The influence of livestock ownership and health on the nutritional status of children in Eastern Africa

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Abstract

The influence of livestock ownership and health on the nutrition status of children in Eastern Africa

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Background: In many rural low-resource settings, chronic malnutrition-induced linear growth faltering is widely prevalent. In these same areas, household livestock ownership is ubiquitous. Livestock may positively or negatively influence nutritional status through a variety of pathways, including improving food security but also increasing exposure to infectious diseases. We sought to determine whether 1) livestock ownership and 2) livestock disease were associated with growth outcomes among children under five years of age in Eastern Africa.

Methods: We used two data sources to address these aims. To test whether livestock ownership influenced child growth outcomes, as measured by stunting prevalence (<-2 standard deviations of Height-for-Age Z-score), we first analyzed the most recent Demographic and Health Surveys (DHS) for Ethiopia (2011), Kenya (2008-2009), and Uganda (2011). We also evaluated the question of livestock ownership and child growth within an ongoing Kenya Medical Research Institute (KEMRI)-Centers for Disease Control (CDC) demographic and health surveillance cohort in rural Western Kenya. The cohort monitors both human and livestock disease, and our team incorporated monthly anthropometry measurements for children. Using data from both the DHS and the surveillance cohort, we tested whether higher numbers of livestock ownership were associated with child nutrition outcomes using linear regression models clustered by household. To assess whether livestock health status was associated with child growth outcomes, we used the Western Kenya surveillance data to

evaluate each child's overall and time-varying exposure to livestock disease and subsequent growth using linear mixed regression models.

Results: The DHS analysis included n=8079 children from Ethiopia, n= 3903 children from Kenya, and n=1645 from Uganda. A ten-fold increase in household livestock ownership was significantly associated with lower stunting prevalence in Ethiopia (Prevalence Ratio [PR] 0.95, 95% CI 0.92-0.98) and in Uganda (PR 0.87, 95% CI 0.79-0.97), but not in Kenya (PR 1.01, 95% CI 0.96-1.07).

In the surveillance cohort in Western Kenya, we monitored the growth of 1097 children at least once over the course of 11 months. Higher household livestock ownership at baseline was not related to baseline child height-for-age z-score (β= 0.006 SD, 95% CI -0.02, 0.04) or prospective monthly child growth rate (β=0.002cm, 95% CI -0.003, 0.006). Further, over the entire duration of follow-up, higher numbers of any livestock disease in a household was not related to average 6 monthly growth rate of children in the same household (under 2 β= -0.045, 95% CI -0.186, 0.096; over 2 years β=0.006, 95% CI-0.031, 0.044). However, in the time-varying models of acute livestock disease and 3-month child growth intervals, we observed a trend by which children grew less after exposure to livestock disease, particularly among those children under age two.

Conclusion: The DHS analysis for Ethiopia, Kenya, and Uganda demonstrated a small beneficial impact of livestock ownership on reducing child stunting. The small effect size may be related to limitations of the DHS dataset or the potentially complicated relationship between malnutrition and livestock ownership, including livestock health and productivity. In the cohort of children in Western Kenya, ownership of livestock did not appear to be significantly associated with improvements in linear growth. However, disease in livestock may be associated with short term growth detriment. One Health, a concept by which human, animal, and environmental

health improvement are integrated, provides approaches to prevent disease in livestock may promote optimal child growth and nutrition in rural households.

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INTRODUCTION

Malnutrition in children under 5 years of age is a complicated global problem, resulting from a collection of interwoven factors. Unfortunately, progress towards global targets for chronic malnutrition- related stunting reduction has been slow, and new approaches to address malnutrition are necessary [1]. A renewed focus on the household environment may help identify novel areas for interventions to prevent stunting. Given the close proximity in which household livestock and young children live in many settings, interventions in livestock may provide new avenues to improve the health of the entire household. The following chapter provides the motivation for evaluating household livestock in relation to child growth by outlining the causes and outcomes of stunting, and by reviewing the human-animal health overlaps with regard to human nutritional status.

Childhood growth failure (stunting) is a seemingly intractable problem afflicting large portions of the population of low and middle income countries [2]. Stunted children have a height measurement in the lowest percentiles for their age compared to a reference population of children of the same age and sex [3, 4]. This poor growth can be the result of infectious disease, insufficient nutrient intake, poor intestinal absorption, stress, or toxin exposures[5-9]. Stunted children not only grow insufficiently in height, they also have deficiencies in the development of other body organs and systems. Physiological and immunological changes resulting from malnutrition include lymphatic atrophy, reduced antibody production, reduced stress response, and decreased fasting fatty acid oxidation [10-12]. Stunted children have an increased risk of mortality, cognitive deficits, and chronic disease, such as obesity and diabetes [13, 14]. Stunting is commonly considered to be irreversible after age two [15]. However, a stunted infant can exhibit catch-up growth in particular settings, such as after infection-mediated growth faltering [16].

Although stunting is rooted in poverty and wealth disparity, improvement in per-capita gross domestic product does not appear to be directly associated with stunting prevalence at the country level [17-19]. As a result, it is likely that direct community and household interventions to improve child growth are necessary to address stunting. However, in contrast with clinical interventions for acute malnutrition, interventions to reduce or prevent stunting have demonstrated minimal effectiveness. Acute malnutrition (define?), despite having a high case-fatality, can often be effectively treated with appropriate clinical management [20]. Interventions to address stunting have focused on prevention, and investigators have conducted trials of breastfeeding, micronutrient provision, supplemental feeding, school feeding, and sanitation to promote child growth [21-23]. However, the impact of these interventions on growth and stunting prevention has been minimal. Even if each were scaled up effectively in low resource settings, only a small portion of stunting would be prevented [21].

The One Health framework promotes the interconnectedness of human, animal, and environmental health in health research and programs [24]. Despite originally gaining hold in the realm of emerging zoonotic disease such as avian influenza, SARS, and bovine spongiform encephalopathy[25], One Health is evolving to encompass the daily interactions of humans, animals, and their environment[26]. Recent studies in One Health have evaluated companion animal obesity, antimicrobial resistance, and other health issues that arise from animals and humans living in close proximity[27-29]. Livestock studies have been a large component of One Health research, often with a focus on diseases such as bovine tuberculosis, brucellosis, and campylobacter[30-33]. New integrated platforms can allow for further research into daily interactions with livestock that could not be addressed in studies of solely human, animal, or environmental systems separately[34].

In rural households within low-resource settings, livestock are ubiquitous. However, the interactions between livestock and the nutritional status of owners and their family members

complex, and household livestock have the potential to either benefit or harm child growth outcomes. In rural areas of low and middle income countries, healthy livestock provide income for families, as well as directly influence food availability and feeding practices[35]. Randomized trials have shown that animal-sourced foods can improve growth and development in children [36, 37]. However, studies of the direct influence of increased livestock ownership on child growth are uncommon. A 2003 cross-sectional econometric modeling investigation found that household dairy cattle ownership is associated with approximately 5% lower prevalence of moderate or severe childhood stunting [38]. A recent cross-sectional study in Kenya of 183 children also showed a small benefit of female-owned livestock for improving child nutrition (an increase of 0.06 weight-for-age z-score per unit livestock), but no benefit for male-owned livestock [39]. Some intervention trials with a focus on livestock production improvement have successfully improved animal-sourced food intake among children, but anthropometric measures are often not collected so that linear growth outcomes cannot be t assessed [40, 41]. Despite this low grade of evidence for the improvement of child growth through livestock production, many non-profit (or NGO) organizations provide livestock to families to alleviate poverty and reduce malnutrition [42-44]. Longitudinal evidence on the influence of livestock ownership numbers on child growth is necessary to make conclusions on the overall benefit of livestock ownership.

Livestock and children share a close household environment in low resource settings, creating opportunities to share certain bacterial, viral, parasitic, and fungal pathogens through zoonotic transmission[45-47]. Livestock in low-resource settings often live very near or within human housing structures, and have the capacity to transmit human pathogens both during a subclinical infection or carriage, or while exhibiting symptoms [48, 49]. As described above, infectious disease and the intestinal malabsorption sequelae of clinical and subclinical infection

in children are important causes of malnutrition [8, 50-52]. However, the impact of zoonotic disease transmission on linear growth in children has not been evaluated [53].

Research into the interactions between livestock and children at the household human-animal interface can provide new information about the home environments where chronically malnourished children live. As the human-focused intervention approaches described above appear to be insufficient impact to reach global targets for stunting reduction, new approaches are necessary. Optimizing the benefits of livestock and concurrently reducing the potential for harm can provide children an ideal home environment for nutrition and growth.

This dissertation provides information on key pathways within the conceptual model of livestock and child growth We leveraged both existing national-level survey data and a comprehensive new surveillance platform in Western Kenya, to evaluate three specific aims regarding livestock ownership and health. Using national-level survey data, the first aim addresses whether livestock ownership is related to cross-sectional child growth outcomes in three East African countries. The second and third aims are based on a prospective cohort of children in Western Kenya, and address whether household livestock ownership patterns and livestock disease exposure in early childhood are related to growth outcomes among children. The data presented in the following chapters can help inform future interventions for child stunting prevention, provide an initiation point for further research into specific pathways within household human-livestock interactions, and promote the One Health framework for human disease alleviation.

CHAPTER ONE: THE RELATIONSHIP BETWEEN LIVESTOCK OWNERSHIP AND CHILD STUNTING IN THREE COUNTRIES IN EASTERN AFRICA USING NATIONAL SURVEY DATA

1.1 INTRODUCTION

More than a quarter of the world's children suffer from chronic malnutrition, resulting in linear growth failure (stunting), cognitive delay and increased risk of morbidity and mortality [54]. These consequences have profound implications for population health and economic development because adults who were stunted as children have been shown to receive less education and achieve lower earnings [14, 55]. Rural areas in many resource-limited settings shoulder an unequal burden of malnutrition; rural children have nearly fifty percent higher stunting prevalence than children in urban areas [56]. In order to address stunting where the need is greatest, appropriate interventions must be developed which will be adopted by the families based on individual and community priorities [57].

Rural children commonly live in close proximity to livestock. These livestock may be an important determinant of child nutritional status and promoting livestock production is a common development strategy. However, the overall influence of livestock ownership on child nutrition and stunting is not well understood, as very few studies have examined the association . A recent cross-sectional study in Kenya showed a small benefit of overall livestock ownership on child weight [39]. This effect could be mediated through livestock serving as direct sources of protein through meat, milk, and eggs, or indirectly by increasing household income for food, education or healthcare expenditures. Randomized trials have also demonstrated that animal-sourced foods can improve weight gain and muscle development in children [36, 37]. However, livestock ownership may also increase exposure to environmental contamination with fecal material and zoonotic pathogens [58, 59]. These exposures can lead to growth stunting by increasing overall metabolic demand, decreasing appetite, and by causing inflammatory patterns that reduce enteric absorption of nutrients [52, 60]. Fecal pathogens are closely linked

to diarrheal illness, which is a strong predictor of stunting in children in low resource settings [61]. Finally, environmental enteric dysfunction, as a result of exposure to contaminated environment, appears to be strongly related to linear growth failure [52, 61]. Understanding the effect of livestock on child growth may provide opportunities for interventions in animals to improve healthy development of young children.

The Demographic and Health Surveys provide publicly available datasets designed to monitor health status in over 90 countries at 5-year intervals. These rich datasets include both child growth and livestock ownership information. Anthropometric measures are recorded from children under 5 years of age and are often used to assess national burden of malnutrition. Several studies have evaluated the cross-sectional risk factors for child stunting using DHS data and have generally reported that greater wealth, education, and sanitation are associated with lower stunting prevalence [62-64]. The DHS also collects data on livestock ownership at the household level, although this measure is not routinely evaluated in association with child stunting.

Using existing DHS data from Ethiopia, Kenya, and Uganda, we sought to determine whether these publicly available cross-sectional data demonstrate a relationship between livestock ownership and child stunting prevalence, and whether these data can identify characteristics of high risk households that could benefit from a targeted animal-human health intervention.

1.2 MATERIALS AND METHODS

Datasets

This study was conducted as a regional analysis of the most recent DHS datasets from three East African countries, including Ethiopia (2011), Kenya (2008-2009), and Uganda (2010). Overall data collection methods for each DHS involved two-stage cluster designs and are described elsewhere [65]. In Ethiopia, a representative sample of 17,817 households was

selected for the 2011 DHS[66]. For Kenya, a nationally representative sample of 8,444 women and 3,465 men was selected from 400 clusters throughout Kenya, which provided representative estimates for eight subdivisions[67]. The Ugandan survey included a representative sample of 10,086 households[68]. Data were restricted to rural households as the relationship between animals, wealth, and nutrition may be very different in urban households. Children sampled in these households were between 0 and 59 months of age.

Variable definitions

Consistent with WHO practices, we categorized children as stunted if they had a height-for-age z score of less than −2 standard deviations below the WHO 2006 reference mean[3]. Children were only included in the analysis if they had a value for both height and age.

Livestock ownership numbers, including number of cattle, chickens, sheep, and goats, are included in the DHS as an asset available for use in the principle components analysis of the wealth index. Horses and donkeys are also enumerated in the dataset, but animals which are owned mainly for the purpose of load-bearing were not included in this analysis. Although camels can be an important livestock species in many areas throughout Ethiopia and Kenya, they were only available in the Ethiopia dataset and so were not analyzed here. The use of livestock as an exposure for human health outcomes is uncommon, so we applied three exploratory approaches for exposure measurement. First we created a total sum variable which gave each livestock species equal weight. This variable was highly skewed and a natural log was calculated. Second, we used counts of livestock as separate species. Third, a weighted measure of livestock was calculated using a Tropical Livestock Unit (TLU) scores. The TLU is a metric developed by the Food and Agriculture Organization (FAO), which allows for the combination of multiple species of livestock into a weighted measure representing total body weight and potentially market value. A single animal weighing 250kg represents a single TLU,

providing weighting factors of 0.7 for cattle, 0.1 for sheep, 0.1 for goats, and 0.01 for chickens[69]. This measure was also skewed, and we created five categories of TLU ownership to be roughly reflective of potential differing household livestock composition.

To identify if animal ownership was associated with a stronger or weaker association with stunting in certain subgroups of children, we stratified the association between livestock ownership and child stunting by diarrheal disease, region, wealth index (not including animals), and animal sourced food (ASF) intake. These variables were chosen *a priori* as categories by which the relationship between livestock and child stunting might be modified (e.g., if the effect of animal ownership is mediated through ASF, then those children who are fed ASF and have livestock may have a stronger positive association). Recent diarrheal disease was defined as a caregiver's positive response to the question "Has (NAME) had diarrhea in the past two weeks?" where 3 loose stools within a single day were classified as diarrhea. Region was defined by the data collection team as the location where the household was sampled. Regions in Kenya have recently been redefined to reflect county-level governments, but due to the time of this survey, original province designations were retained. Children were considered to have consumed ASF if the child's caregiver answered "yes" to whether the child ate eggs, meat, organ-meat, or dairy products in a 24-hour food recall. Although other feeding variables would potentially provide further information on the relationship between livestock ownership and child nutrition, indicators such as feeding frequency and dietary diversity were not consistently available in the datasets and thus were not included.

To create a wealth index which did not include livestock, we conducted a Principal Components Analysis (PCA) using indicators for use of surface water, the time it takes to obtain water, roof type, floor type, number of people per room, household electricity, television ownership, refrigerator ownership, bicycle/motorcycle/car ownership, telephone ownership, mobile phone ownership, use of shared toilet, and amount of land owned[70]. We used the first component

score to create a quintile measure of wealth. We also included a binary indicator for maternal education as a further adjustment for confounding. This indicator was defined as yes if the child's mother completed any education.

Statistics

All analyses were conducted using Stata/SE 11 (StataCorp, LP). We described livestock and child health indicators by country using means and proportions. These were weighted by sample weights provided in the DHS dataset to account for sampling scheme, which allowed for accurate national-level estimates. For proper weighting, the datasets were maintained separately by country.

The association between household animal ownership and child stunting was assessed using General Estimating Equations (GEE). For this analysis, we included all children under age 5 in each dataset, which included some children living in the same household who were therefore exposed to the same levels of livestock ownership. To account for the correlation of stunting outcomes of children living in the same household, we used an exchangeable correlation structure. The GEE used a log-link and a binomial family to provide comparative prevalence ratios. This model did not use survey weights, in accordance with DHS data user recommendations[65]. After evaluating the univariable association, the association between livestock ownership and stunting was adjusted for wealth index and region. The model was further stratified by ASF, diarrheal disease, region, and wealth index. Hypotheses were tested using Wald statistics.

This analysis resulted in extensive multiple comparisons within the three datasets. Due to these multiple comparisons, we chose to adjust for False Discovery Rate (FDR) using the Benjamini-Hochberg method, as a less-conservative method compared to the Bonferroni method[71]. All of the subgroup analyses in each country dataset were considered part of a total of 97 tests. We

used this total number of tests to correct the statistical significance cut-off in order to control the percentage of significant results that were false positives. Using this method, the highest p-value was compared to a cut-off of 0.05, but the lowest p-value was compared to a cut-off of 0.0005.

This study used publicly-available de-identified data, for which IRB review and approval was not necessary from the University of Washington, nor from the coauthors' institutions.

1.3 RESULTS

Among 8720 children from Ethiopia, 8079 had height measurements among 5528 households. In Kenya, 3903 children (of 4203 total children) in 2602 households had height measurements, and in Uganda 1645 in 1025 households (of 1740 total children) had height measurements. Livestock ownership was variable by country (Table 1.1). Overall, Ethiopia had the highest number of animals per household. The highest mean type of animal per household in Ethiopia and Kenya was goats, while in Uganda it was chickens. However, the most commonly owned animal in both Kenya and Uganda was chickens (63.2% and 60.3% of households owned chickens). The most commonly owned animals in Ethiopia were cattle, which were owned by 71.8% of the households. Within countries, the pastoralist regions in northern Ethiopia and northern Kenya had much higher average herd sizes than in the southern regions, where farms tend to be small and comprised of a mixture of livestock-based and cash-crop farming. Stunting prevalence was high in each country at 29.1% in Kenya, 29.4% in Uganda, and 39.7% in Ethiopia (Table 1.1).

Higher numbers of livestock and of Tropical Livestock Units (TLUs) were marginally associated with lower stunting prevalence (Table 1.2), although many of the associations were not statistically significant. Log livestock count was associated with decreased stunting prevalence in Ethiopia (adjusted PR 0.95, 95% CI 0.92-0.98) and Uganda (adjusted PR 0.87, 95% CI 0.79-

0.96), but not in Kenya (PR 1.02, 95% CI 0.97-1.07). Higher TLU households trended towards lower stunting prevalence, but the association was not significant at a cut-off value of α=0.05. Stunting prevalence decreased slightly by TLU livestock category, but the trend was not significant (Figure 1.1).

After adjusting for wealth, recent diarrheal illness in children was associated with an increased prevalence of stunting in Ethiopia (adjusted PR 1.10, 95% CI 1.02-1.08) and Kenya (adjusted PR 1.13, 95% 1.00-1.27), but the association was not significant in Uganda (PR 1.14, 95% CI 0.96-1.35) (Supplementary Table 1.1). Similarly, the asset-based wealth score demonstrated a protective association with stunting in Ethiopia (PR 0.94, 95% CI 0.92-0.97) and Kenya (PR 0.87, 95% CI 0.84-0.91), but not in Uganda (PR 0.97, 95% CI 0.91-1.03). The measure for 24-hour recall of ASF intake was not found to be associated with stunting in any country.

The association between TLUs and stunting prevalence did not vary by wealth index or diarrheal illness (Table 1.3). In Ethiopia, the association differed across region; in Dire Dawa an increase of one TLU was associated with 22% less stunting (Table 1.4). The child's 24-hour ASF intake did not modify the association between the livestock and stunting prevalence in any country. In each country, we also evaluated whether ownership of cattle, goats, sheep, or chickens as individual species were related to child stunting. Holding constant wealth, region and other animal ownership, none of these species was independently related to stunting status (data not shown).

Although several of these tests suggested small but statistically significant impact of animal ownership on stunting using a universal p-value = 0.05, when the Benjamini-Hochberg method to adjust for multiple comparisons was applied, only three regional associations in Ethiopia remained statistically significant (Oromiya, SNNPR, and Dire Dawa). None of the overall

associations between livestock and stunting were strong enough to remain significant after adjusting for multiple comparisons.

1.4 DISCUSSION

This analysis provides an initial approach towards understanding child stunting and livestock ownership at the household level in rural Eastern Africa. Livestock are nearly ubiquitous in these households and have many potential relationships with child growth. In most of the analyses presented here, the relationship between livestock ownership and child stunting demonstrated a trend towards a protective association. Only a few other studies have examined the overall association of livestock ownership and stunting, and prior reports have been consistent with our findings of small effect sizes [38, 39]. These results suggest that there may be room to improve the extent to which livestock provide benefit to child nutrition. Additional research is necessary to understand the specific populations for whom, and methods by which, livestock can benefit child growth.

Healthy child growth and development is contingent on a delicate balance between optimal diet, sanitation and overall health. As such, stunting in children could be the result of protein or calorie insufficiency, micronutrient deficiency, repeated infection, environmental enteropathy, or intra-uterine growth restriction [52, 57, 61, 72]. Household livestock may be related to many of these causes. Livestock can have positive impact on macro and micronutrient deficiency through animal-sourced food provision. However, if families do not utilize animal-sourced foods, the livestock cannot provide this direct benefit. In this analysis, 24 hour recall of animal-sourced foods was not related to stunting status or livestock ownership, which may partially explain the lack of significant benefit. However, the animal-sourced foods measure itself was only a cross-sectional 24 hour yes/no recall of intake, which may be an unstable estimate of true usual consumption. The average of multiple days of intake is necessary to obtain a reasonable

estimate of long-term intake due to large day-to-day variation in diet, even in low resource settings [73].

Livestock may also be related to infectious disease among children. Livestock-associated wealth may allow households greater ability to access services such as improved water and sanitation, or health care. As a result, ownership of healthy livestock may improve the child's environment and decrease the impact of infectious diseases that would otherwise lead to stunting. However, it is plausible that sick or asymptomatically infected livestock could also transmit zoonoses and add to contamination. This pathway has not been well-evaluated and further research is needed. Interventions addressing the complex nature of livestock, sanitation and veterinary care practices could be optimized to improve child nutrition and growth in these settings.

The small effect sizes reported here may suggest a truly negligible relationship between livestock and child stunting, as there are some causes of stunting which would not be related to childhood environmental conditions. For example, children who were born small-for-gestational age comprise up to 20% of children who are stunted between ages 1 and 5 years [72] and these children may not be impacted by livestock ownership during childhood. In addition, previous studies have suggested that a very minimal proportion of stunting can be reversed with optimal nutritional intervention, suggesting that interventions such as livestock provision may be unable to dramatically alter the progression of linear growth failure in these children [57]. In these instances, stunting status may not show the full benefit of livestock ownership. Additional outcomes such as cognitive development and educational attainment may be importantly linked to livestock ownership, but we are unable to evaluate these outcomes with the DHS dataset.

Finally, from a statistical standpoint, these DHS datasets provide national-level data from multiple years. The associations reported here are averaged over many groups of people, each

with different relationships with their animals. Fortunately, large datasets such as these also provide the opportunity to identify subgroups in which the effect size might be larger or smaller. In this analysis, effect modification, other than by sub-region, was not strongly apparent. Additionally, the DHS data were not collected to analyze livestock ownership patterns. The DHS collects animal ownership data for the purpose of creating a wealth index, and thus the exposure measures are not sufficiently comprehensive enough to distinguish nuances of the patterns of livestock ownership and child growth. There is no estimate of household ASF production levels, and the feeding measures are restricted. However, because it is a health survey, the outcomes in the DHS are at the child level, which allows for an analysis of child nutrition that is not possible using several extant livestock datasets which lack human data. This analysis is also limited in that it is cross-sectional. A longitudinal survey would allow for a temporal understanding of the influence of livestock on child growth, such as whether higher livestock ownership is important during critical growth windows, or whether livestock influence growth velocity.

Livestock production improvement programs are common in rural areas of developing countries as a means to promote income generation and improve nutritional status. Further, many large international charities provide assistance to rural families in the form of livestock gifts. Although this study did not formally evaluate this practice, it suggests that animal ownership alone may have only a small influence on the prevalence of stunting among young children. Longitudinal trials of livestock donation on nutritional outcomes would help to inform these interventions to ensure the maximum benefit. Potential ways to enhance livestock production interventions could include provision of education on ASF feeding practices, ensuring veterinary care for animals, and promoting livestock sanitation. Although national-level data can give some insight into the overall influence of livestock ownership on child stunting status, further specific studies with

greater resolution on household livestock ownership and production are necessary to dissect these relationships.

1.5 TABLES AND FIGURES

Table 1.1: Household and child characteristics from Ethiopia 2011, Kenya 2008-2009 and Uganda 2010 DHS surveys

Characteristic	Ethiopia (N=8720)	Kenya (N=4203)	Uganda (N=1740)
Household indicators			
Traditional livestock unit, mean(SD)	2.34 (0.10)	1.55 (0.21)	0.90 (0.09)
Chicken count, mean (SD)[a]	3.18 (0.13)	5.91 (0.36)	5.25 (0.29)
Cow count, mean (SD) [a]	3.41 (0.15)	1.53 (0.28)	1.50 (0.18)
Sheep count, mean (SD) [a]	1.92 (0.19)	3.78 (0.63)	0.56 (0.11)
Goat count, mean (SD) [a]	4.27 (0.42)	6.10 (0.85)	2.22 (0.13)
Wealth score, mean(SD)[b]	2.68 (0.04)	2.64 (0.07)	2.67 (0.06)
Child Indicators			
Height for age, mean Z-score (SD)	-1.53 (1.67)	-1.19(1.57)	-1.30 (1.46)
Stunting[c], %(SD)	39.7% (0.01)	29.2% (0.01)	29.4% (0.02)
Recent diarrheal illness %(SD)	14.4% (0.01)	15.0% (0.01)	22.3% (0.01)

[a] Among households which own any animals
[b] No animals are included in the wealth score
[c] Defined as height-for-age z-score lower than 2 standard deviations below the reference mean

Table 1.2: Log-binomial models for the relationship between household livestock ownership and stunting prevalence in Ethiopia, Kenya, and Uganda

Model	Prevalence Ratio Estimate (95% CI)		
	Ethiopia	**Kenya**	**Uganda**
Log total livestock, unadjusted	0.98 (0.95-1.01)	1.02 (0.97-1.08)	0.87 (0.79-0.96)
Log total livestock, adjusted*	0.95 (0.92-0.98)	1.01 (0.96-1.07)	0.87 (0.79-0.97)
TLU**, unadjusted	0.99 (0.98-1.00)	1.00 (0.99-1.01)	0.92 (0.87-0.99)
TLU, adjusted*	0.99 (0.98-1.00)	1.00 (0.98-1.01)	0.94 (0.88-1.00)
TLU category, adjusted*			
No animals (ref)	-	-	-
<0.1 TLU (a few chickens)	1.10 (0.94-1.28)	0.97 (0.83-1.13)	0.99 (0.78-1.24)
0.2-0.7 TLU (chickens or goats)	1.03 (0.92-1.15)	0.96 (0.84-1.10)	0.81 (0.66-0.98)
0.8-1.4 (one or two cows)	0.98 (0.87-1.10)	0.80 (0.66-0.98)	0.91 (0.68-1.22)
>1.5 TLU (more than 2 cows)	0.89 (0.80-1.00)	0.92 (0.78-1.09)	0.80 (0.61-1.05)

*Adjusted for wealth score, education, and region
**Tropical Livestock Unit: weighted livestock score combining chickens, cows, sheep, and goats

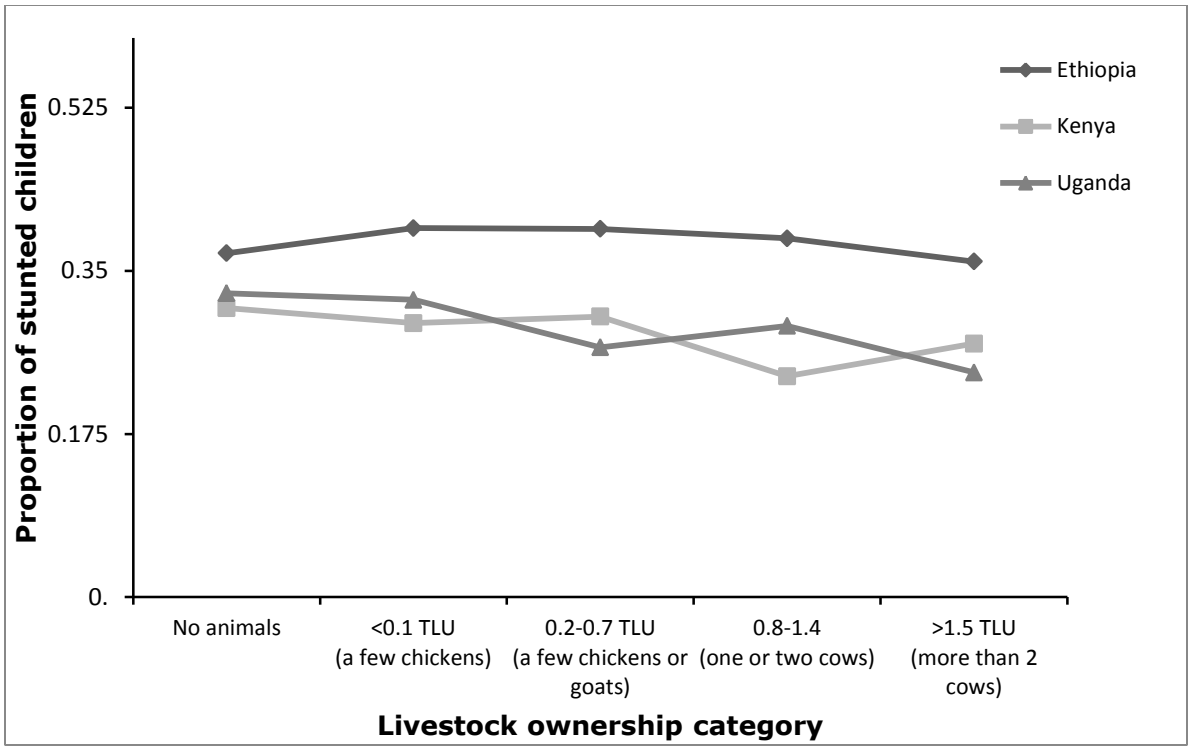

Figure 1.1: Unadjusted proportion children in households who are stunted, by livestock ownership category in Ethiopia, Kenya, and Uganda

Table 1.3: Log-binomial models for the relationship between household livestock ownership and stunting prevalence, as stratified by wealth and two-week diarrheal disease history

Model	Level	Prevalence Ratio Estimate (95% CI)		
		Ethiopia	**Kenya**	**Uganda**
TLU stratified by wealth index	Poorest	0.99 (0.97-1.01)	1.01 (0.99-1.02)	0.88 (0.72-1.07)
	Poorer	0.99 (0.97-1.01)	1.01 (0.98-1.03)	0.75 (0.61-0.94)
	Moderate wealth	0.95 (0.92-0.99)	0.89 (0.80-1.00)	0.98 (0.84-1.13)
	Wealthier	0.99 (0.96-1.01)	0.85 (0.75-0.97)	0.99 (0.90-1.10)
	Wealthiest	1.03 (0.96-1.08)	1.04 (0.95-1.15)	0.97 (0.87-1.08)
	Interaction	p=0.576	p=0.131	p=0.022
TLU stratified by diarrheal illness	(Yes)	0.98 (0.97-1.00)	1.00 (0.99-1.02)	0.93 (0.86-1.00)
	(No)	1.01 (0.98-1.03)	0.99 (0.96-1.03)	0.97 (0.87-1.07)
	Interaction	p=0.065	p=0.961	p=0.607

Table 1.4: Log-binomial models for the relationship between household livestock ownership and stunting prevalence, as stratified by region

Ethiopia		**Kenya**		**Uganda**	
Region	**TLU PR (95% CI)**	**Region**	**TLU PR (95% CI)**	**Region**	**TLU PR (95% CI)**
Tigray	0.99 (0.96-1.03)	Central	0.84 (0.64-1.09)	Central 1	0.91 (0.66-1.26)
Affar	1.00 (0.99-1.01)	Coast	0.98 (0.85-1.14)	Central 2	0.90 (0.71-1.13)
Amhara	0.97 (0.93-1.01)	Eastern	1.01 (0.99-1.03)	East Central	1.11 (0.94-1.31)
Oromiya	0.93 (0.89-0.98)	Nyanza	0.96 (0.81-1.14)	Eastern	0.92 (0.77-1.11)
Somali	0.99 (0.95-1.04)	Rift Valley	0.99 (0.96-1.02)	North	0.78 (0.54-1.13)
Benishangul-gumuz	0.96 (0.91-1.02)	Western	0.78 (0.50-1.20)	Karamoja	0.95 (0.80-1.10)
SNNPR	0.92 (0.88-0.97)	Northeastern	0.99 (0.97-1.01)	West-Nile	0.92 (0.79-1.08)
Gambela	1.01 (0.99-1.04)	Interaction	p=0.355	Western	1.01 (0.91-1.12)
Harari	1.06 (0.90-1.24)			Southwest	0.63 (0.39-1.00)
Dire Dawa	0.78 (0.67-0.91)			Interaction	p=0.389
Interaction	p=0.0004				

CHAPTER TWO: THE IMPACT OF LIVESTOCK OWNERSHIP AND HEALTH ON CHRONIC CHILDHOOD MALNUTRITION IN WESTERN KENYA

2.1 INTRODUCTION

Optimal early childhood growth is necessary for long-term health and cognitive function [14]. As such, child growth measures serve as important indicators for population health and economic well-being [54]. Post-natal growth is determined by multiple factors, including adequate access to nutritionally diverse foods, prevention and management of childhood infections and social support within the community [54]. In many rural settings, household livestock such as cows, goats, sheep and chickens may be key determinants of many of these factors, as livestock can provide animal-sourced foods and may serve as an important source of income. However, animal husbandry practices may also include trade-offs, such as an increased risk of exposure to pathogens [58, 59, 74, 75].

Child growth velocity varies by age and individual, and follows a daily model of rapid increases followed by static periods [76]. Throughout infancy, children experience steep gains in length, the velocity of which slows around one year of age [77]. Growth trajectory can be diminished by negative energy balance, either from decreased nutrient intake or increased metabolic demand from infections. Although catch-up growth can occur after these insults, it may be incomplete, leading to a permanent height deficit [78, 79]. This height deficit, known as stunting, is associated with higher risk of acute morbidity, long-term chronic disease and educational deficits [14].

Although few studies have tested the direct effect of livestock ownership on child growth, several studies have shown a slight benefit of livestock ownership on child growth outcomes [38, 39]. Livestock production is a common income-generating activity, which can potentially benefit child nutrition through better access to healthcare, sanitation, or nutrition [35]. Additionally, direct consumption of eggs, meat, and dairy can improve children's dietary

diversity, which is an important contributor to linear growth [75, 80]. Consumption of cow's milk has been demonstrated to improve child growth in both developed and developing settings [81, 82]. As a result of these benefits, livestock production interventions, such as dairy intensification or livestock donation, often have the goal of improving child nutrition. However, child growth and health outcomes have not been commonly evaluated as metrics in programs providing these interventions. In a systematic review of the impact of livestock production improvement on nutrition-related outcomes, only one study evaluated child growth and no significant associations were reported [41].

While the negligible effects of livestock ownership reported in previous studies may be the results of study design and sample size issues, they may also reflect potential disadvantages to increased family livestock ownership. For example, a recent study suggested that dairy intensification could result in a decrease in exclusive breastfeeding behaviors due to the availability of cows' milk [83]. Further, livestock in resource limited settings often have a high incidence of disease, and many livestock pathogens have zoonotic potential [8]. Environmental contamination with livestock fecal pathogens could increase the risk of clinical and subclinical infection in children, which can directly impact linear growth [84, 85]. Disease in livestock may also lead to loss of household income through the loss of investment in these animals [34, 86, 87]. As a result of livestock's' roles as a source of wealth, food, and as a potential source of fecal contamination and pathogen transmission, disentangling the relationship between household livestock and child growth has proven challenging.

In this study, we sought to evaluate the relationships between household livestock ownership, individual-level animal-sourced-food intake, episodes of livestock disease, and child growth trajectory in a surveillance cohort in Western Kenya.

2.2 METHODS

Study Design

This large prospective cohort study was nested within a human-animal health platform in Western Kenya in collaboration between the Kenya Medical Research Institute (KEMRI), CDC-Kenya, Washington State University and the University of Washington. Human health and livestock surveillance systems overlap in 10 villages (1500 households) within a 5km radius near Lake Victoria [34]. All households participating in both surveillance systems with children who were under 5 years in June 2014 were eligible for inclusion in this cohort. Caregivers provided informed consent for participation in the study. The follow-up timeline for the study is depicted in Figure 2.1.

The anthropometric component of this study was approved by the KEMRI Ethical Review Board as an amendment to the parent surveillance study in March, 2014.

Data collection

A study team of four experienced human health interviewers were trained in anthropometric measurements and questionnaire delivery. From June 2014 to May 2015, the team collected height and weight measurements on children under 5 years every month during household visits within the existing KEMRI-CDC data collection infrastructure [88, 89]. The team measured length for children under age two and height for children over two years using a Shorrboard®. Weight was measured using a digital Mother/Child standing scale. Mid-upper arm circumference (MUAC) was assessed using standardized tape for the purpose of screening and referral for acutely malnourished children (defined as an arm circumference of <11.5cm for children over 6 months of age [90]). The study team also conducted a three-day food frequency questionnaire with the child's caregiver. The 32 item survey was based on locally-appropriate and pretested food questionnaires, and included the number of times each food was given to the child over the

previous three days. Information on consumption of eggs, milk, meat, and chicken was specifically requested.

Baseline livestock ownership and economic data were collected by a separate team through an in-depth quarterly economic questionnaire between February 2013 and June 2014, as described elsewhere [34]. Trained interviewers asked the household head a series of questions about assets, livestock (cows, sheep, goats, and chickens), income, and expenditures. Based on the quarterly assessment schedule, these data were available for up to 5 potential time points.

Livestock disease information was collected between March 2014 and January 2015 by a team of veterinary technicians, also described elsewhere [34]. Briefly, human health community interviewers requested information on whether or not there were any sick livestock in the household during a biweekly human health interview. Farmers could also report an animal disease directly through a toll-free phone line. If a household reported a livestock illness, veterinary technicians responded within 48 hours to examine and diagnose the animals, collect biological specimens, and provide treatment if necessary. Livestock disease reports subsequently subcategorized disease for cows, goats, and sheep into nine syndromes: Death, reproductive issues (abortion, stillbirths or neonatal deaths), respiratory issues (cough, nasal discharges, difficulty breathing), digestive problems (diarrhea, bloody diarrhea, bloating), urogenital issues, mastitis, musculoskeletal problems, skin diseases, and nervous system disorders. Chicken reports were only recorded through the system if multiple birds within a flock had died.

Data analyses

All analyses were conducted in Stata/SE 11 (StataCorp, LP). All tests were two-sided and were considered statistically significant if the confidence intervals did not include zero, or if the p-value was <0.05.

Cohort Description

To describe the cohort, we calculated means and proportions of baseline child and household characteristics across a binary variable indicating whether any livestock illness had been reported over the entire livestock disease reporting period (March 2014-January 2015). We compared child height, age, and sex measurements to the World Health Organization (WHO) 2006 reference to create continuous measures for Height-for-Age z-score (HAZ) and Weight-for-Height z-score (WHZ) [3, 4]. Stunting and wasting were defined as less than -2 standard deviations below the reference mean [91].

We described animal ownership over the baseline period by providing mean household counts and variability. Livestock disease reports are described as the proportion of disease reports in each syndrome category, as well as the incidence rate of new livestock disease. We considered the livestock population as an open population and was calculated the incidence rate by dividing the syndrome report counts in each species by the estimated total livestock population for each species among these households over six months.

Analysis of baseline livestock ownership and child nutrition

Variables

In order to include as many households as possible and to account for variability and wealth at the household level, we calculated baseline livestock and wealth scores over a 15 month period before child growth measurements began in June 2014 (see Figure 2.1). For baseline livestock counts, we created a single average household count for each livestock species (chickens,

cows, sheep, and goats). To create the total livestock score, we combined livestock species into both an unweighted sum and a weighted sum using Tropical Livestock Units (TLU). The TLU is a standardized livestock score for a household which weights each livestock species according to their mass [69]. To account for some of the variability in livestock ownership over the baseline period, we calculated a second summary measure as the maximum number of each animal within each household to create cut-off categories: for example, low chicken ownership (never owned above 5 chickens over the baseline period), moderate chicken ownership (owned, at some point, more than 5 chickens, but never owned more than 15 chickens), high chicken ownership (ever owned more than 15 chickens). We created an asset-based wealth score using the household's ownership of farming implements, bikes, vehicles, radios, tractors, phones, motorbikes, televisions, computers, electronics, many buildings, and latrines in a principle components analysis based on cofactors from previous literature and cultural aspects relevant to our study population [70]. The first component was used as a wealth score for each time point, which was then averaged across all baseline time points at the household level. Both livestock ownership and wealth score were household level variables.

The child's baseline HAZ (or LAZ), baseline WHZ and prospective monthly growth rate were considered the outcomes for this analysis. We analyzed a subset of the cohort for which an initial child height measurement was available within three months of June 2014, and a second measurement was available at least six months after their initial measurement. Baseline HAZ and WHZ were used in their first available instance between June and August 2014. Monthly growth rate was calculated as the difference in height over at least a six-month interval divided by the number of months of measurement available for that child. HAZ and height measurements were excluded *a priori* if the HAZ was below -5 SD. Animal-sourced food consumption was measured as a binary indicator for whether or not the primary caregiver

reported that the child ate eggs, milk, meat, or chicken in the three days before the anthropometric measurement.

Statistical analysis

The primary analysis tested the influence of baseline livestock characteristics on baseline child nutrition and subsequent growth trajectory. We constructed a multi-level linear regression model and used the child's baseline HAZ, baseline WHZ or prospective monthly growth rate as the outcome. The models used random effects for the household because many households included more than one child. Each child was only included in the model once. The main predictor in this model was baseline livestock ownership, which we included as the livestock sum or the categorical variable for maximum number of animals owned as described above. After evaluating the unadjusted association, we controlled for the *a priori* factors of child age, child sex, and household wealth. We conducted an additional model which adjusted for whether the child received any breastfeeding at baseline.

We also tested whether reported animal-sourced food intake at baseline was associated with child baseline HAZ, baseline WHZ, or prospective monthly growth rate, also using a multi-level linear regression with random effects for the household. We further tested whether animal-sourced food consumption was related to child age, wealth status, or livestock ownership in a single model. These analyses were restricted to children who were greater than 6 months of age at baseline.

Analysis of livestock disease and child growth

Variables

For this analysis, we evaluated whether livestock disease was related to child growth over three-month intervals as well as over the entire duration of follow-up. Data were restricted to

those households which owned any livestock over the baseline period, as described above. Livestock disease was prospectively evaluated at the household level from March 2014 through January 2015. Disease was defined in two ways: as a report of any livestock syndrome within the household over time-varying 30-day increments, or as the total household sum of reports of disease between March 2014 and January 2015. A disease report could consist of a report of livestock death, digestive disorder, skin disorder, reproductive disorder, urogenital disorder, udder disorder, neurological disorder, respiratory disorder, or musculo-skeletal disorder in any livestock species. We also separately evaluated report of animal digestive syndromes as an exposure due to the relationship between diarrhea and child stunting [8]. Further stratifications by syndrome type or livestock species type were not conducted due to small numbers of disease reports on the household level.

The child growth outcomes for this analysis were either time-varying 3 month height gain following the monthly livestock disease report interval, or average 6-monthly growth over the duration of follow-up. These durations of follow-up was chosen *a priori* as a length of time by which livestock illness might influence growth through zoonosis or loss of wealth. For the time-varying three month growth intervals, height gain outcomes were calculated as the difference between the child's height three months after the livestock syndrome report interval minus the child's height directly after the livestock syndrome report interval. Children could also only be included in these models if they had at least two height measurements three months apart. However, each child could provide multiple observations to this analysis. The six-monthly growth rate was calculated as the last height measurement available minus the first available, divided by the number of months contributed and multiplied by six. Children had to provide a height measurement within three-months of baseline, and needed to have at least three months of follow-up to be included. Children could only provide one average growth rate observation to this analysis.

Covariates in this analysis included child age (as a quadratic term), child sex, and month of livestock disease exposure (an indicator of season). The household-level baseline wealth and baseline livestock ownership were also used for adjustment.

Statistical analysis

To assess whether livestock disease influenced immediate three-month growth intervals, we generated linear mixed models with a random intercept for household, which accounted for multiple children within each household. We stratified these models by month of exposure, to allow for a single-month time varying exposure to be associated with a three-month growth outcome. The primary exposure was the binary variable for whether or not livestock disease had been reported in the child's household over the course of one month prior to the three-month growth follow-up. We recombined the exposure month-stratified estimates using Stata's "metan" function to assess the overall effect size of livestock disease report on short term growth outcomes.

To assess the overall effect of higher livestock disease burden on average growth rate, we generated a linear mixed model (also with a random effect for household) with the outcome of 6-monthly growth rate. The primary exposure was the sum of reported livestock within a household in the period between March 2014 and January 2015. The primary outcome was the average six-monthly growth rate of children in each household between June 2014 and May 2015.

After evaluating the unadjusted associations in each of these models, we controlled for child age, child sex, household baseline wealth, and baseline household livestock ownership count. Covariates were chosen *a priori* as potentially important predictors of child height, HAZ, or stunting incidence, or as potential confounders in the relationship between livestock syndromes

and child growth. We also chose to provide results separately for children under age 2 and children over age 2, due to the differences in growth slope at these ages.

Sensitivity analysis

Because this cohort was based within a surveillance system, some outcome data points were missing, which we assumed were missing at random. As a sensitivity analysis, we multiply imputed missing height values using Markov Chain Monte Carlo (MCMC) multivariate normal regression technique. We used ten imputed datasets. Estimates of the imputed regressions were derived using Rubin's combining rules through Stata's "mi" package [92].

2.3 RESULTS

Cohort description

Within the surveillance area, at least one height measurement was available for 1495 children. Among these children, 1097 children in 755 households also had baseline data on livestock and wealth and were included in the analysis. Children were available for an average of 6.5 out of 8 growth measurement visits, and 81% of monthly height observations were available overall. Those children who were missing height measurement data points in this cohort were found to be older, more likely to be female, and had higher household livestock ownership. In the sensitivity analysis where missing height values were imputed, no major differences in the direction or significance of the effects below were noted. Each of the primary exposures became slightly more strongly related to the growth outcomes after imputation.

The baseline age of children ranged from 1 month to 60 months. Figure 2.2 shows child height by age over the course of the 11 months of follow-up, as categorized by stunting status at each age. Among those for whom baseline and endlpoint values were available, 24.7 percent of children were stunted at baseline. At the end of follow-up, 26.4 percent of these children were stunted. Nine percent (46 children) of those who were not stunted at baseline became stunted

over the course of the study, while twenty-six percent (46 children) of those who began the study stunted were no longer stunted at the endpoint.

At baseline, there were a few notable differences in characteristics between children whose families reported livestock disease and whose families did not (Table 2.1). The percent of children with wasting was low in both groups, but lower among those families who reported any syndromes. Concordantly, the mean WHZ was higher among those families who reported any syndromes. Baseline wealth status quintile was higher, and baseline livestock ownership counts were much higher in those families who reported any syndromes throughout follow-up. Age, sex, HAZ, and percent stunting were fairly balanced between the two groups.

Analysis of baseline livestock ownership, animal-sourced foods, and child growth status

Livestock ownership varied both between households and within households (Figure 2.3). Households tended to have both high counts of chickens and high variability between quarterly measurements (within-household standard deviation: ±6.1 chickens). Households had lower counts of cows, sheep, and goats as well as lower variability both across households and within households over time.

Seven hundred fifty-seven children in 546 households had a height measure both at baseline and at least 6 months later, and were included in the regression analysis of baseline livestock and child growth. The mean baseline HAZ was -1.27SD (SD 1.12) and the subsequent mean child growth per month was 0.67cm (SD 0.19). In both adjusted and unadjusted analyses, baseline household livestock count was neither related to baseline child HAZ nor subsequent growth rate (Table 2.2). Baseline household livestock count was also unrelated to baseline WHZ (adjusted β= -0.005, 95% CI -0.03, 0.02). Using the weighted Tropical Livestock Unit score as the exposure did not meaningfully change this association, nor did the evaluation of maximum categories of livestock ownership. In exploratory analyses, baseline livestock ownership counts

were significantly positively related to both livestock disease ($p<0.001$) and baseline wealth ($p<0.001$) (data not shown). Supplementary Table S2.1 shows the association between livestock ownership and child growth adjusted for whether the child received any breastfeeding at baseline.

The caregivers of the children in the sample described baseline dietary patterns that varied across age. Among children under six months of age, 58.3% were reportedly exclusively breastfed at baseline. In the entire sample, 27.4% received any breast milk, and the oldest child receiving any breastmilk was 40 months. Children most commonly ate ugali (maize flour porridge), fish, and tomatoes. Eggs were the most commonly consumed livestock-sourced food, followed by milk, meat, and chicken. Three-day recall of animal-sourced food consumption of any kind was neither related to HAZ at baseline nor monthly growth rate among children over 6 months of age (data not shown). Table 2.3 shows the associations between 3-day recall of animal-sourced food consumption and child age, household livestock ownership and wealth. Each additional chicken owned at baseline was associated with a 14% increase in the likelihood of having consumed eggs in the past three days of the child's initial household visit ($p<0.001$). Milk was consumed more commonly by younger children (6% decrease in report of cow's milk consumption for additional month of age, $p<0.001$). Baseline household wealth was also significantly related to 3-day recall of any animal-sourced food, egg, and meat consumption.

Analysis of livestock disease and child growth

Between March 2014 and January 2015, 777 cases of any animal disease or death were reported among 258 households. Digestive disorders were most commonly reported, followed by death, skin disorders, and respiratory disorders (Table 2.4). Over any given month of follow-up, approximately 92% of households reported no livestock disease, 7% reported one livestock disease, and 1% reported more than one livestock disease. The incidence rate of "any syndrome reported" was highest among cattle, at 30.9 cases per 100 cattle-years (Table 2.5).

Digestive syndromes were also reported at the highest incidence rate among cattle, at 21.0 cases per 100 cattle-years. However, reported mortality rate was highest among goats at 6.8 deaths per 100 goat-years. Diseases other than death were not reported for chickens, though chickens had the second highest mortality rate at 6.4 per 100 chicken-years.

Seven-hundred sixty seven children had height measurements that were at least three months apart, and these children contributed 4,202 three-month growth intervals. The mean height gain over three months in this group was 2.1cm (SD 1.0cm). Age, sex, and measurement month were strong predictors of growth over time (Supplementary Table S2.2). Children grew at the greatest velocity under one year of age, and girls had larger gains in height over each 3 month period. Child growth demonstrated a strong parabolic association with season, where growth performance decreased from June through August, and then improved from September through December ($p<0.001$ for both first and second order trends).

Across three month growth intervals, an immediately prior report of livestock disease in the household was associated with a decreased growth slope, particularly among children under age two (-0.39cm per three months under two years, 95%CI -0.64, -0.13; vs. -0.07cm per three months over two years, 95% CI -0.18, 0.04) (Figure 2.4). This association was also apparent using digestive livestock disease as the exposure, but the effect was less strong among children under age two (-0.25 cm per three months, 95%CI -0.64, 0.13), and slightly stronger among children over age two (-0.12cm per three months 95% CI -0.25, 0.01) (Figure 2.5).

Over the duration of follow-up, the average six-month growth rate was 4.0cm (SD 1.2). The average six-month growth rate of children was not associated with the number of reports of livestock disease in a household (Table 2.6). The association did not differ across age group, and was not different using digestive livestock disease as the primary exposure.

2.4 DISCUSSION

This study describes the growth of children in rural Western Kenya over the course of 11 months in relation to family livestock ownership and health. Neither livestock ownership at baseline nor total count of livestock disease reports had a significant effect on child linear growth outcomes over follow-up. However, we showed a trend by which immediate livestock illness was associated with diminished growth over the following three months. Subgroup analysis suggests that the detrimental effect of livestock syndromes on child growth were largest for children under two years of age.

In this cohort, livestock ownership was not related to baseline HAZ, baseline WHZ, or prospective growth rate. Other studies have shown that a benefit of livestock ownership may be mediated through provision of animal-sourced foods [39]. Here, the proportion of recall of animal-sourced foods was moderately high at each time point, but we did not find a relationship between 3-day recall of animal sourced food and child growth rate. However, the recall period was short and we did not evaluate the portion sizes of eggs, milk, meat, or chicken consumed. The benefits of animal-sourced food for nutrition have been well-documented in other contexts [36, 39, 75], and educational support to ensure parent's knowledge of the value of a diverse diet in young children could help address this issue. A recent cluster randomized trial of community livestock education found a benefit for child growth even before livestock donation [93], suggesting that education can facilitate a beneficial relationship between child nutrition and livestock ownership.

We found a strong seasonal effect on child growth. Children grew increasingly poorly between July and October, and had a faster rate of growth between November and January. Consequently, the HAZ of children decreased between July and October and then increased between November and January. This effect may be related to the seasonality of diarrheal disease in Kenya [94, 95], and warrants further investigation.

We also saw that immediate livestock disease was associated with poor subsequent child growth outcomes, with large effect sizes among children under two years of age. There are multiple pathways that could explain this observation. First, zoonotic disease in animals could be transmitted to children, resulting in infection-mediated linear growth failure [8]. A recent study in the same cohort showed that livestock disease reports were significantly associated with human disease reports within the same household [34], which is supported by previous studies of animal husbandry [96]. Digestive issues, including diarrheal disease, were the most commonly recorded livestock syndrome in this study, and childhood diarrhea has widely known impacts on child growth [97]. Several diarrheal pathogens have the potential for zoonotic transmission, including *Salmonella spp, E. coli, Campylobacter spp,* and *Cryptosporidium* [98]. Aside from the potential for zoonotic disease, the livestock illness could result in decreased production and direct consumption of animal-sourced foods, and might further represent a loss in wealth. Disease in livestock could also possibly divert time and resources away from child caretaking.

However, over the duration of follow-up, we did not see that increased reports of livestock disease were associated with average six-monthly child growth rate. There are two potential reasons for this disparity in findings. First, given the strong improvement in growth seen in the latter half of the study period, it is possible that the children who experienced short term growth faltering exhibited catch-up growth. Children have been shown to catch-up in their growth after acute diarrheal disease [78], and several stunted children in the current study attained non-stunted status by the end. A second possibility is the influence of wealth-related confounding. In this study, wealth was not strongly related to short term growth rate, but was associated with higher baseline HAZ. Higher wealth was also strongly positively associated with higher livestock disease reports, due to each of their associations with higher livestock numbers. Potentially, over longer periods of time the positive association between wealth and child growth

outweighed the short term detriment of livestock disease. Although we adjusted for wealth based on the household assets, the possibility of residual confounding exists. We anticipate that this confounding could have attenuated a detrimental relationship between livestock disease and child growth outcomes.

This study has several key strengths. First, the use of systematically collected data on childhood growth, animal ownership and animal illness is unique. In addition, the multi-directional interactions driving these associations were carefully considered in the analysis. We used time-varying linear mixed models of livestock syndrome report and child growth outcomes, which allowed us to evaluate a temporally-appropriate relationship between livestock and child growth. However, the study also had limitations. Livestock disease reports, despite a reasonably high overall incidence, were low in number on the household level. For most analyses, we were unable to divide by species and syndrome type due to small numbers. Missing data was another limitation of the analysis, although the multiple imputations models showed a size and direction of effect for livestock ownership and disease similar to the complete case analysis. Additionally, the "metan" function in Stata does not account for correlated data, and some of the errors in the three-month growth analysis may have been underestimated due to the use of monthly child growth datasets. Finally, we were unable to include some major predictors of child growth, such as parental height and child birth weight. Although we believe that we have included the most important confounders, the precision of the estimates could have been improved by including these strong predictors of the outcome.

Domestic livestock are ubiquitous in rural households throughout many resource-limited settings, and provide a means of economic development for families. However, the household relationships between livestock and humans are complex, and our analyses suggest that there might be key intervention areas to improve the welfare of both humans and animals in rural settings. Optimizing the benefits of livestock for family economic gain and for child health

require integrated programs including veterinary care, water and sanitation interventions, education, and childhood disease prevention and control.

2.5 TABLES AND FIGURES

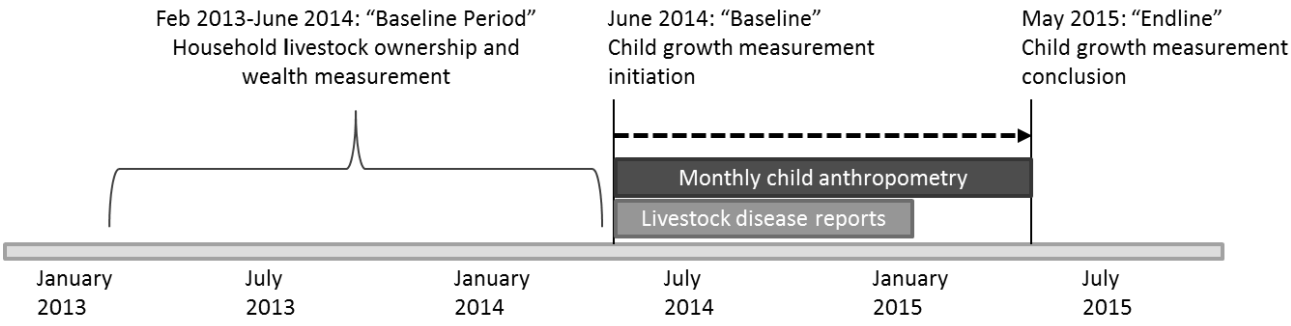

Figure 2.1: Follow-up timeline for livestock and child measurements in Asembo, Kenya

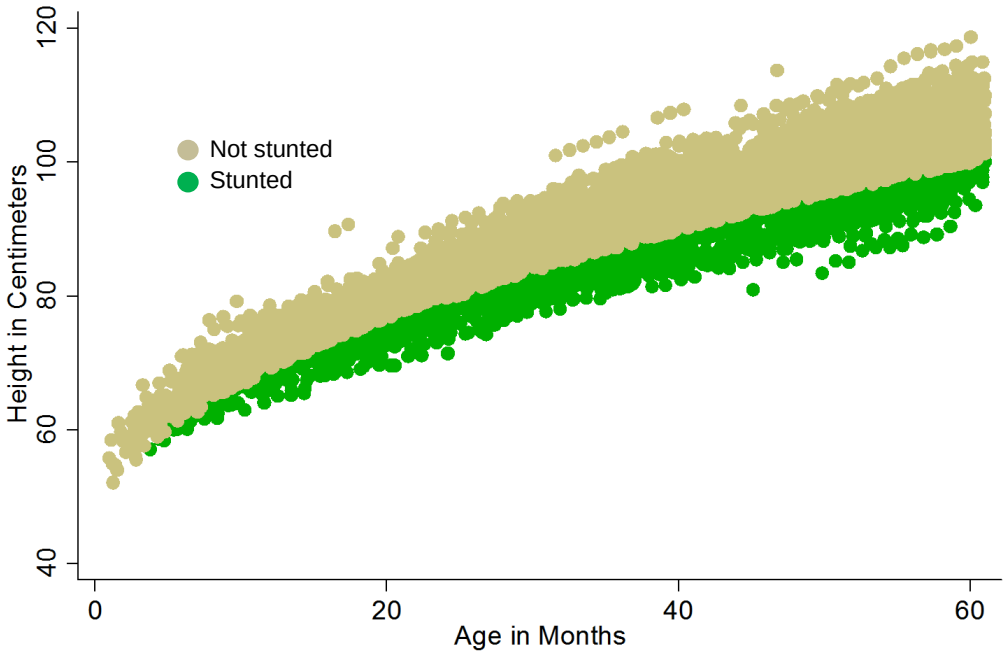

Figure 2.2: Child height by age over 11 months of follow-up, Asembo, Kenya

Table 2.1: Baseline cohort characteristics by livestock disease reports, Asembo, Kenya

Characteristic	Any livestock disease reported (n=298)	No livestock disease reported (n=478)	Overall
Child indicators			
Child age, months, mean (SD)	35.2 (16.2)	34.9 (16.7)	35.0 (16.5)
Child female, n(%)	168 (56.4%)	240 (50.2%)	408 (52.6%)
HAZ, mean(SD)	-1.2 (1.2)	-1.3 (1.2)	-1.3 (1.1)
WHZ, mean(SD)	0.0 (1.0)	-0.2 (1.1)	-0.1 (1.1)
Wasted, n(%)	7 (2.4%)	27 (5.8%)	23 (3.5%)
Stunted, n(%)	70 (23.4%)	118 (25.3%)	188 (24.7%)
3-day recall of animal-sourced food intake at baseline, n(%)			
Eggs	80 (26.9%)	109 (22.8%)	189 (24.4%)
Milk	49 (16.4%)	65 (13.6%)	114 (14.7%)
Chicken	23 (8.0%)	38 (7.9%)	61 (7.9%)
Meat	53 (17.8%)	77 (16.1%)	130 (16.8%)
Fish	197 (90.0%)	402 (86.0%)	599 (87.1%)
Household indicators			
Household wealth quintile*, mean (SD)	3.1 (1.4)	2.8 (1.4)	2.9 (1.4)
Livestock count*, mean (SD)	4.6 (3.6)	2.6 (2.0)	3.4 (2.9)
# cattle	3.1 (4.1)	1.0 (1.8)	1.8 (3.1)
# chickens	10.9 (7.8)	8.2 (6.8)	9.2 (7.2)
# goats	2.4 (3.4)	0.8 (1.5)	1.4 (2.6)
# sheep	1.8 (4.0)	0.3 (1.2)	0.9 (2.8)

*Household average over the past 15 quarters before baseline

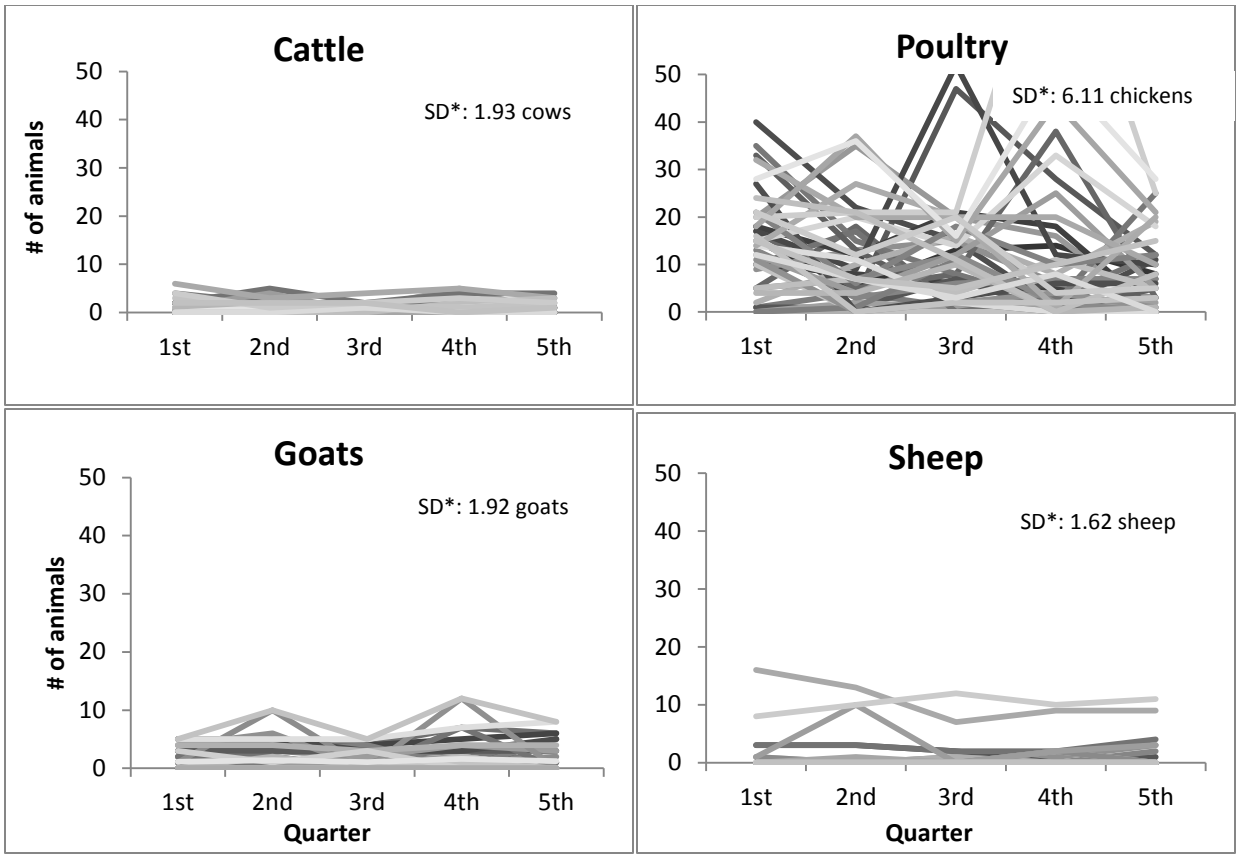

Figure 2.3: Livestock variability within and between households over 15 months before baseline. Each line represents the livestock ownership counts within one household over five 3-month intervals. SD*=Within household standard deviation over 5 quarters

Table 2.2: Linear mixed model of baseline average livestock ownership and baseline child height-for-age z-score (HAZ), clustered by household (N=757 children in 546 households)

	Baseline child HAZ		Monthly child growth rate	
Model	**Unadjusted, β (95% CI)**	**Adjusted, β (95% CI)**	**Unadjusted, β (95% CI)**	**Adjusted, β (95% CI)**
Overall livestock count				
Baseline livestock**	0.015 (-0.015, 0.045)	0.006 (-0.024, 0.036)	0.001 (-0.003, 0.006)	0.002 (-0.003, 0.006)
Child age, linear		-0.080 (-0.100, -0.060)		-0.012 (-0.015, -0.009)
Child age, quadratic		0.0012 (0.0009, 0.0014)		0.0001 (0.0001, 0.0002)
Child sex		0.179 (0.030, 0.329)		0.067 (0.043, 0.091)
Household wealth score		0.076 (0.003, 0.148)		0.010 (-0.001, 0.021)
Separate species				
Maximum cattle category				
No cattle (ref)				
Never > 4 cattle	0.087(-0.134, 0.308)	0.089(-0.127, 0.305)	-0.012 (-0.048, 0.025)	-0.002(-0.035, 0.030)
Ever > 4 cattle	0.095 (-0.150, 0.340)	0.074 (-0.165, 0.314)	0.001 (-0.040, 0.042)	0.006(-0.031, 0.042)
Maximum poultry category				
Never > 4 chickens (ref)				
More than 4, never > 14	-0.236 (-0.500, 0.027)	-0.266 (-0.522, -0.009)	-0.027 (-0.070, 0.017)	-0.030(-0.068, 0.009)
Ever > 14 chickens	-0.176 (-0.449, 0.098)	-0.216 (-0.484, 0.053)	-0.016 (-0.061, 0.029)	-0.016(-0.057, 0.024)
Maximum goat category				
No goats (ref)				
Never > 3 goats	-0.036 (-0.262, 0.191)	-0.019 (-0.240, 0.203)	0.011 (-0.027, 0.048)	0.010 (-0.023, 0.043)
Ever > 3 goats	0.027 (-0.196, 0.250)	-0.001 (-0.220, 0.219)	0.013 (-0.024, 0.050)	0.013 (-0.020, 0.046)
Maximum sheep category				
No sheep (ref)				
Never > 4 sheep	-0.044(-0.292, 0.205)	-0.091 (-0.333, 0.151)	0.025 (-0.016, 0.065)	0.016 (-0.020, 0.053)
Ever > 4 sheep	-0.028 (-0.339, 0.283)	-0.001 (-0.307, 0.305)	0.020 (-0.031, 0.071)	0.015 (-0.031, 0.061)
Child age, linear		-0.081 (-0.101, -0.061)		-0.012 (-0.016, -0.009)
Child age, quadratic		0.0012 (0.0009, 0.0015)		0.0001 (0.0001, 0.0002)
Child sex, female		0.187 (0.036, 0.338)		0.067 (0.043, 0.091)
Household wealth score		0.083 (0.009, 0.156)		0.009 (-0.002, 0.020)

** No notable differences in model results when TLU was used instead of counts

Table 2.3: Three-day baseline recall of child animal-sourced food (ASF) consumption relationships with livestock ownership, wealth, and child age, among children over 6 months of age

	3-day reported ASF intake at baseline, OR (p-value)			
Covariates	**Any ASF**	**Eggs**	**Milk**	**Meat**
Livestock ownership				
Average cow count	1.07 (0.379)	1.01 (0.871)	0.94 (0.516)	1.15 (0.233)
Average chicken count	1.07 (0.025)	1.14 (<0.001)	1.07 (0.020)	0.99 (0.908)
Average goat count	0.95 (0.571)	0.93 (0.480)	0.90 (0.333)	0.89 (0.397)
Average sheep count	1.00 (0.968)	0.90 (0.307)	1.02 (0.856)	0.99 (0.915)
Age, months	0.99 (0.354)	1.02 (0.082)	0.94 (<0.001)	1.00 (0.962)
Wealth score	1.65 (0.003)	1.41 (0.038)	1.32 (0.104)	2.07 (0.001)

Table 2.4: Livestock disease reports, June 2014-January 2015

Syndrome category	Signs	Reports, N(%)
Digestive	diarrhea, bloating, loss of appetite	410 (47.2%)
Death	death	105 (12.1%)
Skin	hair loss, itching, lumps	94 (10.8%)
Respiratory	disorders cough, nasal discharge, difficulty breathing	93 (10.7%)
Musculo-skeletal	lameness, recumbency	58 (6.7%)
Nervous	circling, incoordination	10 (1.2%)
Urogenital	vaginal discharges, preputial discharges, scrotal swelling	5 (0.6%)
Udder	mastitis, drop in milk yield	2 (0.2%)
Reproductive	abortions, stillbirths, neonatal deaths	0 (0%)

Table 2.5: Crude animal disease incidence rate and mortality rate

	Livestock syndrome reports (incidence rate)		
Livestock species	Any syndrome	Digestive syndrome	Mortality rate
Cattle, per 100 cattle-years	30.9 cases	21.0 cases	2.1 deaths
Goats, per 100 goat-years	29.7 cases	20.1 cases	6.8 deaths
Sheep, per 100 sheep-years	18.0 cases	10.6 cases	5.8 deaths
Chickens, per 100 chicken-years			6.4 deaths

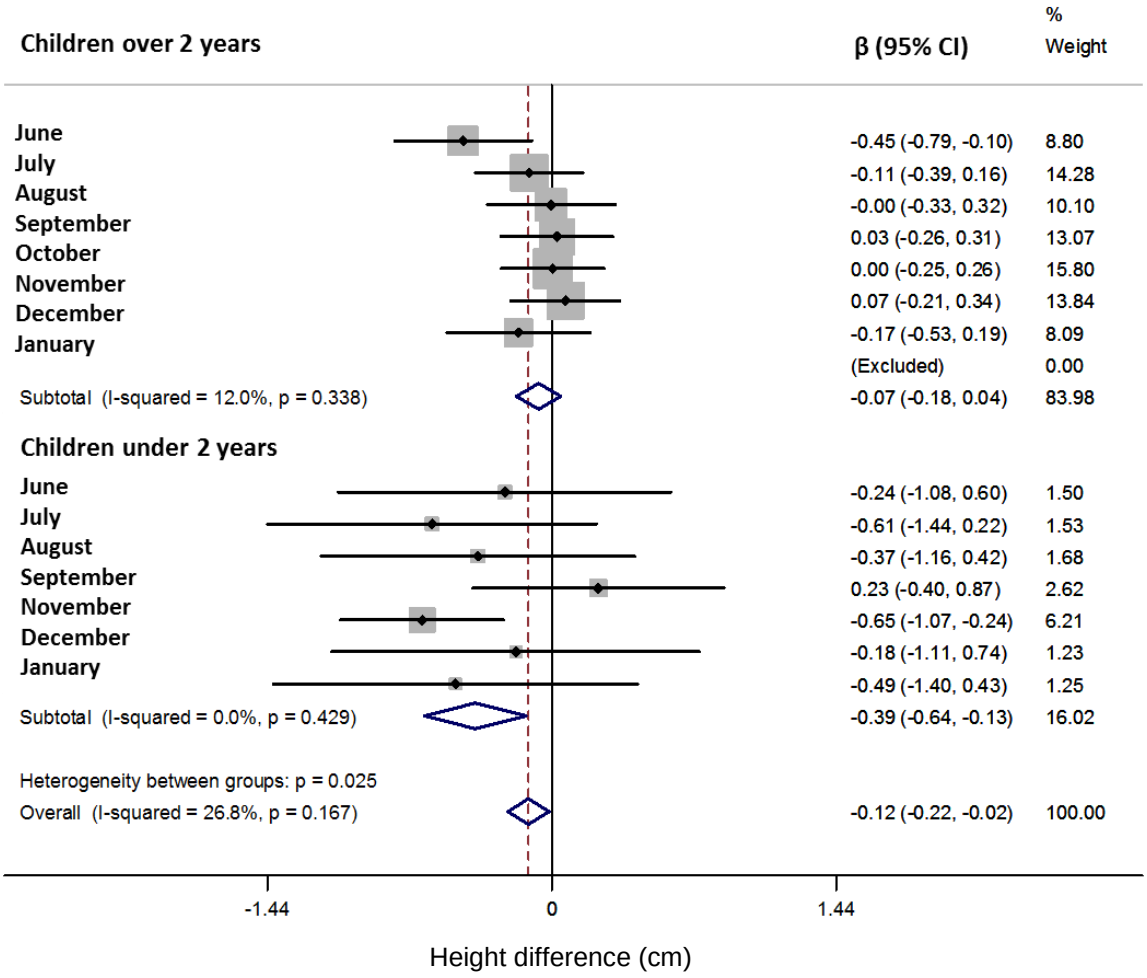

Figure 2.4: Association between household livestock disease report and subsequent 3-month child growth, separated by child age and by month of exposure.

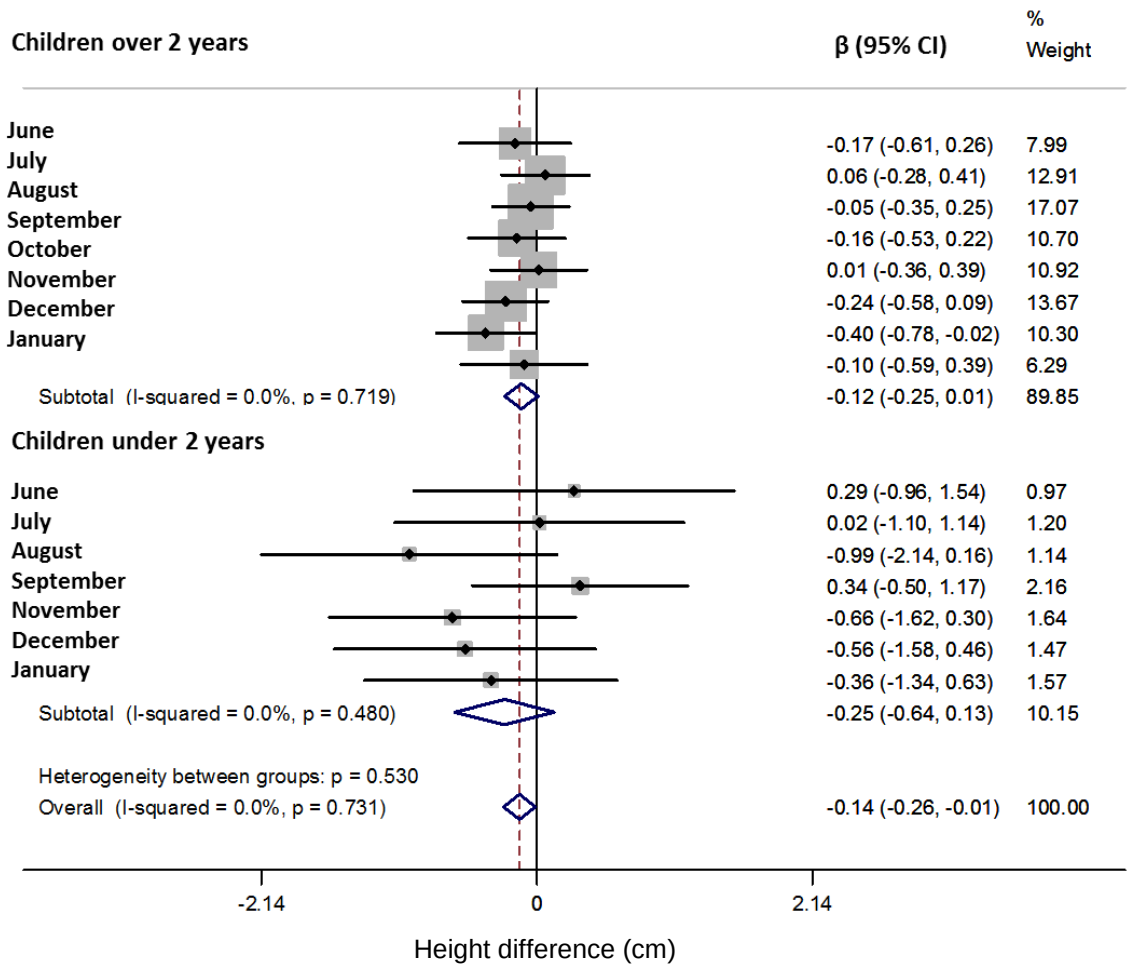

Figure 2.5: Association between household livestock digestive disease report and subsequent 3-month child growth, separated by child age and by month of exposure.

Table 2.6: Adjusted linear mixed model of household livestock disease count over the duration of follow-up and average 6 month growth rate.

	Average 6-month growth difference, β(95% CI)	
Any livestock disease model	Under age 2 (cm)	Over age 2 (cm)
Livestock disease (continuous count)	-0.002 (-0.10, 0.09)	0.01 (-0.03, 0.04)
Livestock ownership (continuous count)	0.02 (-0.06, 0.10)	0.01 (-0.02, 0.04)
Household wealth (continuous score)	0.12 (-0.04, 0.27)	0.02 (-0.05, 0.08)
Child sex (female)	0.41 (0.07, 0.75)	0.33 (0.19, 0.47)
Age (linear)	-0.49 (-0.62, -0.36)	0.01 (-0.05, 0.08)
Age (quadratic)	0.014 (0.01, 0.02)	-0.0004 (-0.001, 0.0003)
Digestive livestock disease model		
Digestive livestock disease (continuous count)	-0.02 (-0.17, 0.13)	0.003 (-0.05, 0.06)
Livestock ownership (continuous count)	0.02 (-0.05, 0.10)	0.01 (-0.02, 0.04)
Household wealth (continuous score)	0.11 (-0.04, 0.26)	0.02 (-0.05, 0.09)
Child sex (female)	0.41 (0.07, 0.76)	0.33 (0.19, 0.48)
Age (linear)	-0.49 (-0.63, -0.36)	0.01 (-0.05, 0.08)
Age (quadratic)	0.01 (0.01, 0.02)	-0.0004 (-0.001, 0.0004)

CONCLUSION

The analyses presented here suggest that the relationship between livestock and child nutrition may be important in order to provide benefit to both children and animals living in rural households. Although we did not find a strong benefit to owning more livestock on the household level, we observed a potential short term detrimental impact on child growth. The results of this dissertation provide several avenues for future research and potential interventions to promote child growth in rural areas of low and middle-income countries.

Across two different study designs, we found little association between higher livestock ownership numbers and child stunting status. In the national survey data used in Aim 1, Ethiopia and Uganda showed between a 5 and 13% decrease in stunting prevalence was found for each tenfold increase in animal ownership in the household. However, these associations were not robust to adjustment for multiple comparisons. The national survey data for Kenya showed no association between household livestock ownership numbers and child stunting outcomes. This result was replicated in our cohort study in Western Kenya; increased livestock ownership at baseline was not related to baseline growth indicators or prospective growth in children under age five. To further answer the question of whether increased household livestock ownership can promote healthy child growth, randomized controlled trials are an important next step.

Although there are many ways by which livestock could benefit child nutrition, there are potential trade-offs. However, the relationships between livestock disease and child growth that we observed in the Kenya cohort were not straightforward. Although we observed a short-term association between livestock disease and lower growth slope over three-months, this effect was not sustained over the duration of follow-up. These findings raise further questions and provide new directions for future research. First, it is unclear exactly how a short term growth detriment may be mediated. Livestock death and disease may lead to lower production, which could lead to food insecurity in the household. Alternatively, sick livestock could contaminate the

household environment or directly transmit zoonotic pathogens to children. Future research could evaluate the subsequent economic losses that result from animal disease, or assess specific pathogen transmission pathways between livestock and children. Additionally, the offsets between increased wealth and increased livestock disease (due to higher numbers of livestock among wealthier households) represent an important area for further research.

Interventions in livestock may present another future direction for this work. Interventions in animals with the goal of improving human health are rare, but exemplify an important way forward for the One Health initiative. The results of this research provide two potential points of intervention that might help prevent short term growth faltering among children. The first point of intervention would be to prevent disease among livestock. Veterinary care, vaccination, and sanitation within livestock settings could serve to prevent primary disease in household animals. The second point of intervention could be to reduce environmental fecal contamination and prevent transmission of zoonoses to children. Water, sanitation, and hygiene (WASH) interventions currently include promotion of water quality, water quantity, latrine use, and hand hygiene in low resource settings and have been shown to improve child growth [23]. However, WASH interventions tend to be focused on human sanitation and do not commonly involve livestock considerations. In the US, several pathogen-specific animal interventions are regularly practice in farm settings. For example, interventions to prevent the transmission of *Salmonella spp.* and *E. coli* to humans are common among pig and cattle farms [99-101]. However, sanitation interventions in livestock in low resource settings have rarely been studied and could be an essential way to prevent disease within livestock and transmission of disease from livestock to young children [102]. Primary prevention of livestock disease and further prevention of livestock-related environmental contamination represent areas for potential benefit for rural families in low and middle income settings.

Overall, we were able to demonstrate that higher numbers of livestock ownership are not necessarily associated with better nutritional status or growth among children. However, diseases in livestock could have a short-term impact on child growth. In order to maximize the benefit of household livestock for child growth and health, animal health should also be optimized.

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APPENDIX: SUPPLEMENTAL TABLES

Table S1.1: Associations between household wealth status, diarrheal disease, animal-sourced food intake and stunting prevalence in Ethiopia, Kenya, and Uganda

	Prevalence Ratio (95% Confidence Interval)		
	Ethiopia	Kenya	Uganda
Report of recent diarrheal illness (yes/no)[†]	1.10 (1.02-1.08)	1.13 (1.00-1.27)	1.14 (0.96-1.35)
Wealth status (linear quintile)	0.94 (0.92-0.97)	0.87 (0.84-0.91)	0.97 (0.91-1.03)
24-hour recall of child animal-sourced food consumption*[†] (yes/no)	0.91 (0.82-103)	1.01 (0.89- 1.14)	0.93 (0.75-1.14)

[†] Adjusted for wealth quintile

*Consumption of eggs, dairy, meat, or poultry, among children over 6 months of age

Table S2.1: Associations between household livestock and child growth outcomes, adjusting for household wealth status, child age, child sex, and whether the child was fed any breastmilk at baseline

	β (95% Confidence Interval)	
	Baseline HAZ (SD)	Monthly growth rate (cm)
Baseline livestock	0.01 (-0.02, 0.04)	0.002 (-0.003, 0.006)
Child age, linear	-0.07 (-0.10, -0.05)	-0.02 (-0.02, -0.01)
Child age, quadratic	0.001 (0.0007, 0.001)	0.0001 (0.00009, 0.0002)
Child sex	0.18 (0.03, 0.33)	0.07 (0.04, 0.09)
Household wealth score	0.07 (0.004, 0.15)	0.009 (-0.002, 0.02)
Reported any breastmilk at baseline*	0.11 (-0.17, 0.39)	-0.07 (-0.11, -0.03)

*Over three-day recall period

Table S2.2: Univariable associations between non-livestock predictors and three-month growth rate among children in Asembo, Kenya

	β(95% confidence interval)	
	3-month growth (cm)	3-month HAZ difference (SD)
Child age, linear	-0.02 (-0.04, -0.01)	0.05 (0.04, 0.05)
Child age, quadratic	0.0001 (-0.00, 0.0003)	-0.0005 (-0.0006, -0.0005)
Child sex (female)	0.17 (0.09, 0.25)	0.029 (-0.002, 0.06)
Baseline household wealth score	0.03 (-0.01, 0.06)	-0.001 (-0.01, 0.01)
Season		
June-September	(ref)	(ref)
September-December	-0.39 (-0.51, -0.26)	-0.05 (-0.10, -0.003)
December-April	0.76 (0.63, 0.89)	0.25 (0.21, 0.30)

VITA

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