical labor work output differences are observed where anemic workers were unable to compete with nonanemic workers (17% differential in Indonesian rubber tappers, Basta et al. 1979; and 5% differential in Chinese female cotton mill workers, Li et al. 1994). In one study, the average reduction in productivity was estimated to be 20% for an anemic person (Yip 1994). In a given country where 50% of women and 20% of men are afflicted with anemia and the sexes are equally represented in the work force, IDA would induce a total loss of 5-7% of the national economic output (Yip 1994).

*Growth (3 studies, average rigor rating: 2.3):*

IDA affects maternal anthropometry, fetal development and offspring growth. Severely anemic mothers had a significantly low pre- and post-pregnancy weight and both a significantly decreased maternal abdominal circumference and fundal height (an anthropometric measure of the size of the uterus to determine fetal growth and development; Mahajan et al. 2008). Severe maternal anemia also appears to affect the offspring where newborns exhibit significant reductions in ponderal index (indicating increased leanness), birth weight and placental weight (Mahajan et al. 2008). Similarly, a linear dose-response relationship is observed between maternal serum ferritin and both birthweight and birth length (Vazirinejad et al. 2007). A dose-response relationship was also found in degrees of severity for anemia. Increasing maternal anemia severity (mild- hemoglobin 9.5 – 12.0 g/dL and moderate- hemoglobin < 9.5 g/dL) was associated with lower birthweight (139 and 192 grams, respectively; Ronnenberg et al. 2004). As a binary variable, maternal IDA was associated with a 242-g decrease in birthweight (Ronnenberg et al. 2004). Mothers with moderate anemia had 6.5 times greater risk of having low birthweight babies (95% CI: 1.6, 26.7; p=0.009) and 4.6 times greater risk of having newborns with fetal growth restriction (95% CI: 1.5, 13.5; p=0.006) as compared to non-anemic mothers (Ronnenberg et al. 2004). In Chinese women, even preconception anemia was associated with reduced infant growth and an increased risk of possible adverse pregnancy

outcomes (Ronnenberg et al. 2004) and this result was mirrored in animal models (Mihaila et al. 2011). The effect of anemia on growth is potentially reversible if treated within an early childhood window of vulnerability. After correcting for anemia in 2-5 year old Indonesian children, there were significantly greater increases ( $p < 0.01$ ) in growth velocity (i.e. height and height-for-age z-score; Angeles et al. 1993). However, if anemia is not addressed in that window of vulnerability, a stunted child (short stature for age) will remain on that stunted trajectory throughout adolescence and adulthood (Li et al. 2004, Adair 2007). Furthermore, these growth trajectories mirror scholastic trajectories with stunted children more likely to repeat grades and drop out of school (Daniels & Adair 2004).

*Morbidity/Mortality (19 studies, average rigor rating: 2.1):*

Iron-deficiency anemia has broad-reaching effects to human wellbeing, including effects to the neurological, renal, cardiovascular and immune systems. IDA may also cause a host of adverse outcomes to pregnant women and their offspring. A mean maternal hemoglobin concentration of 10.0 g/dL or less was statistically associated with a nearly 4-fold increased rate of schizophrenia spectrum disorders (adjusted rate ratio, 3.73; 95% confidence interval, 1.41-9.81;  $P = .008$ ) compared with a mean maternal hemoglobin concentration of 12.0 g/dL or higher, adjusting for maternal education and ethnicity (Insel et al. 2008). For every 1-g/dL increase in mean maternal hemoglobin concentration, a 27% decrease in the rate of schizophrenia spectrum disorders was observed (95% confidence interval, 0.55-0.96;  $p = .02$ ) (Insel et al. 2008). Correction of anemia resulted in a 25% improvement ( $P < 0.05$ ) in previously iron-deficient mothers' depression and stress scales whereas anemic mothers given placebo showed no improvement, demonstrating an association between iron status and depressive symptoms in poor, post-partum African mothers (Beard et al. 2005). Similarly, in a group of Iranian medical students, there was a statistically significant difference ( $p < 0.0001$ ) in hemoglobin concentrations between depressed and healthy individuals (Shariatpanaahi et al. 2007). There was a negative association between hemoglobin concentration on 7<sup>th</sup> day postpartum and depressive symptoms on the 28<sup>th</sup> day postpartum ( $r = -4.26$ ;  $p = 0.009$ , Corwin et al. 2003). Thus, postpartum anemia may put women at a disproportionate risk for developing postpartum depression (Corwin et al. 2003). From a retrospective case-controlled study of Canadian children six months to three years of age, there was nearly a doubled odds (95% CI, 1.02-3.31) of febrile seizures in anemic patients (Hartfield et al. 2009).

In addition to neurologic impairment, IDA can lead to an increased risk of infectious disease. Increases in hemoglobin concentrations may be associated with reduced infection rates for schistosomiasis, although the mechanism of effect is not well-understood (Mwanakasale et al. 2009). In Indian children, anemia was a significant risk factor for persistent diarrhea (OR=3.74, 95% CI: 1.55-9.15, (Umamaheswari et al. 2010). In a prospective cohort study of Israeli Bedouin infants, anemia at six months was an independent risk factor for diarrhea and respiratory illness from 7 to 18 months of age when controlling for prior infection and a variety of other important environmental and socio-economic confounding factors (Levy et al. 2005b). Anemia at 6 months tripled the future risk for diarrhea (95% CI: 1.6-5.3;  $p = 0.001$ ) and doubled the risk for respiratory disease (95% CI: 1.1-3.7;  $p = 0.03$ , Levy et al. 2005b). After correcting for anemia in

2-5 year old Indonesian children, the frequency of fever, respiratory infections and diarrhea became significantly less in the treatment group (Angeles et al. 1993).

There are also links between IDA and a disproportionate risk of non-communicable diseases. In a case-control study of 12-38 month Canadian children, there was a ten-times greater likelihood of previously healthy stroke victims having iron-deficiency anemia than their healthy counterparts (Maguire et al. 2007). Additionally, IDA accounted for more than half of all stroke cases with unknown etiology (Maguire et al. 2007). Several studies have found that anemia is associated with damage to renal tubular function (Ozcay et al. 2003, Sadeghi-Bojd et al. 2009) and a ten-fold increased odds of telogen hair loss in anemic women (Moeinvaziri et al. 2009). Perhaps of greatest concern because of the large scale of the epidemic, IDA has been hypothesized to be a risk factor for obesity because anemia can lead to sluggishness (Neymotin & Sen 2011). Using a nationally representative nutritional survey in the US, a negative association between blood iron levels and individual BMI (Neymotin & Sen 2011), demonstrating the possibly integral role of micronutrient nutrition in preventing the onset of obesity.

Maternal IDA impacts both the health of the mother and that of the offspring, typically leading to adverse outcomes for both (Lozoff & Georgieff 2006). In developing countries, maternal IDA plays a role in troubling 30-50% of pregnancies whereas less than <1% of pregnancies in developed countries are complicated by IDA (Lozoff & Georgieff 2006). In a multivariable analysis, several pregnancy complications were significantly associated with maternal anemia including placental abruption (the placental lining separates from the uterus), placenta previa (the placenta grows in the lowest part of the womb and obstructs the cervix), non-vertex presentation (non-head-first delivery), and labor induction (Levy et al. 2005a). Anemic women required significantly more cesarean sections (20.4% vs. 10.3%;  $p < 0.001$ ) and anemia was an independent risk factor for both preterm delivery (OR = 1.2; 95% CI: 1.1–1.2,  $p < 0.001$ ) and low birthweight (OR = 1.1; 95% CI: 1.1–1.2,  $p = 0.001$ , Levy et al. 2005a). The effects of maternal IDA also have far-reaching effects to the health of the infant. In a hospital-based cohort in South Korea, pregnant women and newborn infants with low hemoglobin concentrations had significantly higher rates of preterm delivery, low Apgar scores (a ten-point scale which measures the physical condition of a newborn) and low birth weight (Lee et al. 2006). Similarly, in a hospital-based retrospective cohort of Israeli women, higher rates of preterm deliveries and low birthweight were observed among anemic women.

Maternal IDA can actually be fatal to both the fetus/infant and the mother. In a retrospective analysis of a nationally representative cohort of American women in 1988, an increased risk of stillbirth (adjusted hazards ratio: 4.4; 95% CI: 1.02, 19.01) was significantly associated with maternal anemia among non-black women (Tomashek et al. 2006) and adverse pregnancy outcomes were also significantly associated with maternal anemia in Chinese women (Ronnenberg et al. 2004). Maternal hemoglobin  $\leq 5$  g/dl in the first trimester was associated with a more than threefold increased risk (hazard ratio CI: 1.4–7.7) of preterm premature rupture of membranes whereas low hemoglobin in the third trimester was protective against premature spontaneous labor (Zhang et al. 2009). It is possible that there is a U-shaped relationship between maternal hemoglobin concentrations and birth outcomes where both dangerously low and high concentrations may produce adverse outcomes (Chang et al. 2011). From a large cohort of

Hungarian mothers, there was a higher rate of preterm birth in anemic pregnant women, which was shown to be reversed by iron supplementation (Banhidy et al. 2011).

IDA is not only linked to increased rates of morbidity but has also been linked to fatal health outcomes. In a specific cohort of HIV-positive Tanzanian women, anemia was found to be significantly associated with an increased risk of all-cause mortality (relative hazard [RH]: 2.06 to 4.56 for moderate to severe anemia, respectively) and AIDS-related mortality (RH: 2.21 to 3.47, for moderate to severe anemia, respectively, O'Brien et al. 2005). Looking for a mechanism of effect, anemia was also associated with a more rapid decline in CD4 counts, a hematological measure that leads to AIDS (O'Brien et al. 2005). This study demonstrates the importance of nutritional status in the treatment and management of infectious disease conditions.

### *Synthesis of Evidence: health consequences of lost access to wildlife*

My review of the literature revealed a complex and powerful array of impacts of IDA on a broad variety of health domains that may be taking place in our study population in Madagascar. Some of these impacts are quantified to the degree that they can be applied to alternative scenarios. We applied these models to predict the possible consequential health effects of removing wildlife from the diet in rural households in northeastern Madagascar. Typically, both children and women are at high risk for developing anemia because of increased iron need from growth and menstruation (WHO 2008). In our study site, however, we found children to be at the greatest risk of anemia, followed by men, and then by women (Table 1). Here, we model the possible future health effects that this population may experience if access to wildlife (and the associated iron-rich animal-source foods) were to be lost. The mean change in hemoglobin from lost access to wildlife in a cohort of pre-adolescent Malagasy children was 0.23 g/dL (SD: 0.17 g/dL, range of 95% CI: 0-1.03 g/dL; Chapter 3).

### *Projected health effects in children*

Using the empirical relationship between wildlife consumption and hemoglobin concentrations, we can estimate the potential future health effects that could occur if access to wildlife were to be lost. Under this scenario, we predict that approximately 77% of children under two years of age would have a hemoglobin concentration less than 10.5 g/dL, which would denote significant declines in motor development and a mean motor development score 10 points lower than iron-sufficient infants (Lozoff et al. 1987, Lozoff 1989). The duration of the deficiency also matters where anemia lasting longer than three months predicted significantly lower motor scores (Walter et al. 1989). The average duration of anemia in a cohort of pre-adolescent children was 4.8 months (SD: 3.3 months; Golden et al. in review), with 57% of children experiencing anemia for more than 3 months in their year-long study (Fig. 3). Removing access to wildlife could thus increase the prevalence of children experiencing anemia for more than three months to 73% (mean 6.1 months, SD: 3.4).



In addition to the effects on motor function, IDA will also have significant effects on cognition in children which will last until adulthood. The prevalence of anemia in infants (i.e. less than two years of age) was 91% and is predicted to increase to 92% with lost access to wildlife, a very small change because the prevalence is already so elevated. These rates of anemia would equate to the majority of this population experiencing a five-point drop in IQ at age five (Walter 2003) and cognitive deficits that would persist through adolescence and adulthood (Walter 2003, Lukowski et al. 2010), as compared to healthy infants elsewhere. Another example of the effects of increased rates of anemia on the efficiency of test-taking showed that this projected population would require an additional twelve seconds to complete a cognitive test (Petranovic et al. 2008). Further, there would be a 6% increased probability of “mild to moderate mental retardation” due to anticipated declines in population hemoglobin levels, with that increased probability potentially as high as 29% increased odds (Hurtado et al. 1999).

In addition to motor and cognitive impacts, losing access to wildlife would also cause an increased risk of infectious disease in children. By applying known associations between anemia and infectious disease to the modeled rates of anemia when the study population loses access to wildlife, we found that over 90% of infants would incur a tripled to quadrupled future risk of diarrhea (Levy et al. 2005b, Umamaheswari et al. 2010) and a doubled risk for respiratory disease (Levy et al. 2005b). Worldwide, more than 50% of the 10.5 million child deaths in 2001 were attributable to acute respiratory infections, measles, diarrhea, malaria and HIV/AIDS (Lopez et al. 2006). Diarrhea alone accounts for 15% of these deaths and an even greater percentage are attributable to respiratory infections (Lopez et al. 2006).

### *Projected health effects in women*

If we assume a similar proportion of women to develop anemia as children due to lost access to wildlife, then we could expect a 1.3 fold increase in anemia in women as well (See Results in Chapter 3). Under a scenario where access to wildlife was lost, 11% more pregnant women would experience anemia (Table 1) and thus be at a 1.1 times greater risk of delivering low birthweight infants (Levy et al. 2005a) with birthweights 242 grams less than those born to non-anemic mothers (Ronnenberg et al. 2004). These same women would be 1.2 times more likely to deliver prematurely (Levy et al. 2005a) and 4.4 times more likely of delivering a stillborn baby (Tomashek et al. 2006). Fetal nutrition and birthweight are incredibly important in predicting life-long health status (Barker 1995, Godfrey & Barker 2000, Fanaroff et al. 2007), demonstrating the importance of anemia beyond the critical window of pregnancy and infancy.

Maintaining access to wildlife would maintain current rates of anemia, a known risk factor of certain psychiatric illnesses. By continuing wildlife consumption, our study population is estimated to avoid a 6% increase in schizophrenia spectrum disorders (Insel et al. 2008). Amongst postpartum breastfeeding women in this population, 15% are currently anemic and an additional 8% would become anemic with lost access to wildlife. Continued access to bushmeat may allow these additional 8% to avoid a 25% decline in depression and stress scales (Beard et al. 2005). These results suggest removing access to bushmeat in this vulnerable population could incur significant physical and psychiatric health consequences.

### *Economic costs of iron-deficiency anemia*

Assuming the maximal workload (kcal/min) for an individual is 0.8 – 1.3 times their hemoglobin concentration (Viteri & Torun 1974), our study population would show an average decline in expended workload by 0.2-0.3 kcal/minute due to loss of wildlife access. Horton and Ross (2003) developed a theoretical and empirical model to demonstrate the economic costs from both reduced physical capacity in adulthood and cognitive losses due to IDA in childhood. 98% of the population in the Makira Protected Area is participating in heavy manual labor and will thus experience a 12% decreased physical capacity rather than a 5% reduction as expected from easier work (Horton & Ross 2003). The annual economic productivity loss per adult is currently \$5.03 per capita (30% of population anemic) but is predicted to increase to \$6.55 per capita (39% of population anemic) with lost access to wildlife in the future (Horton & Ross 2003). This represents a 0.3% increase in per capita losses as a proportion of national GDP. The projected annual economic productivity loss due to children's IDA from persisting cognitive deficits is currently \$2.16 per capita (61% of population anemic) but could become as high as \$2.80 per capita (79% of population anemic) if access to wildlife is lost (Horton & Ross 2003). In the Makira Protected Area, median annual income is \$70 (Chapter 2) and nationally, GDP is \$488 (UNdata 2008). These are economic productivity costs that underestimate the overall economic burden because the estimates do not reflect the economic burden to this region of the indirect effects of increased rates of anemia and its associated health consequences.

### **Discussion**

The results described here demonstrate the powerful and far-reaching effects of lost wildlife access on a variety of human health outcomes, ranging from cognitive and psychiatric to motor and physical effects. Using results from the literature review and models from Golden et al. (in review), it is possible to predict the possible future effects of changes in hemoglobin and anemia status stemming from reduced dietary animal-source foods (i.e from restricted access to wildlife). These potential future health effects suggest that the economic and health costs arising from either conservation enforcement or biodiversity loss from unsustainable exploitation need to be mitigated through public health or development assistance.

Although the links between changes in anemia status (or hemoglobin concentrations) and consequential health outcomes described in the literature review are convincing, the validity of applying these results to a particular site in rural Madagascar relies on a series of assumptions. We included in the literature review only studies with significant linkages between anemia and consequential health outcomes. In certain studies (i.e. Golden et al. in review) that included only hemoglobin without additional biomarkers such as serum ferritin, zinc protoporphyrin, or transferrin receptors to substantiate cause, we assumed that iron-deficiency was the source of anemia rather than other rarer etiologies such as folate or Vitamin B deficiencies. We also assumed that the model linking wildlife loss to shifts in hemoglobin that was designed for pre-adolescent children (Chapter 3) was applicable to reproductive-aged women. Finally, we assumed that studies from the literature review could be validly used to predict health effects at

our study site. Many of the estimates of potential health effects that may be experienced in Madagascar may be conservative because this population is more remote, underdeveloped and underserved with regard to support infrastructure than all of the populations in the literature review (Table 2). Thus, the ability for this population to adapt to or mitigate the impacts of nutritional deficiencies on further health consequences may be limited.

In an underdeveloped setting like rural Madagascar where support infrastructure is limited, alleviating iron-deficiency anemia is difficult yet of the utmost importance to prevent both its proximate effects on health and associated health sequelae. Reducing the incidence of anemia is unlikely given the rapidly rising global population, rising food prices and large-scale declines in biodiversity (Diaz et al. 2006, Myers & Patz 2009). Public health specialists find it difficult to improve iron-deficiency anemia on a population level without widespread initiatives for dietary diversity or fortification/biofortification (Neumann et al. 2003, Lee et al. 2009, Bouis & Welch 2010, Bouis et al. 2011). Many countries choose not to invest in fortification schemes because they believe that economic development in itself will improve nutritional status; however, in a meta-analysis of 32 countries, Alderman and Linnemayr (2009) found that there is only a modest decrease in anemia as income increases. Economic development and income increases are more effective in correcting underweight, improving macronutritional status four times as fast as micronutritional status (Alderman & Linnemayr 2009).

Although not perfectly understood, there appear to be critical periods where iron-deficiency has major effects and where correction of anemia can no longer reverse damage. Most studies indicate that gestation and early lactation are these critical periods that may produce long-lasting effects into adulthood (Beard 2008). The importance of the gestational period to offspring health is not limited to birth outcomes. The fetal environment plays a significant role in programming the trajectories for future health from infancy to adulthood (Barker 1995, Godfrey & Barker 2000), demonstrating the instrumental importance of maternal health for population health. The mechanisms that underlie this relationship are fetal programming due to nutritional stimuli during gestation (Barker 1995, De Boo & Harding 2006). After this trajectory is started in gestation, some health domains may still be correctable through proper nutrition during infancy. Even with complete adherence to proper breastfeeding, complementary foods must contribute nearly 100% of dietary iron for infants since breastmilk contains very small amounts of bioavailable iron (Brown et al. 1998). Thus, careful attention to maternal and child nutrition is needed for proper population health outcomes throughout individuals' lifetimes.

Iron supplementation has been shown to be efficacious (Yip 1996), and both fortification (Mannar & Gallego 2002) and biofortification (Lucca et al. 2002) to be effective under certain circumstances. Although the prior interventions are feasible strategies to alleviate iron-deficiency anemia, there are also several drawbacks. First, supplementation is very expensive and only designed for short-term interventions (Levin 1986). Also, fortification, although cost-effective in the long-term, is often prohibitively expensive for developing countries that may be particularly at risk for a heavy burden of anemia (Levin 1986). Secondly, because iron supplementation improves iron status without improving general health and other micronutrient deficiencies, blood may become richer in red blood cells without correlative improvements in other micronutrient health and immunity. In certain cases, this has been shown to place populations at increased risk for malarial morbidity (Oppenheimer et al. 1986). Third, iron supplementation may foster imbalanced nutrition because iron is a +2 cation and may compete for ionic unions

with other +2 cations such as copper, calcium and zinc due to limited ionic bioavailability (Meadows et al. 1983, Whittaker 1998, Kordas & Stoltzfus 2004). In terms of logistics, supplementation is efficacious in improving iron status when there is strong adherence under a controlled clinical trial, yet is typically ineffective at improving iron status when administered at a population level by public health care infrastructures (Yip 1996). Further, supplementation has been shown to be ineffective in improving hemoglobin concentrations (Cogswell et al. 2003), especially when multiple micronutrient deficiencies were present (Allen et al. 2000). Because of the importance of holistically improving nutritional status, dietary diversification is the best solution to iron-deficiency anemia on a population level.

Dietary diversification and access to natural resources are strongly related in developing countries where over 300 million people rely on forest products for nutritional support in the developing world (Pimentel et al. 1997). Anticipated wildlife depletion and biodiversity declines will threaten dietary diversity in those who are economically and geographically marginalized (Allen-Wardell et al. 1998, Grivetti & Ogle 2000, Brashares et al. 2004, Golden 2009, Brashares et al. in press). Domesticated meats are often prohibitively expensive (Steinfeld 2003) and thus a loss in wild meats may often not be substituted by a meat alternative. Therefore, mitigating trends in biodiversity loss and habitat destruction that threaten wildlife populations, preserving access to wildlife and creating subsidized options for animal-source food alternatives should be prioritized by the public health community to prevent the onset of a nutritional crisis.

Table 1: The current and predicted future prevalence of anemia categorized by age and sex

	Children (<5)	Children (5-12)	Pregnant and Breastfeeding women	Non-pregnant women	Men
Hemoglobin threshold (WHO 2001)	<11.0 g/dL	<11.5 g/dL	<11.0 g/dL	<12.0 g/dL	<13.0 g/dL
Anemia prevalence (sample size)	61% (n=215)	46% (n=293)	24% (62)	26% (145)	35% (n=161)
Projected prevalence (1.3 fold increase)	79%	60%	31%	34%	46%

Table 2: Studies included in the literature review with a rating of epidemiological rigor and the health domains associated with iron-deficiency anemia

Authors	Year	Study population	Health domain affected	Rigor rating
Chang et al.	2011	Four-year old Chinese children (n=164)	Behavior	2
Armony-Sivan et al.	2010	Urban American mother-infant dyads (n=68)	Behavior	2
Corapci et al.	2010	Cohort of Costa-Rican children followed for 19 years (n=185)	Behavior	1
Lozoff et al.	2008	9-12 month old African-American infants (n=77)	Behavior	3
Kordas et al.	2008	6–18 month old Zanzibari and Nepali infants (n=66; 77)	Behavior	2
Corapci et al.	2006	5 year-old Costa Rican children (n=142)	Behavior, Physical Capacity and Cognition	1
Hudaoglu et al.	2006	Turkish mothers (n=60)	Behavior	2
Wachs et al.	2005	Peruvian newborns (n=148)	Behavior	3
Soemantri et al.	1985	10-12 year old Indonesia schoolchildren (n=588)	Behavior and Cognition	3
Palti et al.	1985	9-month old Israeli infants (n=76)	Behavior	3
Walter et al.	1983	15 month old Chilean infants (n=37)	Behavior	2
Lukowski et al.	2010	Cohort of 19-year old Costa Rican individuals (n=114)	Cognition	3
Carter et al.	2010	Urban American infants (n=77)	Cognition	1
Fararouei et al.	2010	Finnish mother-offspring dyads followed from pregnancy through to age 31 (n=11,656)	Cognition	2
Gupta et al.	2010	Six-month to five-year old Indian children (n=35)	Cognition	3

Khedr et al.	2008	Adult Egyptian hospital patients (n=28)	Cognition and Neurologic Function	3
Petranovic et al.	2008	32-60 year old Croatian hospital patients (n=61)	Cognition	3
Corapci et al.	2006	5 year-old Costa Rican children (n=142)	Cognition, Physical Capacity and Behavior	1
Sen and Kanani	2006	9-14 year old Indian schoolgirls (n=230)	Cognition, Physical Capacity and Physiology	3
Akman et al.	2004	6-30 month old Turkish children (n=108)	Cognition	1
Walter	2003	Chilean infants (n=1,700)	Cognition and Neurologic Function	2
Sunghong et al.	2002	First to sixth grade Thai schoolchildren (n=427)	Cognition	2
Halterman et al.	2001	6-16 year old American children (n=5,398)	Cognition	3
Stoltzfus et al.	2001	6-59 month old Zanzibari children (n=614)	Cognition and Motor Function	1
Lozoff et al.	2000	11-14 year old Costa Rican children (n=166)	Cognition and Motor Function	1
Hurtado et al.	1999	10 year old American public school students enrolled in WIC (n=3,771)	Cognition	2
Wasserman et al.	1994	Mother-children dyads from Kosovo (n=388 for 3 year old children; n=332 for 4 year old children)	Cognition	2
Idjradinata and Pollitt	1993	12-18 month old Indonesian infants (n=141)	Cognition	2
Lozoff et al.	1991	5 year old Costa Rican children (n=191)	Cognition and Motor Function	1
Walter et al.	1989	12-15 month old Chilean infants (n=196)	Cognition and Motor Function	2
Lozoff et al.	1987	12- to 23-month-old Costa Rican infants (n=191)	Cognition and Motor Function	1
Pollitt et al.	1985	Egyptian children (mean age 9.5, n=68)	Cognition	3
Soemantri et al.	1985	10-12 year old Indonesia schoolchildren (n=588)	Cognition and Behavior	3
Palti et al.	1983	Israeli children (n=873 (2-year olds), 388 (3 year olds) and 239 (5 year olds)).	Cognition	3
Cantwell	1974	Full-term American neonates followed to 7 years (n=61)	Cognition and Motor Function	3
Mahajan et al.	2008	Indian mother-infant dyads (n=300)	Growth and Physiology	2

Ronnenberg et al.	2004	Chinese women at preconception (n=405)	Growth and Morbidity	2
Angeles et al.	1993	2-5 year old Indonesian children (n=76)	Growth and Morbidity	3
Banhidy et al.	2011	Hungarian pregnant women and newborn dyads (n=60,994)	Morbidity	1
Umamaheswari et al.	2010	Indian children afflicted with diarrhea (n=120)	Morbidity	2
Hartfield et al.	2009	Cohort of 6 to 36 month-old Canadian children (n=751)	Morbidity	2
Moeinvaziri et al.	2009	15- to 45-year old Iranian women (n=60)	Morbidity	2
Sadeghi-Bojd et al.	2009	6- to 72-month old Iranian children (n=40)	Morbidity and Physiology	3
Zhang et al.	2009	Singleton live births delivered at 20–44 weeks to Chinese women (n=160,700)	Morbidity	2
Insel et al.	2008	American infants followed for approximately 30 years (n= 6872)	Morbidity	1
Maguire et al.	2007	12-38 month old Canadian infants	Morbidity	2
Lee et al.	2006	South Korean pregnant women followed through to delivery (n=248)	Morbidity	2
Shariatpanaahi et al.	2006	Female Iranian medical students (n=192)	Morbidity	3
Tomashek et al.	2006	Pregnant American women (n=5,574)	Morbidity	2
Beard et al.	2005	18-30 year old South African mothers of newborns (n=81)	Morbidity	2
Levy et al.	2005a	Israeli pregnant women (n=153,396)	Morbidity	2
Levy et al.	2005b	Israeli Bedouin newborns to 18 months (n=293)	Morbidity	2
O'Brien et al.	2005	HIV-positive Tanzanian pregnant women (n=1,078)	Morbidity and Physiology	2
Ronnenberg et al.	2004	Chinese women at preconception (n=405)	Morbidity and Growth	2
Corwin et al.	2003	American women within 24 hours of labor (n=37)	Morbidity	2
Özçay et al.	2003	6–72 month old Turkish children (n=40)	Morbidity and Physiology	2
Angeles et al.	1993	2-5 year old Indonesian children (n=76)	Morbidity and Growth	3
Angulo-Barroso et al.	2011	9-month-old infants from urban areas of China, Ghana, and USA (n=209)	Motor Function	2

Shafir et al.	2009	10-month old African-American infants (n=9)	Motor Function	3
Shafir et al.	2008	Full-term urban African-American 9- to 10-month-old infants (n=106).	Motor Function	3
Olney et al.	2007	5–19 month old Zanzibari children (n=771)	Motor Function	2
Shafir et al.	2006	12-23 month old Costa Rican children (n=185)	Motor Function	1
Armony-Sivan et al.	2004	Israeli premature infants (n=53)	Motor Function	2
Lind et al.	2004	6-month old Indonesian children (n=666)	Motor Function	1
Angulo-Kinzer et al.	2002	Chilean infants (n=26)	Motor Function	3
Sherriff et al.	2001	8-18 month old British children (n=1,141)	Motor Function	2
Stoltzfus et al.	2001	6-59 month old Zanzibari children (n=614)	Motor Function and Cognition	1
Harahap et al.	2000	Indonesian infants (n=36)	Motor Function	2
Lozoff et al.	2000	11-14 year old Costa Rican children (n=166)	Motor Function and Cognition	1
Lozoff et al.	1991	5 year old Costa Rican children (n=191)	Motor Function and Cognition	1
Dommergues et al.	1989	10 month to 4 year old French children (n=147)	Motor Function	3
Walter et al.	1989	12-15 month old Chilean infants (n=196)	Motor Function and Cognition	2
Lozoff et al.	1987	12- to 23-month-old Costa Rican infants (n=191)	Motor Function and Cognition	1
Aukett et al.	1986	17-19 month old British children (n=470)	Motor Function	1
Cantwell	1974	Full-term American neonates followed to 7 years (n=61)	Motor Function and Cognition	3
Kececi and Degermenci	2008	Fifty-one 20 year-old Turkish hospital patients (n=51)	Neurologic Function	3
Khedr et al.	2008	Adult Egyptian hospital patients (n=28)	Neurologic Function and Cognition	3
Burden et al.	2007	9-12 month old African-American infants (n=34)	Neurologic Function	2
Peirano et al.	2007	6 month-old Chilean infants (n=26)	Neurologic Function and Physiology	2
Algarin et al.	2003	Chilean children under 5 years old (n=84)	Neurologic Function	1
Walter	2003	Chilean infants (n=1,700)	Neurologic Function and Cognition	2



Sen and Kanani	2009	9-13 year old Indian schoolgirls (n=163)	Physical Capacity	2
Li et al.	1994	Chinese female cotton mill workers aged 19-44 (n=447)	Physical Capacity	1
Edgerton et al.	1979	Sri Lankan female tea plantation workers aged 20-60 (n=217)	Physical Capacity	3
Basta et al.	1979	16 to 40 year old male plantation workers (n=302)	Physical Capacity	1
Corapci et al.	2006	5 year-old Costa Rican children (n=142)	Physical Capacity, Behavior and Cognition	1
Sen and Kanani	2006	9-14 year old Indian schoolgirls (n=230)	Physical Capacity, Physiology, and Cognition	3
Lozoff et al.	2010	9- to 10-mo-old urban American infants (n=61)	Physiology	2
Mahajan et al.	2008	Indian mother-infant dyads (n=300)	Physiology and Growth	2
Sadeghi-Bojd et al.	2009	6- to 72-month old Iranian children (n=40)	Physiology and Morbidity	3
Peirano et al.	2007	6 month-old Chilean infants (n=26)	Physiology and Neurologic Function	2
Felt et al.	2006	11-14 year old Costa Rican children followed since infancy (n=167)	Physiology	2
Sen and Kanani	2006	9-14 year old Indian schoolgirls (n=230)	Physiology, Physical Capacity, and Cognition	3
O'Brien et al.	2005	HIV-positive Tanzanian pregnant women (n=1,078)	Physiology and Morbidity	2
Özçay et al.	2003	6–72 month old Turkish children (n=40)	Physiology and Morbidity	2
Roncagliolo et al.	1998	4-18 month old Chilean infants (n=55)	Physiology	2

Figure 1: Study selection flowchart for the literature review

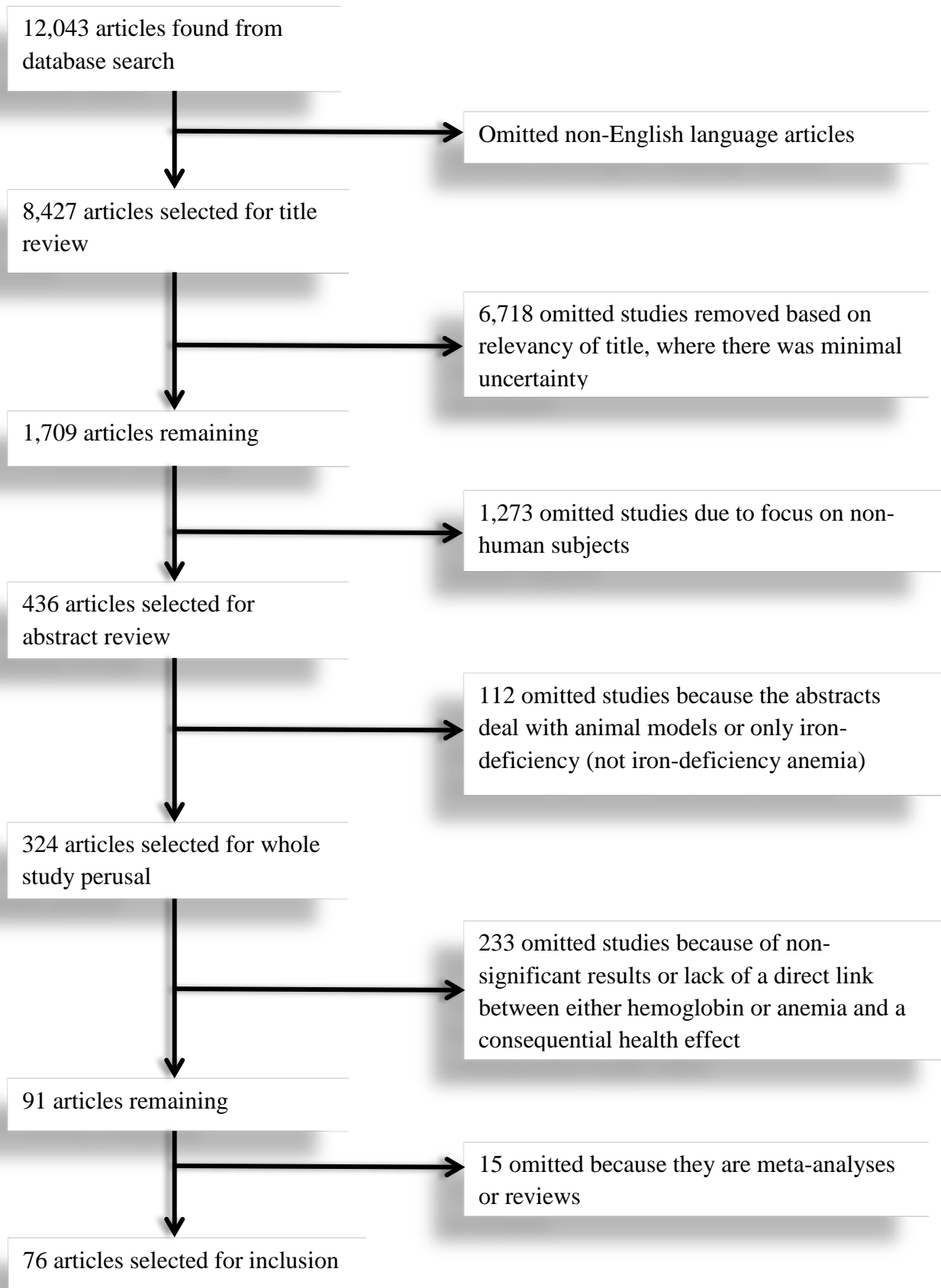


Figure 2: Health domains affected by iron-deficiency anemia (percent of final set of articles developed in the literature review)

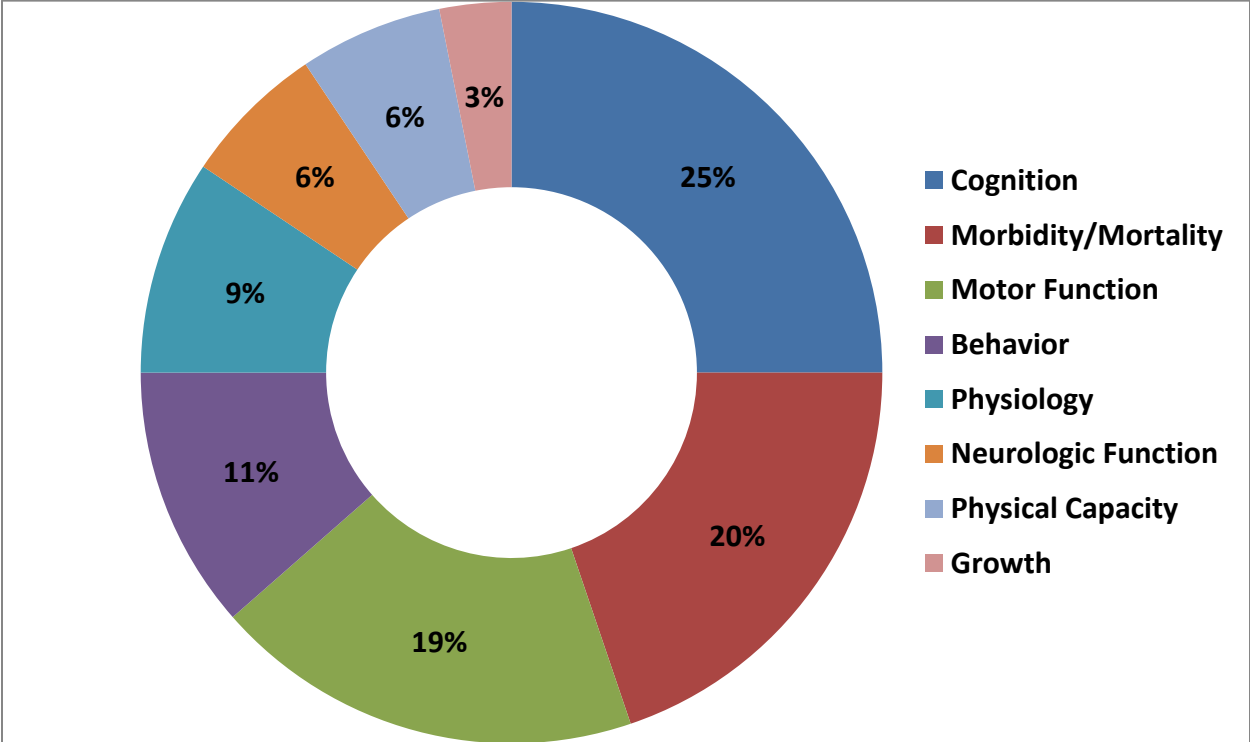
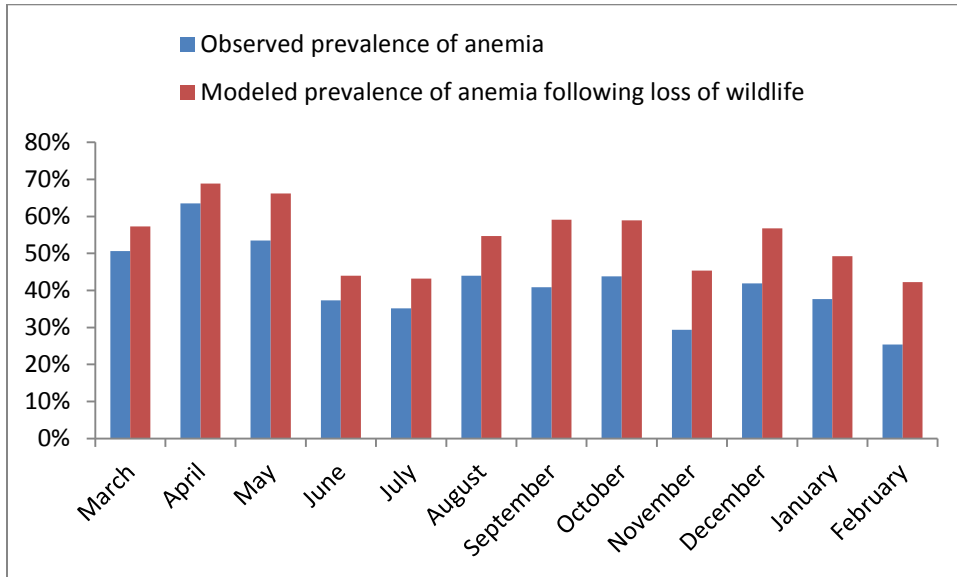


Figure 3: Seasonal variation in the prevalence of anemia in a cohort of pre-adolescent Malagasy children



From March 2008 until February 2009, hemoglobin from 77 pre-adolescent children in a village in the Makira Protected Area was sampled to understand the seasonal dynamics of anemia. We show the observed prevalence of anemia (blue) and the modeled prevalence of anemia (red) to demonstrate the predicted increases in anemia following the loss of access to wildlife for consumption.

#### Appendix 4.1: Search criteria for literature review

Topic=(anemia OR anaemia AND iron AND deficiency)

Refined by: [excluding] General Categories=( SOCIAL SCIENCES OR ARTS & HUMANITIES ) AND [excluding] Subject Areas=( PUBLIC, ENVIRONMENTAL & OCCUPATIONAL HEALTH OR PHARMACOLOGY & PHARMACY OR ENVIRONMENTAL SCIENCES & ECOLOGY OR NUCLEAR SCIENCE & TECHNOLOGY OR GENETICS & HEREDITY OR HEALTH CARE SCIENCES & SERVICES OR COMPUTER SCIENCE OR MATHEMATICAL & COMPUTATIONAL BIOLOGY OR CHEMISTRY OR SUBSTANCE ABUSE OR BIOPHYSICS OR MEDICAL INFORMATICS OR ENTOMOLOGY OR SURGERY OR NURSING OR INFORMATION SCIENCE & LIBRARY SCIENCE OR INSTRUMENTS & INSTRUMENTATION OR GERIATRICS & GERONTOLOGY OR LIFE SCIENCES & BIOMEDICINE - OTHER TOPICS OR ACOUSTICS OR REHABILITATION OR MICROSCOPY OR INTEGRATIVE & COMPLEMENTARY MEDICINE OR AGRICULTURE OR BIODIVERSITY & CONSERVATION OR VETERINARY SCIENCES OR BIOTECHNOLOGY & APPLIED MICROBIOLOGY OR MATERIALS SCIENCE OR FORESTRY OR ANESTHESIOLOGY OR LEGAL MEDICINE OR TRANSPLANTATION OR IMAGING SCIENCE & PHOTOGRAPHIC TECHNOLOGY OR TOXICOLOGY OR METEOROLOGY & ATMOSPHERIC SCIENCES OR PLANT SCIENCES OR MEDICAL ETHICS OR MARINE & FRESHWATER BIOLOGY OR SPECTROSCOPY OR THERMODYNAMICS OR ZOOLOGY OR OPTICS OR MEDICAL LABORATORY TECHNOLOGY OR PHYSICS OR AUTOMATION & CONTROL SYSTEMS OR ALLERGY OR WATER RESOURCES OR ANTHROPOLOGY OR PALEONTOLOGY OR ENGINEERING OR ENERGY & FUELS OR SCIENCE & TECHNOLOGY - OTHER TOPICS OR FISHERIES OR POLYMER SCIENCE OR EVOLUTIONARY BIOLOGY OR ELECTROCHEMISTRY OR MATHEMATICS OR TELECOMMUNICATIONS OR RADIOLOGY, NUCLEAR MEDICINE & MEDICAL IMAGING OR GEOLOGY ) AND Document Type=( ARTICLE )

Timespan=>1970.

Appendix 4.2: Quality assessment detailed results:

Article Authors	Pub. Yr.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Angulo-Barroso et al.	2011	2	5	2	7	N			3	1	1	1	3	3	2	1	1	1	N/A	N/A	N/A	2
Banhidy et al.	2011	1	1	1	4	N			2	3	1	1	3	2	2	1	1	1	N/A	N/A	N/A	1
Chang et al.	2011	2	1	2	3	N			2	2	1	1	2	2	1	1	1	1	1	3	3	2
Lukowski et al.	2010	1	5	2	3	N			2	1	1	1	3	3	3	1	1	1	3	4	3	3
Armony-Sivan et al.	2010	2	5	2	7	N			3	2	1	1	3	3	2	1	1	1	1	1	1	2
Carter et al.	2010	1	1	1	3	N			2	2	1	1	2	2	1	1	1	1	1	2	2	1
Corapci et al.	2010	1	1	1	3	N			2	2	1	1	2	2	1	1	1	1	1	2	2	1
Fararouei et al.	2010	1	1	1	3	N			2	2	1	1	3	2	2	1	1	1	1	2	2	2
Gupta et al.	2010	3	5	3	3	N			2	2	1	1	3	2	2	1	1	1	3	4	3	3
Lozoff et al.	2010	2	1	2	3	N			2	2	1	1	3	2	2	1	1	1	1	2	2	2
Umamaheswari et al.	2010	2	5	2	4	N			2	1	1	1	3	3	2	1	1	1	N/A	N/A	N/A	2
Hartfield et al.	2009	3	1	3	4	N			2	1	1	1	2	2	1	1	1	1	N/A	N/A	N/A	2
Moeinvaziri et al.	2009	3	5	3	4	N			2	1	1	1	2	1	2	1	1	1	N/A	N/A	N/A	2
Sadeghi-Bojd et al.	2009	3	5	3	4	N			2	3	4	3	3	3	2	1	1	1	N/A	N/A	N/A	3
Sen and Kanani	2009	2	1	2	1	Y	Y	Y	1	3	3	3	3	1	2	1	1	1	1	2	2	2
Shafir et al.	2009	3	5	3	7	N			3	2	2	2	2	2	1	1	1	1	N/A	N/A	N/A	3
Zhang et al.	2009	1	1	1	3	N			2	1	1	1	2	2	1	1	1	1	3	4	3	2
Lozoff et al.	2008	3	5	3	4	N			2	2	1	1	3	2	2	1	1	1	1	3	3	3
Shafir et al.	2008	3	5	3	4	N			2	2	1	1	3	2	2	1	1	1	1	3	3	3
Insel et al.	2008	1	1	1	4	Y	Y	Y	2	2	1	1	2	2	1	1	1	1	N/A	N/A	N/A	1
Kececi and Degermenci	2008	3	1	3	5	N			3	2	1	1	1	1	3	1	1	1	1	1	1	3
Khedr et al.	2008	3	1	3	3	N			2	1	1	1	3	3	3	1	1	1	3	4	3	3
Kordas et al.	2008	1	1	1	7	N			3	2	1	1	2	2	1	1	1	1	1	1	1	2
Mahajan et al.	2008	2	1	2	3	N			2	1	1	1	3	3	2	1	1	1	3	4	3	2
Petranovic et al.	2008	3	5	3	7	N			3	1	2	2	3	2	2	2	2	3	N/A	N/A	N/A	3
Burden et al.	2007	2	1	2	4	N			2	2	1	1	3	2	2	1	1	1	1	3	3	2
Peirano et al.	2007	2	5	2	4	N			2	1	1	1	3	2	2	1	1	1	3	4	3	2
Maguire et al.	2007	3	1	3	4	N			2	1	1	1	2	2	1	1	1	1	N/A	N/A	N/A	2
Olney et al.	2007	1	1	1	7	Y	Y	Y	3	2	1	1	2	2	1	1	1	1	N/A	N/A	N/A	2
Felt et al.	2006	1	1	1	3	N			2	2	1	1	3	2	2	1	1	1	1	1	1	2
Shafir et al.	2006	1	1	1	3	N			2	1	1	1	3	3	2	1	1	1	1	1	1	1

Corapci et al.	2006	1	5	2	3	N				2	2	1	1	2	2	1	1	1	1	1	1	1	
Hudaoglu et al.	2006	3	1	3	4	N				2	2	1	1	3	1	2	1	1	1	N/A	N/A	N/A	2
Lee et al.	2006	2	1	2	3	N				2	2	1	1	3	1	2	1	1	1	N/A	N/A	N/A	2
Sen and Kanani	2006	2	1	2	7	N				3	3	3	3	3	1	2	1	1	1	N/A	N/A	N/A	3
Shariatpanahi et al.	2006	3	1	3	4	N				2	3	4	3	3	3	2	1	1	1	1	1	1	3
Tomashek et al.	2006	1	1	1	7	N				3	2	1	1	2	2	1	1	1	1	N/A	N/A	N/A	2
Wachs et al.	2005	2	1	1	7	N				3	2	1	1	3	2	2	1	1	1	1	1	3	3
Beard et al.	2005	1	3	3	1	Y	Y	Y	1	1	1	1	2	2	1	1	1	1	1	1	1	1	2
Levy et al.	2005a	1	1	1	7	N				3	1	1	1	2	2	1	1	1	1	N/A	N/A	N/A	2
Levy et al.	2005b	2	1	2	3	N				2	1	1	1	2	2	1	1	1	1	3	4	3	2
O'Brien et al.	2005	1	1	1	3	Y	Y	Y	2	1	1	1	2	2	1	1	1	1	1	3	4	3	2
Armony-Sivan et al.	2004	2	1	2	7	N				3	2	1	1	3	2	2	1	1	1	N/A	N/A	N/A	2
Lind et al.	2004	1	1	1	1	Y	Y	Y	1	1	1	1	2	2	1	1	1	1	1	1	1	1	1
Akman et al.	2004	2	5	2	2	Y	Y	Y	1	2	1	1	2	1	2	1	1	1	1	1	1	1	1
Ronnenberg et al.	2004	2	5	2	3	N				2	1	1	1	3	3	2	2	2	3	1	2	2	2
Algarin et al.	2003	1	2	2	1	Y	N	N	1	1	1	1	3	2	2	1	1	1	1	1	1	1	1
Walter	2003	1	5	2	1	Y	Y	Y	1	1	1	1	2	2	1	1	1	1	1	3	4	3	2
Corwin et al.	2003	2	5	2	3	N				2	2	1	1	2	1	2	1	1	1	1	1	1	2
Özçay et al.	2003	3	5	3	4	N				2	2	1	1	3	3	2	1	1	1	N/A	N/A	N/A	2
Angulo-Kinzer et al.	2002	3	5	3	7	N				3	2	1	1	3	2	2	1	1	1	3	3	3	3
Sungthong et al.	2002	1	5	1	7	N				3	1	1	1	3	3	2	1	1	1	N/A	N/A	N/A	2
Sherriff et al.	2001	1	1	1	7	Y	N	N	3	3	1	1	3	2	2	1	1	1	1	1	1	1	2
Halterman et al.	2001	1	1	1	7	N				3	2	1	1	3	3	3	1	1	1	N/A	N/A	N/A	3
Stoltzfus et al.	2001	1	1	1	1	Y	Y	Y	1	2	1	1	2	2	1	1	1	1	1	1	1	1	1
Harahap et al.	2000	1	1	1	3	Y	Y	Y	2	3	4	3	2	2	1	1	1	1	1	1	1	1	2
Lozoff et al.	2000	1	1	1	3	Y	Y	Y	2	2	1	1	2	2	1	1	1	1	1	1	1	1	1
Hurtado et al.	1999	2	2	2	7	N				3	1	1	1	3	2	2	1	1	1	1	1	1	2
Roncagliolo et al.	1998	1	1	1	5	N				2	2	1	1	3	2	2	1	1	1	1	2	2	2
Li et al.	1994	2	1	2	1	Y	N	N	1	2	1	1	2	2	1	1	1	1	1	1	1	1	1
Wasserman et al.	1994	1	2	2	3	N				2	1	1	1	3	3	3	1	1	1	1	2	2	2
Angeles et al.	1993	3	1	3	3	Y	N	N	3	2	1	1	2	2	1	1	1	1	1	1	1	1	3
Idjradinata and Pollitt	1993	2	1	2	1	Y	Y	Y	1	2	1	1	2	2	1	1	1	1	1	3	4	3	2
Lozoff et al.	1991	1	1	1	3	Y	Y	Y	2	2	1	1	2	2	1	1	1	1	1	1	1	1	1

Dommergues et al.	1989	3	5	3	3	N			2	3	4	3	3	3	3	1	1	1	3	4	3	3
Walter et al.	1989	2	5	2	3	Y	Y	Y	2	2	1	1	2	2	1	1	1	1	1	2	2	2
Lozoff et al.	1987	1	1	1	3	Y	Y	Y	2	2	1	1	2	2	1	1	1	1	1	1	1	1
Aukett et al.	1986	1	5	2	1	Y	Y	Y	1	2	1	1	2	2	1	1	1	1	1	1	1	1
Palti et al.	1985	3	5	3	3	N			2	2	1	1	2	2	1	1	1	1	3	4	3	3
Pollitt et al.	1985	1	1	1	1	Y	Y	Y	1	3	4	3	3	3	3	3	3	3	3	1	1	1
Soemantri et al.	1985	1	3	3	1	Y	Y	Y	1	2	1	1	3	3	3	1	1	1	3	4	3	3
Palti et al.	1983	3	5	3	3	N			2	2	1	1	2	2	1	1	1	1	3	4	3	3
Walter et al.	1983	1	3	3	3	Y	Y	Y	2	2	1	1	2	2	1	1	1	1	1	2	2	2
Edgerton et al.	1979	2	1	2	1	Y	N	Y	1	2	1	1	3	1	3	1	1	1	2	4	3	3
Basta et al.	1979	2	2	2	1	Y	N	N	1	3	2	2	2	2	1	1	1	1	1	1	1	1
Cantwell	1974	4	5	3	3	N			2	3	3	3	3	3	3	3	3	3	1	1	1	3

For more information on the descriptions of the following categories, please refer to McMaster 2011 reference. <sup>1</sup> Are individuals representative of target population? <sup>2</sup> Percentage agreed to participate <sup>3</sup> Selection bias rating <sup>4</sup> Type of study design <sup>5</sup> Was study randomized? <sup>6</sup> Randomization described? <sup>7</sup> Was the method appropriate? <sup>8</sup> Study design rating <sup>9</sup> Were there important differences between groups prior to the intervention? <sup>10</sup> Percentage of confounding variables addressed in either design or analysis <sup>11</sup> Confounding rating <sup>12</sup> Were the assessors aware of the intervention or exposure status of participants? <sup>13</sup> Were the study participants aware of the research question? <sup>14</sup> Blinding rating <sup>15</sup> Valid data collection tools? <sup>16</sup> Reliable data collection tools? <sup>17</sup> Data collection rating <sup>18</sup> Withdrawals reported as numbers and/or groups? <sup>19</sup> Withdrawal percentage <sup>20</sup> Withdrawal rating <sup>21</sup> Global score rating of epidemiological rigor.



## CHAPTER 5

### Conclusions and Future Directions

In this dissertation, I attempted to answer several questions about the interrelationships between ecosystem service provisioning and human health outcomes in northeastern Madagascar. Specifically, I investigated both the economic and health importance of wildlife harvest and consumption to rural people in the Makira Protected Area. I found that although wildlife harvest has an absolute monetary value typically less than other provisioning services and even less than wildlife harvest from other geographic regions (Chapter 2), the relative economic value as a proportion of annual household income and the health value to household members are of major importance, contributing 12-37% of median annual income and preventing a 30% increase in the incidence of anemia (Chapters 2 + 3). Based on these findings, I suggest that strict monetary valuations of ecosystem services may vastly underestimate the importance of services to human health and livelihoods.

In the future, there are several ways in which the research community can improve assessments of human reliance on ecosystem services and better inform decisions regarding land use change. In my research, this includes better wildlife monitoring, complete socio-economic assessments and investigating the mechanisms by which wildlife may influence human health. Further research alone will not solve the disconnect between the status quo of conservation planning and the needs of local people who rely on protected natural resources. This disconnect can only be bridged by a willingness on the part of the conservation community to allow all aspects of the costs of natural resource restrictions to be incorporated into conservation planning.

Long-term wildlife monitoring will be necessary to understand the population dynamics and trajectories of each species that is affected by hunting. This monitoring will allow conservation decision-makers to determine which species should be avoided for harvest and which ones could be harvested with and without restrictions. There are many examples of monitoring research undertaken to inform wildlife quotas and sustainability (Bodmer et al. 1994, Lewis & Alpert 1997, Carrillo et al. 2000). But, the effectiveness of implementing policy in guiding actual harvest rates is still minimal in many developing country settings (Ribot & Peluso 2003, Keane et al. 2008, Golden 2009). Without knowledge of wildlife population dynamics, the conservationist's precautionary principle would likely prevent local people from receiving legalized access to wildlife for food (Myers 1993, Kriebel et al. 2001, Cooney 2004, Vardas & Xepapadeas 2010), leading to both socio-economic and health havoc (Garcia 1994).

Traditionally, ecosystem service assessments use an absolute monetary estimate as the currency for expressing a given value (Godoy et al. 1993). These per hectare estimations are valuable for making decisions about land-use alternatives because they can be systematically compared across sites (Kremen et al. 2000, Chan et al. 2006). Complete socioeconomic assessments combined with analyses of ecosystem service flows to households will add nuance to the local importance of ecosystem services (Chapter 2). By having this secondary data, researchers can estimate the proportional value of service flows to household wealth. Relative valuations will offer insights into both household and individual-level incentives and disincentives for compliance with conservation policies (Chapter 2). Furthermore, development

and public health targeting can be informed by these fine-scale data to compensate those negatively affected by conservation policy (Chapter 2).

Investigating the mechanisms by which wildlife may affect human health will produce conceptions of value that may be overlooked in traditional monetary valuation schemes. Ecosystem services provision myriad varieties of food that offer nutritional value to resource-dependent people across the globe (Worm et al. 2006, Swinton et al. 2007, Luck et al. 2009). To understand the nutritional importance of these provisioned foods, one must consider both macronutrient and micronutrient composition. Macronutrients are proteins, carbohydrates and fats which provide energy, promote growth and development and regulate bodily function (Waterlow & Payne 1975). Although macronutrients contribute to all of these activities, their primary function is to provide energy (Waterlow & Payne 1975). On the other hand, micronutrients (vitamins, minerals and trace elements) do not provide energy but almost all are essential in that they cannot be created by the body itself and are indispensable to life processes (Bhaskaram 2002).

As their names imply, macronutrients are required by the body in large quantities and micronutrients are required in small quantities. However, it is more difficult to obtain certain essential micronutrients in small quantities than large amounts of macronutrients (McLaren 1974, 2000, Bhaskaram 2002). Thus, micronutrients, especially iron, are found to be the limiting factor in many diets (Gibson et al. 1998, Dewey 2007). The reason underlying this is that easily obtained staples (i.e. rice, grains and tubers) are rich in proteins and carbohydrates while micronutrient rich foods tend to be luxury foods that are less commonly available to poorer subgroups in developed countries and to all citizens of developing countries (Black et al. 2008). A prime example of a luxury food rich in micronutrients is animal source foods (Murphy & Allen 2003, Neumann et al. 2003). Although animal source foods are often perceived as major protein sources (and even called proteins), vegetarian sources of protein are often highly bioavailable (Millward 1999, Hunt 2003). Animal source foods possess high values of iron, zinc, Vitamin A, B complex vitamins, omega-3 fatty acids, and fats that are often difficult to obtain from other non-fortified foods in developing countries (Hunt 2003, Murphy & Allen 2003, Neumann et al. 2003). This has significant implications for people living in resource-dependent nations that rely on wildlife as the primary form of animal-source foods.

Dosing is the most important consideration to recognize in developing programmatic approaches to reduce the prevalence and incidence of multiple micronutrient deficiencies. For a micronutrient like Vitamin A, a technical fix has been shown to be highly effective and efficient (Herrera et al. 1992, Glasziou & Mackerras 1993). Supplementation is only required twice per year with huge doses of vitamin A which prevents deficiencies and cannot cause toxicity (Ross 2002). Also, vitamin A supplementation does not affect the bioavailability of other micronutrients (Ross 2002). On the other hand, a micronutrient like iron is ideally consumed in very small doses on a daily or weekly basis (Schultink et al. 1995). A very large dose would be toxic to an individual (Allen 2002, Goldhaber 2003) and thus presents major difficulty in developing a technical fix like supplementation that would be efficient on a large population scale with limited financial resources. Furthermore, iron can prevent the absorption (i.e. bioavailability) of other similar micronutrient cations such as zinc, copper and magnesium (Solomons & Jacob 1981, Solomons 1986, Whittaker 1998, Sandstrom 2001).

These facts about dosing and bioavailability should also influence the way in which we conceive of wildlife harvest regulations and sustainability. If conservation practitioners forbid hunting or only allow harvest during particular seasons, then these policies would likely lead to permanent or seasonal nutritional deficiencies. The tension between nutritional food security and biodiversity conservation presents to stakeholders a clear conflict that is uneasily resolved. As mentioned above (and in more depth in chapter 4), issues with dosing and bioavailability illuminate that dietary diversity is the best possible solution to many micronutrient deficiencies in resource-dependent nations. This is because it provides combinations of micronutrients that are less likely to preclude the absorption of others and can be received at regular doses throughout the year during mealtimes rather than in massive quantities from supplementation. There are two possible options to ease the concerns of both stakeholder communities while trying to address both malnutrition and biodiversity loss. First, it is possible to create a regime of wildlife harvest that is sustainably managed where local people receive a year-long supply of wild meat. Because of dosing issues, it is better to receive small portions throughout the year rather than having a system of fixed quotas during a given season or year that could be harvested and used rapidly. The other option is to go the way of wild meats in Britain. Historically, the British heavily relied on hares, venison and squab for meat and now the society has progressed to the extent that these wild meats are not needed for nutritional food security. A drawback to this option is that the path to non-reliance on wild meats must coincide with a path of environmental conservation and this is not an easy task. In fact, I am unable to provide any concrete examples of areas where this has occurred in the past.

Food security and biodiversity conservation are some of the globe's most pressing issues and are difficult to solve in isolation. These problems are even more difficult to rectify once the research and development communities realize that these topics are interconnected and project goals may be oppositional (Diaz et al. 2006). This dissertation is merely a preliminary attempt to illustrate the web of relationships between human health and the natural environment. Provided with this knowledge, stakeholders interested in both health and the environment need to transcend their traditional boundaries and broaden their constituencies to jointly deliver a concerted effort from the development, health and conservation communities. With this intersectoral cooperation, the currently bleak status of food security and environmental integrity could be managed to develop a trajectory for success.

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