EFFECT OF NEWCASTLE DISEASE VACCINATION ON SURVIVAL OF INDIGENOUS CHICKEN AND ASSOCIATION WITH CHILD NUTRITION AND GROWTH IN SIAYA COUNTY, KENYA

A thesis submitted in fulfilment of requirements for Doctor of Philosophy of University of Nairobi [Applied Microbiology (Virology option)]

Elkanah Sillus Orwa Otiang, BVM, MSc.

Department of Veterinary Pathology, Microbiology and Parasitology

2023

DECLARATION

This thesis is my original work and has not been presented for a degree in any other University.

Signature .... Date 4th December 2023

Elkanah Sillus Orwa Otiang

This thesis has been submitted for examination with our approval as university supervisors.

Prof. Philip Njeru Nyaga, BVM, MPVM, PhD.

Department of Veterinary Pathology, Microbiology and Parasitology,

Faculty of Veterinary Medicine, University of Nairobi, Kenya.

Signature ……………………… Date 4th December 2023

Dr. Lucy Wanjiru Njagi, BVM, MSc, PhD.

Department of Veterinary Pathology, Microbiology and Parasitology,

Faculty of Veterinary Medicine, University of Nairobi, Kenya.

Signature .... Date 4th December 2023

Prof. Thumbi Mwangi, BVM, MSc, PhD.

Centre for Epidemiological Modelling and Analysis, University of Nairobi, Nairobi, Kenya

Paul G. Allen School for Global Health, Washington State University, Pullman, WA, USA

Institute of Immunology and Infection Research, University of Edinburgh, Edinburgh, UK

Signature .... Date 4th December 2023

DEDICATION

This thesis is dedicated to my loving parents; the late Robert Otiang and Janet Oduol, and family; Beryl, Elyn, Rachel, Jean, and Robyn for their great support, encouragement, sacrifices and prayers during this journey.

ACKNOWLEGMENTS

This thesis would not have been possible without the support and guidance of many people. First, I acknowledge the Almighty God for His grace and favor in the opportunity to participate in this exciting research work anchored within Kenya Medical Research Institute (KEMRI) platform.

I acknowledge the following institutions and individuals for making this research a reality; Washington State University Paul G. Allen School for Global Health under the stewardship of Prof. Guy Palmer for funding, and mentorship, Prof. Jonathan Yoder, and Dr. Zoe Campbell for mentorship and Adaptive Management and Research Consultants (AMREC) under Dr. Vincent Were, and Mudis Ogutu for statistical guidance. My sincere gratitude to my university supervisors; Prof. Philip Njeru Nyaga, Dr. Lucy Wanjiru Njagi, and Prof. Thumbi Mwangi for their mentorship, guidance, support, and critique during this work. Also, I thank the following who made the field research possible: Field team supervisor; James Oigo, Animal Health Technicians; Samwel Asembo, Geoffrey Odima, Bob Miseda, Beryl Oyoo, and Austin Ochung'; Community Health Interviewers: Joseph Onyango, Daniel Odongo, Rosemary Warinda, Bridon Kojo, Thomas Wachiaya, Fredrick Ong'wen, Meresia Owuor; Martin Aundi, Fredrick Adhiambo, Benedine Warinda, Jane Okal, and Fred Ojode; Data Programmer and Manager, Linus Otieno; and Data Clerk, Judith Oduol.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

ACUC	Animal Care and Use Committee
AHTs	Animal Health Technicians
APMV	Avian Paramyxovirus
ASF	Animal Source Foods
BAUEC	Biosafety, Animal Use and Ethics Committee
CI	Confidence Interval
CBS	Central Bureau of Statistics
CGHR	Centre for Global Health Research
CSC	Centre for Scientific Committee
HAZ	Height-for-age z-score
HDSS	Health and Demographic Surveillance System
HIV	Human Immunodeficiency Virus
ID	Identification
IFAD	International Fund for Agricultural Development
FAO	Food and Agriculture Organization
IEIP	International Emerging Infections Program
KEMRI	Kenya Medical Research Institute
MAM	Moderate Acute Malnutrition
MoH	Ministry of Health
MUAC	Mid-upper arm circumference
NAM	No Acute Malnutrition
WOAH	World Organization for Animal Health
PBASS	Population Based Animal Syndromic Surveillance
PBIDS	Population Based Infectious Disease Surveillance

PhD	Doctor of Philosophy
SAM	Severe Acute Malnutrition
SDG	Sustainable Development Goal
SERU	Scientific and Ethics Review Unit
SES	Socio-Economic Surveillance
SSC	Scientific Steering Committee
UNICEF	United Nations International Children's Emergency Fund
UNDP	United Nations Development Programme
US CDC	United States Centre for Disease Control and Prevention
US$	United States of America Dollar
USA	United States of America
WA	Washington State
WHO	World Health Organization
WHZ	Weight-for-length/height z-score
WOAH	World Organization for Animal Health
WSU	Washington State University

ABSTRACT

Newcastle disease (ND) is a significant factor in chicken deaths worldwide and more so in smallholder flocks resulting in reduced household incomes, food security, and increased risk of malnutrition among rural communities in developing economies. Reducing mortality associated with ND through vaccination can lead to increase in egg and chicken numbers. This study determines: the effect of ND vaccination on survival and poultry production among local breeds in Siaya county; nutritional and socioeconomic factors associated with child growth and development for chicken owning small holder households; and estimates the impact of ND vaccination of indigenous chicken on the child growth and nutrition.

Data were obtained from households in a rural region in Siaya county, Kenya over an 18-month period (December 2016 to September 2018) that were participating in a human and animal population-based syndromic survey study. A randomized control trial was used to compare households in the intervention group (whose chicken were vaccinated against ND) and those in the control group (did not receive ND vaccination). Households were eligible for the study if they owned indigenous chicken, had a child below five years of age or had a pregnant woman. All households that consented were recruited and randomly selected for either control or intervention. Chicken flocks in the intervention category received ND AVIVAX I-2 thermostable vaccine quarterly. All households got parasitic control. Each month, information on the number of domestic chickens and any reported gains or losses were gathered for the study period. Anthropometric assessments of children, dietary intake and household socioeconomic data were completed at recruitment and at every 3 months for the period of the study. A total of 537 households (n=254 across the intervention, n=283 within the control) were recruited with 471 households (n=222 intervention, n=249 control) having children below 3 years of age at enrolment. Monthly data over the study period showed households in the intervention owned more chickens (mean of 13.06±0.29) compared to those in the control (12.06±0.20) (p=0.0026). Children in households receiving treatment demonstrated general gains in Z results for both Z-score for height versus age (HAZ) and Z-score for weight-for-length/height (WHZ) by 0.158 and 0.075 respectively compared to control households, favouring both boys and girls.

Households where the flock received vaccination combined parasiticide treatment, increased flock size by a mean of one bird per home with a subsequent increase in eating foods high in protein and micronutrients, and a relative decrease in intake of grains abundant in carbohydrates but poor in protein therefore providing new proof that changing one's diet improves height for age and weight for height, important indicators of childhood stunting and wasting. The findings suggest that maximizing productivity of indigenous chickens through integrated disease control can significantly improve livelihoods and nutrition in children lowering the likelihood of stunting and wasting in children.

The outcomes show proof of positive gains in directing interventions to livestock assets managed by women in rural communities, potentially providing a pathway for women empowerment and prevention of malnutrition among children and women in these settings.

CHAPTER ONE: GENERAL INTRODUCTION

1.1.Informational context

Chicken raised locally can significantly influence economies and welfare of rural households through being a source of nutritious foods (meat and eggs), household earnings from sales, and a form of investment and savings (Randolph *et al*., 2007).

The extent of their influence on family health and welfare is greatly limited by production constraints such as diseases, lack of feeds, and low access to markets (Maina, 2005; Njagi *et al.*, 2010; Thumbi *et al.*, 2015). ND has a high morbidity and mortality rates (Ahlers *et al.*, 2009) and it's vaccination reduces mortality rates and improves survival, thus increasing chicken numbers and flock productivity.

Chicken rearing is primarily the responsibility of women who directly benefit from the financial gains from chicken sales and the sale of their products (Kristjanson *et al*., 2012). This may lead to healthier families since women are more prone to put spending first of their income on education, wholesome nutrition, and medical care (Valdivia, 2001).

Three major paths connect human and animal health: (i) Socioeconomic pathway, which promotes improved livestock health and welfare and production which goes hand in hand with improved household incomes and wealth hence better livelihood that would impact on better educational accessibility and healthcare, (ii) nutritional pathway, where owning healthy animals provides access to animal source nutrients, which lowers the risk of child malnutrition and diseases, and (iii) zoonotic pathway, where healthier livestock are less likely to spread zoonotic and food-borne illnesses (Randolph *et al.*, 2007; Thumbi *et al.*, 2015). This research is anchored on socio-economic and nutritional pathways as the two would reflect on impacts of ND vaccination on the well-being of households and populations which has not been studied in Kenya.

ND is a contagious viral disease effectively preventable through vaccination (Zeleke *et al*., 2005). The vaccines exist as either live or killed forms, containing avirulent, lentogenic, mesogenic, and velogenic strains (killed form), respectively, which confer varied immune responses (Copland and Spradbrow, 1997; Gallili and Ben-Nathan, 1998; Copland and Alders, 2005). Despite local availability of the vaccine, and resultant vaccination benefit of increased flock sizes and improved household welfare, their uptake by smallholder farmers is low and the factors that impede it together with other existing disease control measures are poorly understood (Ochieng *et al*., 2012). The rural households in Siaya county, Kenya own an average of fourteen birds per household with availability and consumption of animal-source foods, namely: both poultry meat and eggs, being directly related to the ownership of chickens (Thumbi *et al.*, 2015).

Up to 95% of rural households own indigenous chickens, with a significant variation in numbers with the mean ownership being fourteen (14) and one-third of households having less than five while others may have as many as forty (40) (Thumbi *et al.*, 2015). Given that there is a significant increase in egg consumption for every ten (10) chickens owned, increasing the flock size is one strategy for enhancing childhood nutrition at the household level through accessible eggs and chicken meat, and enhanced income levels from sale of chicken and their products (Mosites *et al.*, 2015; Thumbi *et al.*, 2015). Hence, it was hypothesized that remedies could ease chicken mortality would result in home decisions affecting children's meals and growth through improved productivity. This study aimed to investigate the impact of ND vaccination on flock morbidity and mortality in indigenous chicken and subsequent benefits on the livelihood and welfare of household specifically on child-mother nutritional status and socioeconomics.

1.2. The objectives

1.2.1 General objective

Investigation of effects of ND on survival of indigenous chicken, child dietary intake and growth, and household socioeconomics in Siaya county, Kenya.

1.2.2 Specific objectives

The study's specific objectives were to:

I. Determine the effects of ND vaccination on survival of indigenous chicken.

II. Determine the relationship between nutritional and socioeconomic factors with child growth and survival of indigenous chicken.

III. Estimate the impact of ND vaccination of indigenous chicken on the child growth and dietary intake.

1.3 Hypotheses

I. ND vaccination does not affect survival and productivity of indigenous chicken.

II. ND vaccination does not contribute to nutritional and socioeconomic factors that enhance child growth and development among smallholder poultry farmers.

III. ND vaccination does not result in significant improvements in dietary intakes and nutritional status of children.

1.4 Justification

Siaya county, Kenya is reported to have high levels of child undernutrition with projected stunting and wasting prevalence at 23.5% and 4.8%, respectively (Mosites *et al*., 2016a). Improved livestock health and productivity has been conceptualized to reduce the risk of child malnutrition through nutritional, socio-economics, and zoonotic disease control pathways and with approximately 90% of households in Siaya county, Kenya having indigenous chicken, their potential to improve household living conditions and food security is inevitable (Thumbi *et al*., 2015).

ND as a single infection contributes to over 50% of chicken deaths (Ondwasy *et al.*, 2006; Olwande *et al.*, 2010), and if controlled through vaccination can lead to higher numbers of chicken and more productivity. Although there are existing ND vaccines for use, there is limited quantitative data on the effect of vaccinating indigenous chicken on household livelihood benefits such as child nutrition and growth.

These previous studies point to enhanced production of indigenous chicken improving livelihoods and nutrition in children below five years and highlight infectious diseases to be a challenge for indigenous poultry production. This study aims to find empirical evidence of removing the constraint of Newcastle disease among indigenous chicken flocks and measures the effect on household chicken ownership, productivity and influence on household economics, diets, and risk of malnutrition. When production outcomes and welfare of household are enhanced, the positive results guide development of public health and agricultural policies associated with ND vaccine usage, poultry productivity and pursuant health, and improved livelihoods.

CHAPTER TWO: LITERATURE REVIEW

2.1 Newcastle disease

2.1.1 Aetiology

ND is an infection caused by an avulavirus of the family Paramyxoviridae. Avian paramyxoviruses are classified into ten serotypes, numbered from APMV-I to APMV-10, with ND being assigned APMV-1. Based on the clinical characteristics of infected chickens, the illness is further divided into five pathotypes: viscerotropic velogenic, neurotropic velogenic, mesogenic, lentogenic or respiratory, and asymptomatic. Groupings of pathotypes are rarely precise (Alexander, 2000; Alders and Spradbrow 2001; Alders *et al*., 2002; Getabalew *et al*., 2019).

The virus is rendered inactive by temperatures of 56°C for three hours, 60°C for 30 minutes, or an acid with a pH of 2. The virus may persist for extended durations at room temperature, especially in feces, and is chemically sensitive to ether, formalin, phenolics, and oxidizing chemicals including chlorhexidine and sodium hypochlorite (6%) (Alexander, 2000).

2.1.2 Epidemiology and clinical signs

In many bird species, both domestic and wild, virulent strains are endemic; they affect all age groups throughout the majority of Africa, Asia, and certain nations in North and South America. Rates of morbidity and mortality vary according to the virus strain, bird species, age, and management, as well as related illnesses and pre-existing immunity brought on by paramyxoviruses. When the virus is exposed to humans in significant quantities, the infection may result in eye reddening that may be unilateral or bilateral, severe lachrymation, oedema of the eyelids, conjunctivitis, and sub-conjunctival hemorrhages (Alders and Spradbrow, 2001; Alders *et al*., 2002; Young *et al*., 2002). In birds, clinical signs include colonic spasms, total paralysis, gasping, coughing, sneezing, rales, tremors, paralyzed wings and legs, twisted necks, and circling (Alders and Spradbrow, 2001; Alders *et al*., 2002).

Direct contact with the secretions of infected birds causes transmission, which mostly happens through eating (fecal/oral route) and inhalation. Feed, water, tools, spaces, people's clothes, boots, bags, and egg trays/crates are some other modes of transmission. The presence of feces helps the infectious agent to survive, and some of the strains may infect hatching chicks through eggs. Seldom can very virulent isolates spread, and there is no conclusive proof that flies play a part in mechanical transmission.

Respiratory fluids and discharges, diseased birds' droppings, and all carcasses' components are potential virus sources. The virus is shed during the incubation stage, the clinical stages, and for a brief time during convalescence. Wild birds and waterfowl may operate as reservoir hosts for lentogenic pathotypes; later, becoming pathogenic upon mutation and introduction in domestic poultry. It has been shown that some psittacine birds can occasionally shed the ND virus for over a year, which is linked to the introduction of this infection into poultry (Alders and Spradbrow, 2001; Alders *et al*., 2002).

The velogenic strain is widespread in Asia, the Middle East, and Africa and is endemic in parts of Mexico, Central America, and South America. It is also found in double-crested wild cormorants in the United States and Canada. While broad mesogenic pathotypes with an unique adaption to pigeons (such as pigeon paramyxovirus) do not appear to easily infect other fowl, lentogenic strains are distributed around the world (Alders and Spradbrow, 2001; Alders *et al*., 2002).

2.1.3 Control and prevention

ND is notifiable in many counties with no treatment available; however, there are two applicable broad prophylactic measures namely, biosecurity and vaccination.

2.1.3.1. Biosecurity measures

These are the recommended sanitary measures according to Alders and Spradbrow (2001); houses, feed, and water sources that are bird-proof, proper carcass disposal, controlling mice and insects in flocks, staying away from wild or feral birds as well as recently obtained domesticated poultry and pet birds with unclear health status, controlling human traffic; facility staff shouldn't interact with wild birds, and a policy requiring them to shower in special clothing should be considered, vehicle traffic regulation and thorough equipment and conveyance cleaning, it is advised to rear only one age group each farm ('all in-all out'); coops should be cleaned and disinfected in between, and during an outbreak, strict movement restrictions, the eradication of all sick and exposed birds, a twenty-one (21) day wait period before restocking, and thorough cleaning and disinfection of the area are all required (Alders *et al*., 2002).

2.1.3.2. Vaccination

The World Organization for Animal Health (WOAH) Terrestrial Manual 2012 provides a variety of vaccination strategies and references that consider the type of vaccine to be used, the immune and disease status of the birds to be vaccinated, the level of maternal immunity in young chickens, and the level of protection necessary in relation to any potential for field virus infection under local conditions. Live and/or oil emulsion vaccinations can significantly minimize poultry flock losses, but they cannot guarantee that virus spread through replication and shedding will not occur.

Live vaccinations tend to be more virulent and therefore more likely to have negative side effects the more immunogenic they are. Lentogenic vaccines (such as Hitchner-B1, La Sota, V4, NDW, I2, and F) and mesogenic vaccines (such as Roakin, Mukteswar, and Komarov) are two categories of conventional live virus vaccines; infections of these vaccinal viruses would be considered ND under the WOAH classification (World Organisation for Animal Health (2012)).

Live virus vaccines offer better seroconversion compared to inactivated ones and are administered to birds by incorporation in the drinking water, delivered as a coarse spray (aerosol), or by intranasal or conjunctival instillation; some mesogenic strains are given by wing-web intradermal inoculation. Fowl pox, vaccinia, pigeon pox, turkey herpesvirus, and avian cells that express the HN gene, the F gene, or both of the ND genes are examples of new recombinant vaccines (Alders and Spradbrow, 2001; Alders *et al*., 2002).

2.1.3.3. Control and prevention of ND in village indigenous chickens

The best way to prevent ND is through vaccination. Since the vaccines were produced in huge, heat-labile multi-dose vials that needed to be kept cool from the manufacturers until administration, commercial poultry producers with big, single-age flocks of chickens benefited most from their use for a long period. In contrast, huge multi-dose vials were inappropriate for village hens kept in small, multi-age, free-range flocks. Maintaining a cold chain in a village setting is challenging, and buying commercial vaccinations is expensive. ACIAR, Australia's Centre for International Agricultural Research, has funded initiatives that resulted in the creation of vaccines safe for village hens. These vaccines were chosen for their thermostability, eliminating the need for an ongoing cold chain (Alders and Spradbrow, 2001).

In order to achieve herd immunity, a minimum of 85% of flock should be vaccinated (Van Boven *et al*., 2008). Various authors have listed vaccines in use in many countries (Copland and Spradbrow, 1997; Alders and Spradbrow, 2001; Getabalew *et al*., 2019) and include: La Sota (live vaccine, thermolabile), Hitcher B1 (live vaccine, thermolabile), NEW/NEW COVER FOR ITA (inactivated vaccine, thermostable), NDV4-HR (live vaccine, thermostable), I-2 (live vaccine, thermostable), and Intervet South Africa Pty Ltd. has released the Clone LZ.58 (Nobilis ND Inkukhu) vaccine, a live, partially thermostable vaccine.

The first three immunizations must never be frozen and must be stored in the refrigerator at a temperature between 4°C and 8°C degrees. After the expiration date, vaccines shouldn't be used. Thermolabile, live vaccine vials should not be stored for use the following day after they have been opened vials should not be exposed to sunlight while receiving vaccines and should instead be kept in a cold box or wrapped in a moist towel. Although thermostable, the NDV4-HR and I-2 vaccines must be kept out of direct sunlight and as cool as possible to maintain their ability to work outside the cold chain (World Organisation for Animal Health (2012).

HB1, La Sota, ND Clone LZ.58, NDV4-HR, and I-2 are given orally or as eye drops. Moreover, the NDV4-HR and I-2 vaccinations can be taken orally after being blended with specific foods (care must be taken to ensure that the chosen food does not contain agents such as disinfectants that can inactivate the vaccine virus). Eye drops are the most effective method of delivery (World Organisation for Animal Health (2012).

Live vaccines are less expensive, more administrable (by eye drop, intranasal, spray, drinking water, oral, and injection), stimulate all forms of immunity, confer immunity differently depending on the route of administration (typically lasting no longer than four months), difficult to store (except for thermostable live vaccines, such as I-2), and are not harmful to the vaccine giver. In addition to what was given in the previous section, those that are inactivated must contain a significant amount of the virus that has been rendered inactive; only stimulate antibody-based immunity, confer immunity for about six months, be easier to store, and present a risk to the vaccine provider in the event of an accidental injection (Alders and Spradbrow, 2001).

After administration, immunity is conferred after one to two weeks. Strategic vaccination should be carried out at least one month before the expected start of a seasonal outbreak. If chickens are not revaccinated, immunity declines. It is preferable to immunize chickens with the eye drops at least three times every year. If oral approaches are employed, a booster dose should be administered two to four weeks after the initial immunization, followed by a booster shot every three months. Village chicken flocks can be vaccinated every three to four months to safeguard freshly hatched chicks (Komba *et al*., 2012). A virus that is antigenically like the disease-producing strains is present in both inactivated and live vaccinations. Every six months, inactivated vaccinations should be given in regions where outbreaks are likely. However, the expertise and energy of youngsters can be essential, especially in locations where chickens roost in trees, so plan vaccination campaigns to coincide with school breaks or weekends to recruit their help. Moreover, in the evenings or early mornings when the birds are in night confinements.

2.2 Significance of ND vaccination

2.2.1 Impact on flock sizes

Vaccination effectively lowers the morbidity and mortality of birds (Msoffe *et al*., 2001), growing flock sizes, food security, and moms' and children's consumption of chicken meat and eggs (Knueppel *et al.*, 2010).

2.2.2 The effect of the ND vaccine on income and child nutrition

Indigenous chickens are kept by majority among low- and middle-income countries of rural families such as Kenya with varied contributions; as sources of ASF, income generation, fallbacks in times of acute financial needs, female empowerment, and socio-cultural activities (Fallou, 2000). With the known financial constraints in these households, these birds are preferred because they require minimal inputs for managing diseases, feeding, and shelter. They are significant sources of high-quality protein and other micronutrients that are lacking in monotonous diets centered around maize, which is a typical meal in Siaya county, Kenya. These sources include eggs and meat from them (Alders and Spradbrow, 2001). Sales of chickens and their eggs provide a significant source of revenue for homes, which can be used to buy other food items, cover school costs, or obtain medical care (Thumbi *et al*., 2015). In many instances, women keep and take care of these birds and would often retain incomes from the chicken sales for household use (Mapiye *et al*., 2008). When compared to other livestock, this business is distinct and advantageous because it has inexpensive start-up and maintenance expenses, enjoys quick reproduction rates, and is simple to market. Because of this, they provide as dependable sources of income for many low-income people and a good source of animal-based goods (Akinola and Essien, 2011).

Because of the supplies of proteins, calcium, vitamins B and A, and iron found in meat, milk, and eggs, ASF consumption has been linked to a decreased occurrence of stunting (Allen and Dror, 2011; Krebs *et al.*, 2011).

Rural livestock keeping plays a critical role in livelihoods and human nutrition as forms of enhanced disposable incomes that can be committed to food purchase and as a direct source of ASF (Staal *et al*., 2009; Krebs *et al*., 2011; Mosites *et al*., 2015; Mosites *et al*., 2016a).

Early studies in Siaya county, Kenya showed a significant relationship between egg consumption and child height gain, as well as a relationship between increased chicken ownership and consumption of eggs and chicken meat. Additionally, some livestock diseases were linked to a lower reported frequency of consumption of ASF in a population where stunting and wasting in children under five years of age were, respectively, about 23.5% and 4.8% (Mosites *et al*., 2016a).

2.3 Childhood malnutrition

Establishing malnutrition problems in children mainly relies on two indices: stunting (height/ length for age) and wasting (weight/ age). Specifically, stunting is characterized as impaired growth and development brought on by inadequate nutrition often resulting in poor health outcomes, delayed mental development, and low economic gains; it is a major public health problem in developing countries (Onis and Branca, 2016). Despite numerous attempts to reduce its prevalence, 40% of children under the age of five in eastern Africa continue to bear the brunt of its consequences (Stewart *et al*., 2013).

The results will show whether increased production of native chicken improves livelihoods and nutrition in young children. They will also paint a picture of the infections that hamper the native poultry production system. This will be done by removing a restriction on a household's nutritional asset, reducing Newcastle disease through vaccination, and evaluating whether households would choose to give their young children diets rich in animal protein that increase consumption of native chicken. If it is demonstrated that this change in ASF (eggs and chicken meat) or enhanced income from chicken intervention promotes better child growth, this would be fresh proof, most notably in height for age but also for weight for height, parameters used to assess childhood stunting and wasting respectively, would be demonstrated.

The findings of previous studies show the potential to optimize the value of a typical family animal asset to lessen the unacceptable hig h incidence of childhood malnutrition in these communities given the high frequency of juvenile grow th failure, especially stunting, in rural Africa. If production outcomes and welfare of household will be proven to have been enhanced, the positive results will guide development of public health and agricultural policies associated with ND vaccine usage, poultry productivity and pursuant health, and livelihoods improvements.

CHAPTER THREE: IMPACT OF THE NEWCASTLE DISEASE VACCINATION ON THE SURVIVAL AND PRODUCTIVITY OF INDIGENOUS CHICKEN

3.1 Introduction

The significance of poverty and undernutrition for reaching the Sustainable Development Goals of the United Nations highlights the magnitude of these issues on a global scale. Undernutrition includes wasting and stunting (Development Initiatives, 2018). Rural households are disproportionately affected by poverty and malnutrition in South America, Asia, and Africa (FAO, IFAD, UNICEF, 2019). According to the Food and Agriculture Organization, there are 500 million smallholder farms worldwide that support 2-2.5 billion people, or a significant portion of the population, who live on less than $2 a day and provide all or part of their household welfare (Lowder *et al*., 2016; UNDP, 2020). Food and income are primarily produced by smallholder farms through mixed crop farming, perhaps in conjunction with small-scale livestock husbandry (Wong *et al*., 2017). Smallholder households most frequently own chickens, which present an opportunity to provide the home with eggs and meat as well as maybe create cash through local sales (Kryger *et al*., 2010; Kingori *et al*., 2010). Also, domestic chicken flocks are a significant source of food and income that are typically controlled by women in rural households and can be a reflection of their priorities for the welfare of their families (Valdivia, 2001; Kristjanson *et al*., 2012; Wong *et al*., 2017).

Despite the fact that chickens are widely regarded as the main animal asset of smallholder farmers, low productivity and small flock sizes prevent potential gains from being maximized (Kryger *et al*., 2010; Campbell *et al*., 2018).

Crucially, the low flock numbers appear to reflect a high level of involuntary losses owing to mortality and predation rather than an economic decision to maximize labor input while maximizing benefits from household consumption or sale (Otiang *et al*., 2020). The mean flock size was found to be roughly 10 and highly steady over time in a recent 4-year longitudinal study of 1,908 families in Siaya county, Kenya, reflecting a balance of new chicks hatched on premises and losses, 60% of which were due to mortality (Otiang *et al*., 2020). Smallholder flocks received very little input. Ninety eight percent (98%) of households said that during the day, chickens forage for all or most of their food, while 93% keep chickens inside the home at night (Wong *et al*., 2017; Otiang *et al*., 2020). Vaccination, supplemental nutrition, and treatment of endo- and ecto-parasites that would be expected to reduce morbidity and mortality were uncommon (Dwinger and Unger, 2006).

ND affects poultry and is widely spread throughout the world (Alexander, 2000; FAO, 2014). While high levels of biosecurity and medical prophylaxis (vaccine) are frequently employed to avoid ND outbreaks in commercial poultry, smallholder households' reliance on free range scavenging for chickens makes it easier for flocks to spread the disease to one another and among themselves (Alders *et al*., 2002). As a result, ND is frequently cited as the biggest obstacle to effective smallholder poultry productivity in Africa (Kryger *et al*., 2010). While vaccination is quite effective in controlled environments, its effectiveness in smallholder settings may be far more varied depending on the flock's overall health and age distribution (Campbell *et al*., 2018).

A randomized controlled trial in 537 households, in which all chickens in 254 households received the ND vaccine every three months while chickens in 283 households served as the unvaccinated controls, was carried out to ascertain whether routinely scheduled ND vaccination led to increases in flock sizes over time.

In conclusion, this chapter examines changes in monthly flock census over an 18-month period and discusses the findings considering enhancing flock productivity and household well-being.

3.2 Materials and Methods

3.2.1 Ethical approval

The Animal Care and Use and Ethics Committees of the KEMRI gave their approval for the study (SSC Protocol no. 3159 Appendices 6 and 7) and by the University of Nairobi’s Faculty of

Veterinary Medicine Biosafety, Animal Use and Ethics Committee (BAUEC) (Appendix 8) to determine whether routine ND immunization leads to gradual increases in flock sizes.

Informed consent included information about the study's goals, potential household benefits, potential hazards to participants, and measures to mitigate those risks, as well as contact details for the medical and veterinary staff working on the project and the ethical review bodies.

There was no communitywide presentation or interdependency among families that would have put pressure on a specific household to participate; participation was completely voluntary, and the decision was made at the household level. The household head gave his or her approval both orally and in writing (in Luo, the local language), and a copy of the written forms were saved (Appendix 1).

3.2.2 Study site

The study was conducted in Rarieda sub-county of Siaya county, Kenya. A health and demographic surveillance system (HDSS) operated by KEMRI, and the US Centers for Disease Control and Prevention provided services at this location (CDC). Within a 5.5 km radius of St. Elizabeth Lwak Mission Hospital, it involved 10 villages with roughly 1,908 families participating in both Population Based Infectious Disease Surveillance (PBIDS) and Population Based Animal Syndromic Surveillance (PBASS) (Figure 1).

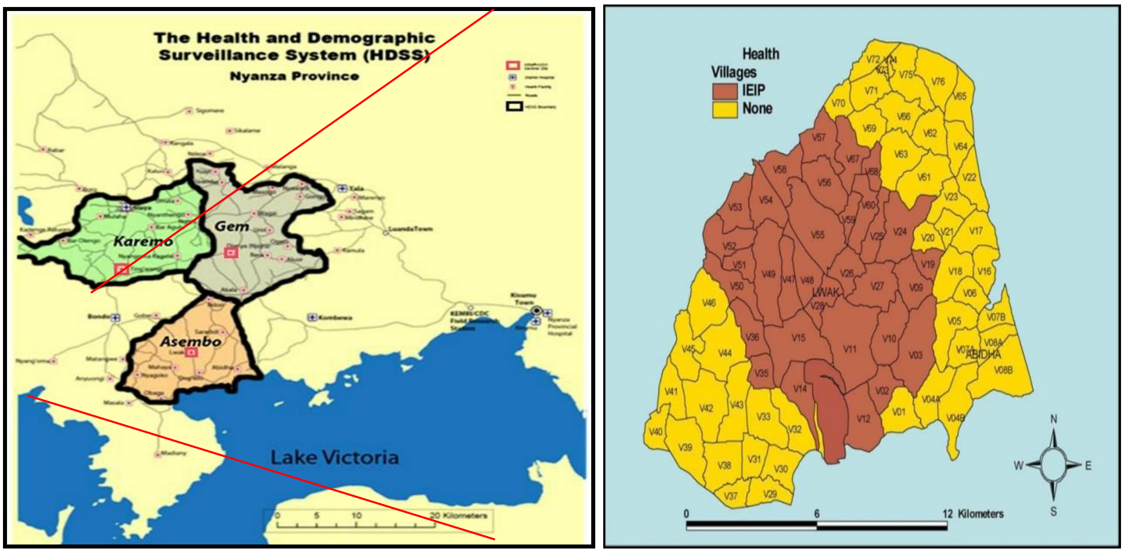

Figure 1: Study Area Map. Locations of villages in Asembo area in Siaya county, Kenya where the study was conducted (Feikin *et al*., 2010).

Several health-related research projects at the individual, household, compound, and community levels had used this site as their starting point.

At the entryway of each HDSS home was a distinctive identifying number. The area was served by St. Elizabeth Lwak Mission Hospital being the primary referral hospital for PBIDS (Feikin *et al*., 2010), PBASS, and Socioeconomic Surveillance (SES) studies were also based there (Thumbi *et al.*, 2015). The population consisted largely of sedentary smallholder farmers and fishermen of the Luo ethnic group. At the study period, Siaya county's population was approximately 1,027,795 people consisting of 488,077 males and 539,718 females (County Government of Siaya, 2018). Demand for health care is high in the area; it is also known to be one of the most impoverished areas in Kenya; where 60–70% of the population is considered to be poor (Central Bureau of Statistics (CBS), Ministry of Health (MoH) [Kenya], 2004). The region experiences ongoing, severe malaria transmission (Tako *et al*., 2004) and a high prevalence of Human Immunodeficiency Virus (HIV) infection i.e. in 2003, >10% men and >20% women aged 13-34 years (Amornkul *et al*., 2009).

The region is also prone to other infectious diseases. As a result, the region's mortality rates reflect the prevalence of infectious diseases there (Bigogo *et al*., 2010).

With an average flock size of ten chickens, the majority of homes in the area have livestock, and over half of all animal deaths reported in participating houses involved chickens (93% 95% of households) (Thumbi *et al*., 2015; Otiang *et al*., 2020). Both temperature and rainfall are high during most of the year, with wet season in March, April, and May for long rains and September, October and November as short rains while dry season in December, January, and February as hot dry and June, July, and August for cool dry (Apopo *et al*., 2020).

According to the annual development plan (2019-2020), there are an estimated one million birds in the county, including broilers, layers, and free-range birds. Other poultry species, which make up 3%

of the total and are growing more significant, include ducks, turkeys, pigeons, ostriches, guinea fowls, and quails. About 1,900 metric tonnes of poultry meat worth 760 million Kenyan shillings and 716,000 trays of eggs worth 214 million Kenyan shillings are produced annually in the county. Both poultry meat and eggs are in short supply in the area, therefore imports help to make up the difference (County Government of Siaya, 2018).

3.2.3 Study design

A total of 537 household participated in the field study, which was an 18-month randomized control trial (intervention n=254, control n=283) enrolled and followed upon meeting an inclusion criterion of chicken keeping, a child below five years, and/or a pregnant mother, and grouped into two in a two-level experiment, the first being on chickens and the latter on children and pregnant mothers. To determine the appropriate sample size, it was assumed that ND vaccination would result in a 10% decrease in flock mortality, with a likelihood of type 1 error set at 0.05 and an 80% detection power. Based on an average flock size of 10 hens per home (range: 4 to 60), this took into consideration different cluster sizes. Of the 667 eligible households, 86 were excluded based on the previously identified child being >5 years of age by the enrolment date, the household no longer kept chickens, the household declined participation, or the household did not plan on consistently remaining in the region (Figure 2). The remaining 581 households were randomly allocated by computer using a unique numerical identifier to either the treatment group (vaccination of all household chickens with ND vaccine and parasite control at project initiation and quarterly thereafter) or the control group (parasite control on the same schedule but no ND vaccination). Allocation was uneven between the two groups (treatment n=276; control n=305) and accounted for an additional 10% loss of control households relative to treatment households during the trial in the event of high mortality in unvaccinated flocks. However, vaccination uptake was low on the study site at below 2% (Otiang *et al*., 2020). The intervention (quarterly vaccination and parasite control) was delivered to 254 treatment households with 283 control households (parasite control only) after a loss to follow up of up to n=130 households and within it, n=44 households from the those already profiled for chicken experiment. The vaccinated group routinely received immunization of two drops of ND AVIVAX I-2 thermostable vaccine intranasal or intraocular depending on chicken's age at recruitment and every three months thereafter. Chickens in the control group were not vaccinated. Ecto-parasiticide of the group carbaryl (Sevin® powder) was dusted on the bodies and oral deworming using piperazine citrate (Ascarex-D®) given in drinking water to all the chicken at recruitment and then every three months. Vaccinations and data collection were carried out by blinded qualified animal health technicians, but vaccinations were done by one unblinded animal health technician to minimize bias this being a randomized trial. Each vial was reconstituted with a corresponding sterile diluent, kept in a cool box at between 4°C to 8°C and aseptic precautions taken at reconstitution and withdrawal before being administered using a pipette. A day to vaccination, the participants in the households due would be asked not to release the chickens from their night confines as the vaccinator would visit the proceeding morning. Vaccinations would be done from early morning to noon. The vaccinator was also the one who would dust the birds and offer the dewormers for administration. These activities were repeated six times every three months to the end of the study. Some households would however miss out due to lack of ownership of chicken at monthly visits but would still be followed up in the subsequent ones in case they acquired any along the way.

Participating households were provided with cards bearing a toll-free phone number catered for by the project through service provider. They would call-in in cases of sicknesses and deaths, besides monthly active data collections. A response would then be mounted to collect data on disease history, clinical signs, and post-mortem findings which would then be relied-upon to arriving at a tentative diagnosis for veterinary advice on needful intervention.

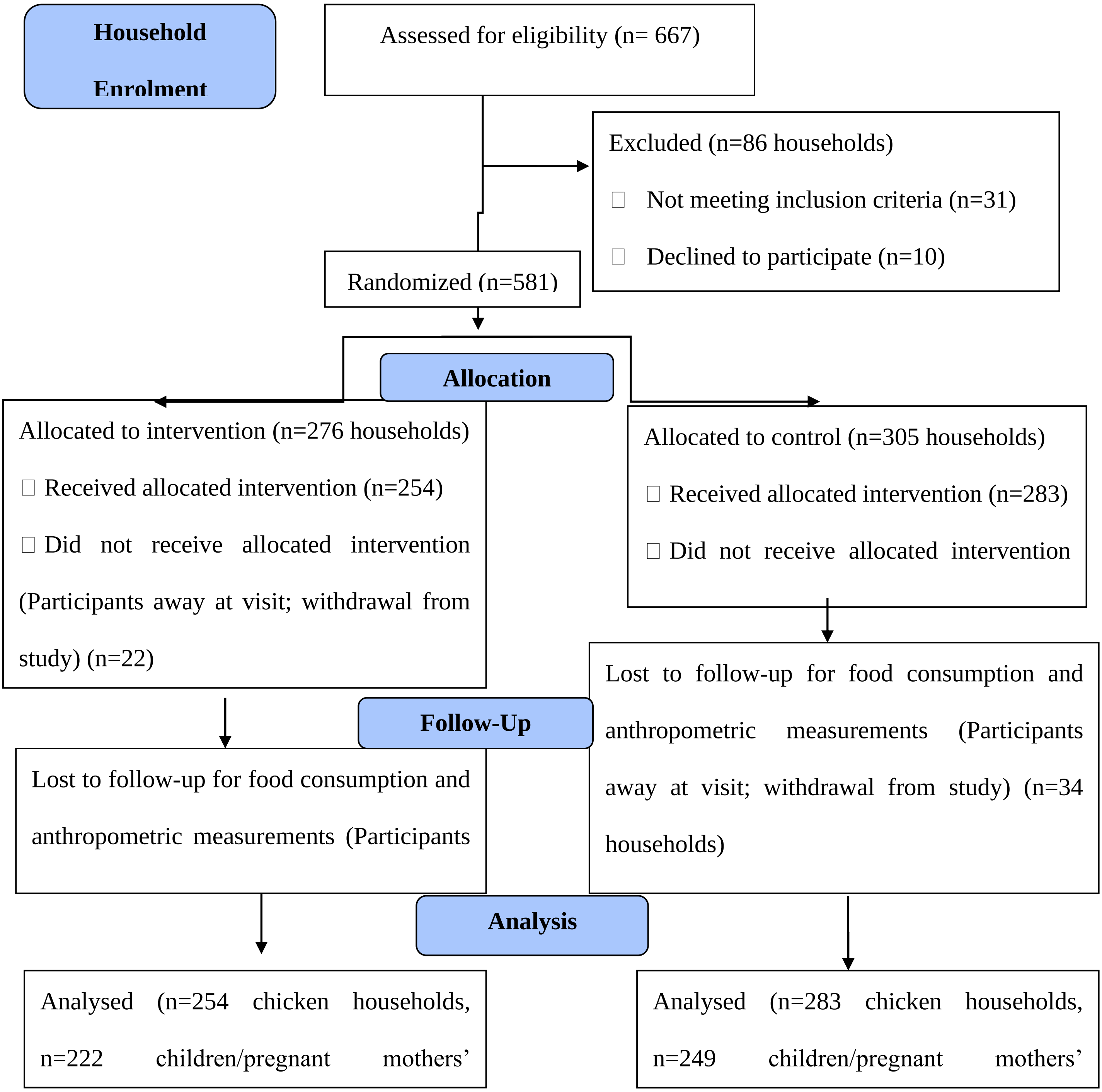

Figure 2: Household allocation: Chicken and child study enrolment, allocation, and follow-ups in Siaya county, Kenya

3.2.4 Data Collection

Trained enumerators and I counted the flock's size and age distribution at each monthly visit. Also, a semi-structured questionnaire was used to conduct a local language interview with the person in charge of the flock or the head of the household to gather recall information about flock growth and decline over the previous three months. The information was gathered using CommCare, a mobile application, and was kept up to date using a Microsoft Access database®.

3.2.5 Data analysis

STATA Version 16.1 was used to clean and analyze the data (Stata, 2019) to assess improvement in local chicken productivity following Newcastle disease intervention by determining flock size changes in the control and treatment groups.

3.3 Results

3.3.1 Study population

Most frequently, the people in charge of the hens (89%) or the compound's leader (11%), responded to the questionnaire, 93% of the people in charge of overseeing the flock were female.

3.3.2 Longitudinal monthly flock census

The mean flock sizes on visit 1, at the time of the first vaccination but prior to any possible effects of vaccination, were 11.63±0.70 for the 254 households in the vaccination arm of the study, and 11.13±0.67 for the 283 control households. Enrolled households-maintained flocks throughout the study period with less than 2% of visits recording no chickens at the monthly census. Over the 18 monthly visits, the flock sizes increased but the total flock sizes were significantly greater in the vaccinated households: there was a cumulative mean of 13.06±0.29 chickens in the vaccinated households versus 12.06±0.20 in the control households (p=0.0026), with the increases occurring across all age categories (Figure 3): the mean number of chicks in vaccinated households was 6.59±0.20 as compared to 6.20±0.13 in controls (p=0.06), mean number of growers was 3.84±0.08 versus 3.63±0.09 (p=0.09), and mean number of adults was 3.32±0.19 as compared to 2.93±0.05 (p=0.03). The increase was sustained throughout the study, whether analyzed by the best-fit over the 18 visits (Figure 4) or by a best-fit tethered to the flock size at the visit 1 and then a best-fit determined (Figure 5).

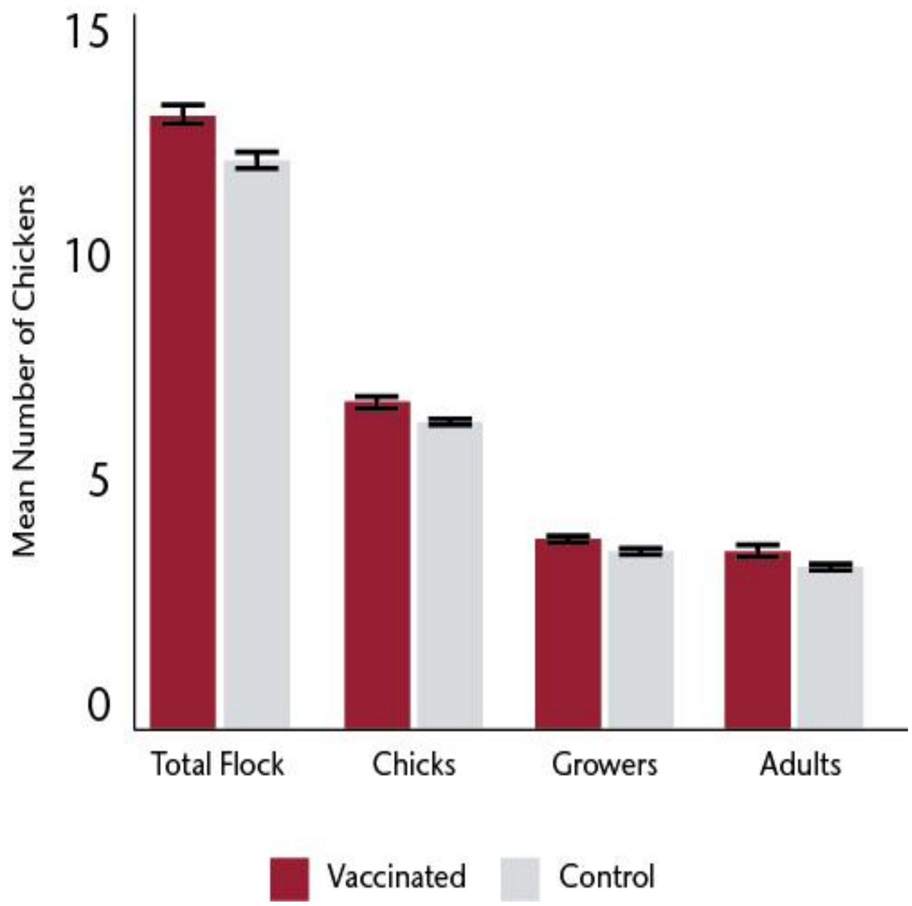

Figure 3: Mean flock size at monthly census over 18 months

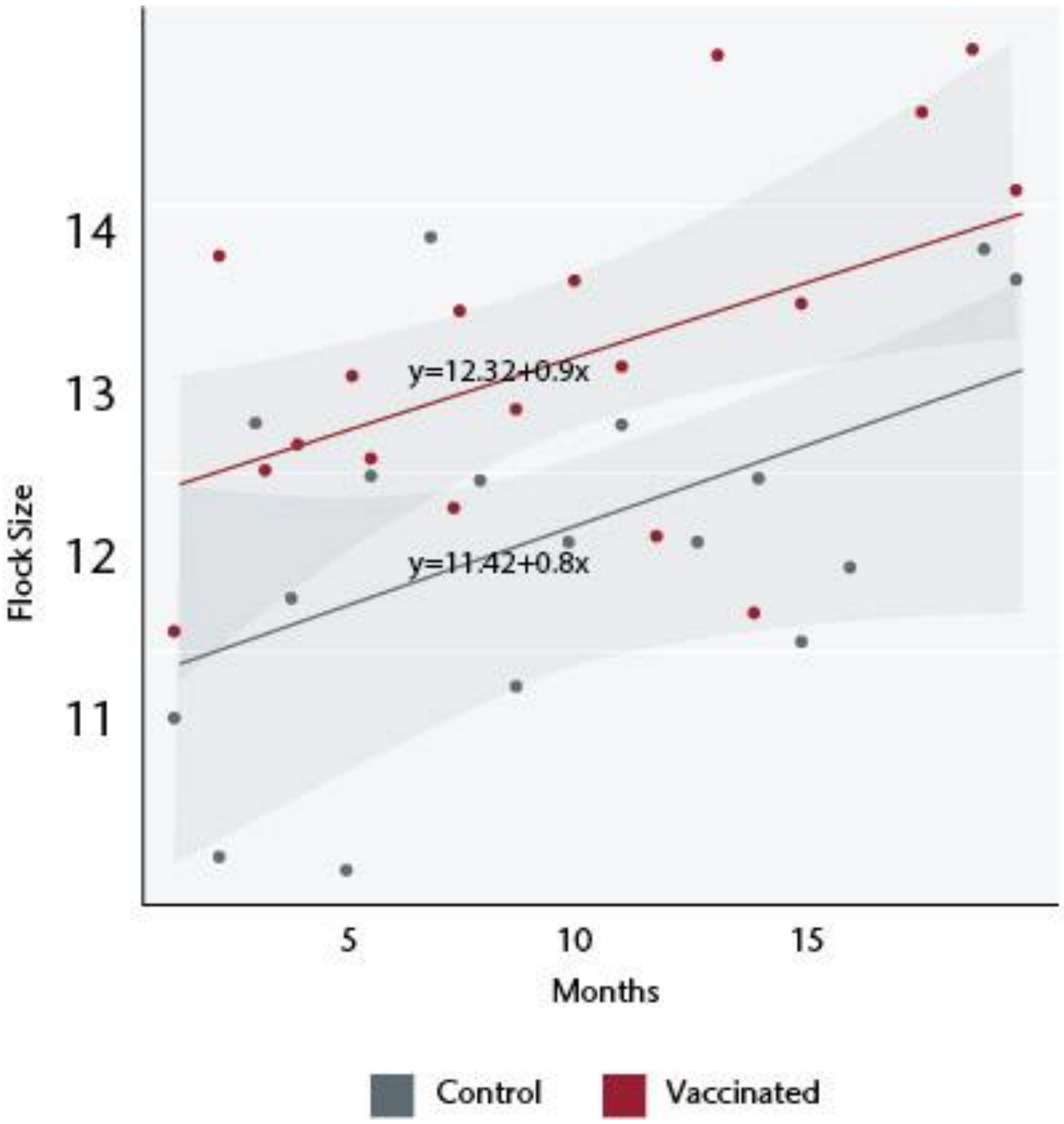

Figure 4: Flock size dynamics over time (best fit of all data points)

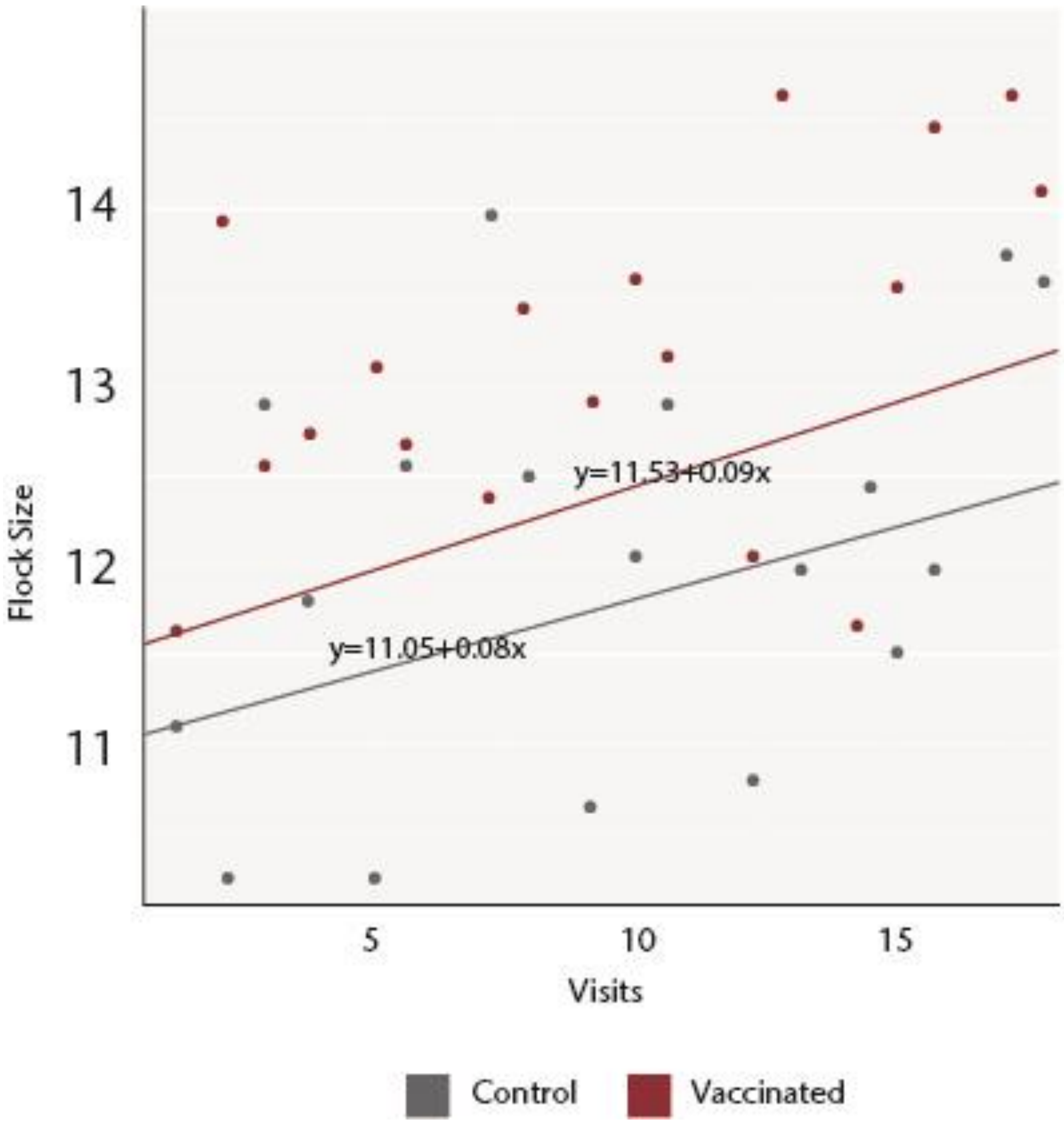

Figure 5: Flock size dynamics over time (best fit of data point tethered to initial flock size)

3.3.3 Household reported gains and losses

During each monthly visit, the household respondent was asked to self-report gains and losses during the prior month. Households that received vaccination reported gains of 4.50±0.12 chickens per month as compared to 4.15±0.11 in the non-vaccinated control households ($p=0.03$). Vaccination households reported total decreases of 2.50±0.09 chickens per month versus 2.43±0.09 in the control households ($p=0.56$). Reported voluntary decreases in flock size, reflecting household decision-making for sales or household consumption, were marginally greater in vaccinated households, 1.10±0.05, as compared to 1.03±0.04 in control households ($p=0.19$), representing 44% and 42% of the monthly decreases in vaccinated and control households, respectively (Figure 6). Involuntary losses, including mortality and both unspecified loss and loss to predation, were reported to be marginally higher in control households, 1.4±0.08, as compared to 1.3±0.08 in vaccinated households (p=0.39), with mortality representing the greatest reported source of loss in both groups (Figure 6). Majority (85%) of naturally dying chicken were buried or thrown in pit latrines with just about 10% eaten by family members.

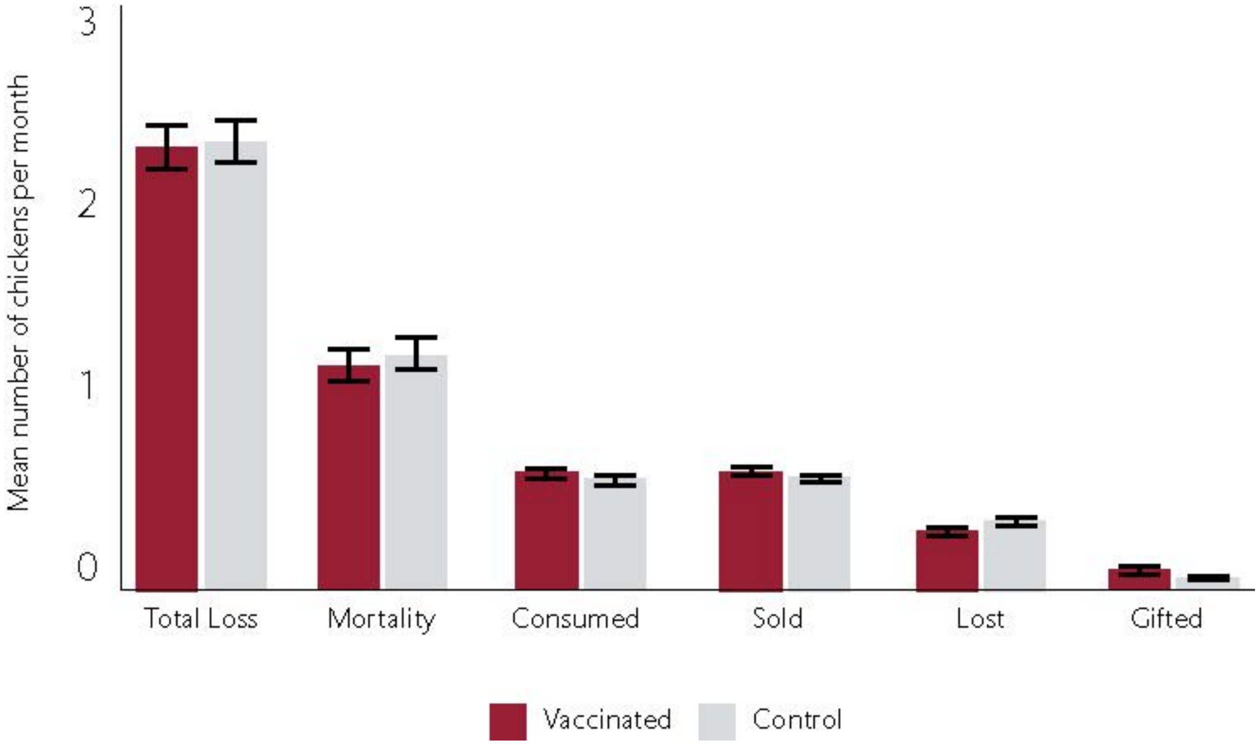

Figure 6: Sources of reported monthly off-take in chickens.

3.3.4 Occurrence of other infections

The study evaluated the clinical presentation at sickness and prior clinical signs and post-mortem findings at death, grouped them by syndromes and made tentative diagnoses to help in interventions for disease episodes at visit response sessions. Of the 805 reported clinical episodes investigated, distributions for responses were 34.9% for sicknesses, 30.6% for deaths and 34.5% for both sicknesses and deaths. To further characterize presenting clinical signs, categorization was done per body system affected as syndromes. Respiratory syndrome presenting with nasal and oral discharges, coughing and difficulty in breathing accounted for 59.6% of the occurring sickness events, neurologic syndrome as stargazing, staggering, torticollis, paralysis, and depression at 59.4%, digestive syndrome primarily diarrhea at 39%, integumentary syndrome as pox lesion at 16.1%, head swelling, loss of feathers and swelling of the foot of the feet at 3.9%. Other clinical signs of general involvement principally loss of appetite was observed in 36.6% of the cases. Post-mortems were investigated for 38 episodes with digestive system lesions as presence of mucus and hemorrhages in the proventriculus at 79%, other hemorrhages at esophageal, caecal, cloacal, and crop were at 50% and intestinal worms at 52.6%. Pox lesions were seen in 13.2% of the episodes with incidental ectoparasites at 29%. Inflammation of bursa of *fabricius* was observed in 18.4% and thigh muscles hemorrhages at 10.5%. Tentative diagnoses pointed out Fowl typhoid at 52.6%, ND at 50%, Infectious bursal disease at 47.4%, and pullorum disease at 13.2%. Biological specimens from investigated cases were collected and archived and would aid in confirming these aetiologies when resources are made available.

3.4. Discussion

The use of a relatively large household randomized controlled trial allowed determination of the impact of ND vaccination on flock size. Routine ND vaccination of all chickens resulted in a mean gain of one chicken per household. While this gain seems modest, it represents an 8-10% increase in flock size from vaccination against a single viral pathogen. Importantly, the increase was sustained throughout the 18-month study. This suggests the opportunity for increased gains over time and an accumulating impact of routine ND vaccination. If these gains were utilized for either household nutrition or income from sale there would be measurable benefits to the family (Mosites *et al.*, 2016a;b). Consumption of either eggs or chicken meat have been shown to reduce childhood stunting, which remains at a high level in the study region and throughout much of rural sub-Saharan Africa (Campbell *et al.*, 2019). Similarly, routine vaccination has potential to increase household income. In a recent study of smallholder households in rural Tanzania, the market price for vaccine to inoculate 10 chickens was US$1.20 while the local market price for an adult chicken was US$3.12.

Notably, respondents in that study were willing to pay twice the market price for vaccine, reflecting that households valued vaccination and perceived a favorable return on investment (Marangon and Busani, 2007). As 93% of the individuals in this study that managed the flocks were women, and women have been shown to devote a much greater proportion of income into family nutrition and health care, this relatively modest increase in income can have a significant impact on familial well-being (Valdivia, 2001; Kristjanson *et al*., 2012; Wong *et al*., 2017).

The current study allowed an estimation of the preventable fraction of mortality due to ND. Vaccination was carried out by qualified animal health technicians supervised by a licensed veterinarian and records were kept on the storage and delivery of the ND AVIVAX I-2 vaccines. While ND AVIVAX I-2 vaccination has been shown in numerous experimental and field studies to be highly effective (Marangon and Busani, 2007; Harrison & Alders, 2010; Komba *et al*., 2012; Mosites *et al*., 2016a; b), the preventable fraction of mortality due to a single vaccine reflects the overall causes of mortality and varies depending on the specifics of poultry management at the household level (Alders *et al*., 2002). The most reliable measure of vaccine impact from this study would be 8-10% increase in total flock size at monthly census. In contrast, the self-reported mortality by the household respondents indicated no significant difference in mortality between the vaccinated and control flocks (Figure 6). This discrepancy between independent census data and self-reported data has been previously observed among households in the study region (Otiang *et al*., 2020). In the prior study, individuals consistently overestimated gains and underestimated losses relative to actual census data (Otiang *et al*., 2020). This pattern is observed in the current study: as an example, the self-reported total monthly gains in vaccinated households were reported as 4.5 with overall monthly decreases of 2.5, inconsistent with census data that indicates a smaller monthly increase. Recall bias, representing systemic errors in remembering past events, and social desirability bias are two possible explanations for the discrepancy (Nega *et al*., 2012). Households in the study region did not maintain written records on flock size, gains, and losses, thus losses that occurred earlier in the month may have been discounted relative to gains that were still represented in the flock. This may be especially true for young chicks, which have a high mortality rate from ND as well as other infectious and non-infectious causes (Alders *et al*., 2002; Nyaga, 2007; Lesnoff, 2009; Otiang *et al*., 2020). Social desirability bias, the tendency for survey respondents to answer questions in a way that will be viewed favorably by others, may also have an impact as the household may want to be seen by the interviewers and animal health team as being a responsible member of the community and thus overstate gains and understate involuntary losses, including mortality (Olwande *et al*., 2010). Notably, there were sustained gains in flock size in households that received vaccination and parasiticidal treatment and the control households that received only parasite control. While a control group with no treatment was not included (as participation required time commitment by the respondents), comparison with both the flock sizes at enrolment and the historical mean flock size of 10 in this study site (Thumbi *et al*., 2015; Otiang *et al*., 2020) suggest that parasiticidal treatment had a significant effect alone, which was further enhanced by ND vaccination. This is consistent with prior studies showing the impact of combined interventions and emphasizes that a comprehensive approach to improved poultry management at the smallholder level is needed (Kryger *et al*., 2010). Integrating supplemental nutrition would highly likely increase the efficacy of vaccination as well as maximize benefits from parasite control. The lack of improved management does not necessarily translate to a lack of knowledge regarding the importance of vaccination as indicated by willingness to pay studies in which respondents were willing to pay more than the actual cost of ND vaccine (Campbell *et al*., 2019). Rather, the primary barrier to improved management appears to be at the level of service delivery. At a household level the incentives for effective delivery of more comprehensive poultry health, and husbandry services are too small for commercial investment. However, at a community level this may provide a larger integrated market that would attract commercial engagement, especially if incentivized by government support for rural communities (Kryger *et al*., 2010).

The study site having enjoyed a pool of qualified professionals for this intervention who would double up to offer free technical insights on animal care and husbandry, there seems to be a stronger contribution as opposed to having vaccinators with lack of animal health related backgrounds, as supported by Fisher (1993).

Similar clinical signs were observed in chicken sicknesses and in mortalities, indicating that most cases occurred as a result of similar etiologies as previously observed by Msoffe *et al*. (2010). The use of clinical signs, history, post-mortem findings, and alternatively applying syndromic evaluation to arrive at tentative diagnoses, has been applied in resource-limited settings to assist rural farmers with animal disease interventions when ideal situation of laboratory confirmation is lacking (Abdisa and Tagesu, 2017), though not accurate, it's a handy tool considering the requirements of a laboratory in the countryside. Based on the use of the tentative tool applied; Fowl typhoid was the most prevalent infection characterized by digestive syndrome followed by ND with outstanding digestive, nervous and respiratory syndromes, this is supported by Msoffe *et al*. (2010) who list these three syndromes as the major associates of the disease in Tanzania. Infectious bursal disease with musculoskeletal lesions, pullorum disease with digestive syndrome, and Fowl pox with nodular lesions of cutaneous form followed in that order. These five conditions accounted for most infectious conditions that caused mortality in rural free-range chicken in the study area. This supports work by other researchers (Okitoi *et al*., 2006; Olwande *et al*., 2010; Mutinda *et al*., 2015; Msoffe *et al*., 2010; Wahome *et al*., 2018). Tentatively, five main infections were incriminated to have been the main culprits ranked in order of prevalence as: Fowl typhoid, ND, Infectious bursal disease, pullorum, and Fowl pox. In a previous study within the study site, only 12% of households reported receiving some form of treatment, vaccination, or de-worming for their chickens, in a subset of 533 households surveyed in which 5.65% reported veterinary service, chicken vaccination was at 1.35%, ectoparasite control at 1.26%, and de-worming at 0.42% (Otiang *et al*., 2020). Considering that this system is majorly extensive, characterized by free mixing of birds during scavenging, and exposure to harsh environmental conditions thereby increased likelihood of disease infection, and transmission worsening mortalities, with possibility occurrences of mixed infections across all age ranges (Olwande *et al*., 2010). Moreover, animal disease events have shown climatic variations as extreme weather events can directly or indirectly affect transmission patterns leading to seasonal distributions (Baylis and Risley, 2012); this analysis indeed confirmed this occurrence as more mortalities were recorded in drier periods of December to February than rainy ones in March to May as exhibited by previous studies in Kenya (Njagi *et al.*, 2010; Apopo *et al*., 2020).

3.5 Conclusions

Comparing households where the flock had only parasiticidal therapy to those where the flock received quarterly ND vaccine plus treatment, it was found that the flock size increased by a mean of one bird per family. There was a large benefit to vaccination in terms of flock size, even if the results indicate that the preventable fraction of mortality owing to ND is relatively minor relative to all-causes of mortality in smallholder families. A comprehensive strategy to improve flock health and increase household benefits of production in the smallholder setting is supported by comparisons between study households' current flock sizes and previous flock sizes, which show a more significant benefit from the combined vaccination and parasiticidal treatment.

CHAPTER FOUR: CHILD GROWTH AND DEVELOPMENT: NUTRITIONAL AND SOCIOECONOMIC FACTORS

4.1 Introduction

Stunting, wasting, and underweight in children under five continue to have a significant negative impact on how people, families, communities, and entire countries develop (Kinyoki *et al*., 2020).

To achieve the World Health Organization's Global Nutrition Goals of a 40% reduction in stunting and a reduction in wasting to less than 5% by 2025, there must be significant decrease in child growth failure (Onis *et al*., 2013; WHO, 2018). Similarly, these reductions are necessary to achieve the Sustainable Development Goals of the United Nations, as 12 of the individual goals are related to nutrition (Grosso *et al*., 2020).

In rural Siaya county, Kenya, growth failure, particularly stunting and wasting, continues to be a significant health and development concern (Bloss *et al*., 2004; Mosites *et al*., 2016a; b). In Siaya County, a cross-sectional survey of 597 households in 2014 found that 4.8% and 23.5% of children under five were wasted and stunted respectively. This was determined by analyzing the height-for-age Z scores (HAZ) for stunting and the weight-for-height Z scores (WHZ) for wasting (Mosites *et al*., 2016a). Although most of these households are classed as having very little or no off-farm income and living in poverty or extreme poverty, many of these households have resources that can be improved on the farm to enhance nutrition and growth outcomes (County Government of Siaya, 2018).

The average flock size is small (roughly 10, of which only half are potential sources of eggs or meat, and the remaining are young chicks), and only 16% of children over the age of six months were reported to have consumed eggs in the three days prior to the study survey, despite the fact that eating eggs has been shown to increase childhood growth by a mean of 5% (Mosites *et al*., 2016b; Thumbi *et al*., 2015). Mortality was found to be the main factor limiting flock size in a follow-up longitudinal census of chickens and decision-making in 1,908 families within the same community. In contrast to voluntary household decisions to sell, eat, or give away chickens to meet demands or maintain a desired flock size based on minimizing the cost of household labor or other management resources, it accounted for 60% of all chicken losses (Otiang *et al*., 2020). Because so few chicks survived to maturity and productivity due to the high mortality rate, flock growth was restricted in terms of both size and composition.

Interventions to ameliorate this restriction of involuntary loss brought on by ND disease were predicted to result in greater protein consumption by children and, ideally, better growth outcomes.
First, it was investigated in a two-arm randomized controlled study if vaccination against ND, which is regarded as the most common infectious cause of mortality in free-range scavenging chickens globally, was effective (Alders *et al*., 2002; Guèye, 2005; Njagi *et al*., 2010), would result in increased flock size as hypothesized in the proposal. Quarterly vaccination over an 18-month period resulted in an increase in average flock size of 11.63±0.70 chickens at enrolment to 13.06±0.29 chickens, a significantly greater increase (p=0.0026) as compared to the unvaccinated arm of the trial. Based on this study, analysis to whether ND vaccination translated to increased consumption of ASF rich in protein and micronutrients relative to a high carbohydrate, low protein, grain-based diet and whether an increase in flock size influenced income earnings and other food stuff purchase ability were done. Furthermore, determination to whether a shift in consumption affected childhood growth was made.

4.2 Materials and methods

4.2.1 Trial design and participant selection

The longitudinal household randomized controlled trial was conducted in Rarieda sub-county of Siaya county in Siaya County, Kenya within a health and demographic surveillance system (HDSS) site run by the KEMRI and the United States Centers for Disease Control and Prevention (CDC) (Feikin *et al*., 2010) (Figure 1).   These households have high poverty levels and food insecurity with agriculture being the main source of livelihood, contributing about 60% of the household income and providing over 60% of all employment opportunities (Siaya County, 2018). Ambient temperature is high during most of the year, with a long wet season between March and May, and a shorter wet season between September and November (Ministry of Agriculture, Livestock and Fisheries (MoALF, 2016)). HDSS data from December 2016 (six months prior to the study initiation) indicated 667 potentially eligible households meeting the criteria of; i) chicken ownership; ii) a child under the age of 5 years or a pregnant mother; and iii) location within a 5.5 km radius of St. Elizabeth Lwak Mission Hospital, which could provide nutritional support for children identified as suffering from acute malnutrition. The age criterion for participation ensured that children would be less than five years of age at the end of the study. Of the 667 eligible households for both experiments, 86 were excluded based on the previously identified child was 5 years of age by the enrolment date, the household no longer kept chickens, the household declined participation, or the household did not plan on consistently remaining in the region (Figure 2). The remaining 581 households were randomly allocated by computer using a unique numerical identifier to either the treatment group (vaccination of all household chickens with ND vaccine and parasite control at project initiation and quarterly thereafter) or the control group (parasite control on the same schedule but no vaccination); details of the intervention are provided in the next paragraph. Allocation was uneven between the two groups (treatment n=276; control n=305) as we accounted for an additional 10% loss of control households relative to treatment households during the trial in the event of high mortality in unvaccinated flocks. The chicken intervention (quarterly vaccination and parasite control) was delivered to 254 treatment households with 283 control households (parasite control only) with 44 households getting dropped off following failure to meet various inclusion criteria thresholds at actual enrolment; food consumption data and anthropometric measurements were obtained from 222 treatment households and 249 control households (Figure 2) after a further 110 households failed to meet the criteria of having a child below 5 years and/or a pregnant mother. For children's experiment, data was analyzed from 471 households.

4.2.2 Newcastle disease vaccination

Specific to chicken intervention, animal health technicians delivered the intervention, independent of the food consumption and anthropometric data collection. All chickens in treatment households were vaccinated with two drops of ND AVIVAX I-2 thermostable vaccine ($10^{9.7}$ egg infectious doses/ml) intranasal or intraocular (depending on chicken's age at recruitment) and every three months thereafter. AVIVAX I-2 is a freeze-dried live attenuated ND vaccine prepared from the La Sota strain and manufactured by the Kenya Veterinary Vaccines Production Institute (KEVEVAPI).

4.2.3 Data collection

Assessments were conducted at the time of enrolment as a baseline and then quarterly for 18 months to collect anthropometric data for mothers and children, qualitative and quantitative dietary intake, and socioeconomic data. Child growth was measured using a Shorrboard® for length (<2 years) and height (≥2 years) and mother/child standing scale for weight. Mid-upper arm circumference (MUAC) was assessed using three standardized colored tapes: red (<115mm), indicating severe acute malnutrition, which triggered referral to a program that provided vitamin A and fortified maize flour and nutritional counselling; yellow (115-124mm), indicating moderately acute malnutrition, which triggered nutritional counselling; and green (>125mm), considered healthy growth and the caregiver was encouraged to continue with health care and feeding. For dietary assessment, caregivers were requested to recall the type of food, quantity, and the number of times the child was fed each type of the food in the three days prior to the interview. The interviewers asked about each specific food in the questionnaire (Appendix 4) and provided a standardized set of containers to assist in estimating the quantity of each food item. Socio-economic data included the mother's age and level of education, number of family members, household income consisting of both on-farm and off-farm earnings where Kenya shillings were converted to USA dollars using 2016 year's average as an exchange rate reference (1 USD = 101.50 Shillings) and discounted at an annual rate of 3.5% (Earnshaw and Lewis, 2008) (Appendix 5). All data were collected by community health interviewers in the local language (Luo), entered onto an electronic data capture tool, downloaded, and stored in a Microsoft Access database®. All datasets underwent validation and consistency checks to identify and resolve errors before they were merged using unique household identifiers on each of the participating households.

4.2.4. Allocation of households and data collectors in experimental groups

Households were not notified of which group they belonged to but were informed (oral and written) that their chickens would receive treatment to prevent disease. However, as household members observed a designated animal health technician conducting the procedures and may have been familiar with ND vaccination (although the vaccine had not previously been used in these households), some likely deduced that they were in a vaccine treatment group. The enumerators who conducted the household interviews and collected both food consumption data and the anthropometric measurements were blind to the control and treatment group allocation. The other animal health technicians and the enumerators were never on the premises at the same time to prevent enumerators from identifying the group. Household allocation data was unmasked only at the end of the study by the investigators.

4.2.5 Data analysis

Child height, age, and sex were referenced to the WHO standards to create continuous measures for Height-for-Age z-score (HAZ) and Weight-for-Height z-score (WHZ) using Epi Info™ (CDC, 2019) upon importation of anthropometric variables. Stunting and wasting were defined as greater than two standard deviations below the WHO reference for mean for HAZ and WHZ respectively. Analysis on all the relational factors over the study period were done using STATA Version 16.1 (Stata, 2019) for both descriptive and inferential.

Two sets of statistical regression analyses were performed to: (a) model the determinants of food consumption by children over the course of the study including treatment effects and, (b) model the determinants of the biometric outcomes, WHZ and HAZ scores, including food consumption and treatment effects. The response variables in the food consumption regressions are transformations of the number of food servings for each of four food groups: animal sourced foods, fruits, vegetables, and grains. A first series of food intake regressions examines intake of each food category independently. The natural logarithm of servings was used as the dependent variable for each food category, based on functional form tests using Box-Cox regressions favoring loglinear over linear regression. The first set of food intake regression equations can be written as:

$$\ln(S_f) = \alpha_0^f + \alpha_t^f t + \alpha_{Tt}^f (T \times t) + \sum_{m \in \mathrm{MAM,SAM}} \alpha_{mt}^f (M_m \times t_m) + \sum_{k=1}^{K} \alpha_k^f X_k + \epsilon_f ,$$

where $\ln(S_f)$ is the logarithmic transformation of the four food group servings, $f \in (A, V, F, G)$ represents ASF, vegetables, fruit, and grains, respectively; Greek letters are parameters to be estimated, t is time since first household visit (*Time in Trial (months*), T is an indicator variable equal to 1 if a household is in the *Treatment Group* and 0 otherwise, M_j is one of three malnutrition indicators, t_m measures time since first record of moderate or severe acute malnutrition, respectively, X represents other control variables (including interaction terms) in each equation respectively, and ϵ_f is a random error term.

Hypothesis that a treatment effect of ND vaccination of household chickens on a child's diet would accumulate over the course of the trial was made. To capture this effect, a creation of an interaction variable $(T \times t)$ in Equations by multiplying T and t was done. Because T equals 0 for control households and 1 for treatment households, $(T \times t)$ equals the time in trial for treatment households and is zero for control households. The control group is therefore the base case and the α_t^f represents the direction and rate of change in consumption of food group f over the course of the trial in the control group. The parameter α^f_{Tt} associated with $(T \times t)$ represents the difference in the rate of change in food consumption in the treatment group relative to consumption in the control group. If $\alpha^f_{Tt} > 0$, consumption of food category f is increasing *relative to* consumption in the control group. For example, if providing ND vaccination resulted in an increasing amount of chicken-based animal sourced food and induced more ASF intake while ASF intake by children in the control group remained unchanged, the parameter on the interaction term will be positive, suggesting that treatment increases ASF intake relative to the control households that did not receive ND vaccination of their chickens.

The variable M in Equations takes one of three values: No Acute Malnutrition (NAM), Moderate Acute Malnutrition (MAM), and Severe Acute Malnutrition (SAM). A finding of MAM or SAM upon any household visit was the basis for triggering nutrition counselling (MAM) or counselling and supplementation (SAM). The malnutrition intervention was triggered, regardless of whether the household was in the treatment or control group. The interaction term $(M_m \times t_m)$ takes the value of zero for an individual until a first finding of either MAM or SAM, after which it counts months since this first finding until the end of the trial to assess whether these interventions had measurable effects on outcomes over time. A positive coefficient suggests that the dependent variable increases after a finding of MAM or SAM.

Food consumption history in the data reflects the transition from breastfeeding as the primary source of child nutrition through a period of increased intake of other food sources up to month 18 and diminishing reliance on breastfeeding throughout the study period (Figure 2). The total number of food servings increased at a decreasing rate through 18 months, and thereafter remained relatively constant (with slight decline), while the percent of children being breastfed declined throughout the study from close to 100% and approached zero around 40 months. This age-dependent transition was captured in the food consumption regressions (represented generally by X_k in Equation by utilizing an indicator variable *Over18mo* taking a value of 1 if a child was over 18 months old at the time of a household visit and 0 otherwise, and interacting this variable with child age, age-squared (to capture the diminishing increase in servings through 18 months), and a variable indicating whether a child was currently being breastfed (*Breastfed=1*, *Not Breastfed=0*). Further regressors in Equations include the natural logarithm of per capita household income, an indicator for female child (versus male), the age and education level of the mother, and a sine/cosine pair (trigonometric functions of month) to capture potential seasonality in food intake.

In addition to the food intake regressions represented by Equations, a fractional multinomial logit model results that estimates the fraction or share of total servings represented by each food type in each visit are included. The multinomial logit regression focuses more precisely on the substitution between food groups than the first set of regressions, which focuses more broadly on how consumption levels change. The multinomial logit regressors are identical to those in Equations, except for the addition of the natural logarithm of total servings (the sum of the number of servings in all four food categories) because food category shares are conditional on the total number of servings. HAZ and WHZ regressions were also estimated to examine factors affecting them over the course of the study. These regressions can be represented as:

$$Z = \beta_0^z + \beta_t^z t + \beta_{Tt}^z (T \times t) + \sum_{m \in \mathrm{MAM,SAM}} \beta_{mt}^z (M_m \times t_m) + \sum_{f=1}^{4} \beta_f^z \ln(S_f) + \sum_{k=1}^{K} \beta_k^z X_k + \epsilon_z, \qquad (2)$$

where $Z \in (WHZ, HAZ)$ are weight to height and height to age measures, and the rest of the content shown in Equations is as described for Equations. Treatment and time in trial variables are used as described above to estimate treatment effects, and the age category indicator (18 months or younger versus over 18 months old) is used to capture differences in response to food and breastfeeding in these different age groups, and several additional control variables are included as in Equations. However, there are differences between regressions (1) and (2). First, Equations include the four categories of food intake, $\ln(S_f)$, as explanatory variables to capture food intake effect on biometric outcomes. Second, because height to age tends to reflect the cumulative effects of nutrition during the entire growth path of a child while weight to height tends to more reflect recent nutrition, the explanatory variables in these regressions differ between the two regressions. Third, I included current breastfeeding status in the WHZ regression, but in the HAZ regression I used an indicator of whether a child was ever breastfed (*Never Breastfed versus Breastfed Ever)* during the trial before a given visit.

My data contain up to six records per child, one for each household visit. I applied a random effects model in all regressions to account for unobserved similarities in children/households between visits. The error component of the regressions can be represented as $\epsilon_{y,it} = u_i + v_{it}$, where $y \in (z, f)$ and the i and t indices identify child and visit number respectively (subscripts are omitted from Equations and to minimize notational clutter). Robust standard errors are clustered by individual child. Of particular interest are the effects of treatment on food intake, the effects of food intake on biometric scores, and the effects of treatment on Z scores conditional on food intake. Equations 1 and 2 allow estimation of what I refer to as direct effects of treatment on food intake and biometric scores, as well as indirect effects of treatment on Z scores through measured food consumption effects. The indirect effects are defined as the effects of treatment on Z scores through food consumption as measured in these regressions. The direct effect is defined as the measured effect of the treatment variables $(T \times t)$ and $(M_m \times t_m)$ included directly in the Z regressions and represents effects of treatment on nutritional outcomes not otherwise represented by our food intake-related data and therefore not captured in the food intake related parameter estimates in regressions (1) and (2). These effects are methodologically "direct" in the sense that they are captured directly in equation (2) parameters, but they may reflect complex and varied pathways from treatment to nutritional outcomes for the children in the study. The full effect of treatment on $Z \in (WHZ, HAZ)$ can be described as:

Total Treatment Effect on Z

$$= \text{Direct Effect on } Z + \text{Indirect effect on } Z \text{ through food intake } (f)$$

$$= (\text{Treatment} \rightarrow Z) + (\text{Treatment} \rightarrow f \rightarrow Z).$$

The sum of direct and indirect treatment effects is calculated mathematically based on Equations and as:

$$\frac{dZ}{dT} = \left(\frac{\partial Z}{\partial Tt} + \sum_{f=1}^{4} \frac{\partial Z}{\partial \ln(S_f)} \frac{dln(S_f)}{dTt} \right) \frac{\partial Tt}{\partial T}$$

$$= \left(\beta^{Z}_{Tt} + \sum_{f=1}^{4} \alpha^{f}_{Tt} \beta^{z}_{f} \right) t, \qquad (3)$$

Where T is a treatment indicator variable equating 1 if a child is in a treatment household and 0 if otherwise. The direct effects of treatment on Z are β^{Z}_{Tt}, and the indirect effects of treatment on Z through measured impacts of treatment on food consumption are the sum of the effects of treatment on food intake (α^{f}_{Tt}) and the effects of food intake on Z (β^{z}_{f}). These effects are conditional on duration of $t = (\textit{Time in Trial (months)})$, up to a maximum of $t = 18$.

A mathematically analogous calculation for estimating the direct effects of MAM and SAM is calculated as:

$$\frac{dZ}{dt_m} = \frac{\partial Z}{\partial M_m t_m} \frac{\partial M_m t_m}{\partial t\_m} + \sum_{f=1}^{4} \frac{\partial Z}{\partial \ln(S_f)} \frac{dln(S_f)}{dM_m t_m} \frac{\partial M_m t_m}{\partial t\_m}$$

$$= \beta^{z}_{mt} + \sum_{f=1}^{4} \beta^{z}_{f} \, \alpha^{f}_{mt}$$

where $(M_m \times t_m)$ in Equations (1) and (2) are abbreviated as $M_m t_m$, $M_m \in (MAM, SAM)$, and $\frac{\partial Z}{\partial M_m t_m} \frac{\partial M_m t_m}{\partial t\_m}$ and $\frac{dln(S_f)}{dM_m t_m} \frac{\partial M_m t_m}{\partial t\_m}$ are estimated by one parameter each in each equation in Tables 2 and 4, respectively, associated with *Time since first MAM* and *Time since first SAM*.

Household and child participation may affect both the control and treatment groups over the course of the trial. For example, if a child was found to be moderately or acutely malnourished, they were referred to a therapeutic feeding program regardless of whether they were in the treatment or control group. The full change in biometric scores Z that occurs over time during the study participation is:

$$\frac{dZ}{dt} = \frac{\partial Z}{\partial t} + \sum_{f=1}^{4} \frac{\partial Z}{\partial \ln(S_f)} \frac{dln(S_f)}{dt}$$

$$= (\beta_t^z + \beta_{Tt}^z T) + \sum_{f=1}^{4} \beta_f^z (\alpha_t^f + \alpha_{Tt}^f T)$$

For the control group, the treatment indicator T=0, so the "time in control group" effect simplifies to:

$$\left.\frac{dZ}{dt}\right|_{T=0} = \beta_t^z + \sum_{f=1}^{4} \alpha_t^f \beta_f^z \qquad (4)$$

Equation 4 is the baseline for potential effects of trial participation on biometric outcomes. Because the control group status is the base case in the regression, it reflects effects to either group.

4.3 Results

4.3.1 Demographics and nutritional status of children at enrolment

Of the 721 children who participated throughout the 18-month study, at enrolment 149 children were less than six months of age (83 children from treatment households; 66 children from control households) from 91 households, and 572 children between 6 and 36 months of age (265 children from treatment households; 307 children from control households) from 380 households. Household demographics and nutritional status at enrolment is provided in Table 1. Based on HAZ and WHZ determination, stunting and wasting were present in 17.9% and 2.7% of the children, respectively. There were no significant differences between the treatment and control households at enrolment (stunting: treatment households 16.9%, control households 18.7%, p=0.53; wasting: treatment households 3.5%, control 2.0%; p=0.23). Using MUAC measurements collected at enrolment, 9.9%

of children suffered acute malnutrition: 3.3%, severe acute malnutrition and 6.6% moderate acute malnutrition.

Table 1: Demographic Characteristics and Baseline Child Growth Measurements

Parameter	**Treatment Households**	**Control Households**
Household size (mean no. occupants)	6.3±2.4	6.1±2.2
Daily household income (mean USD)	$2.71±5.61	$2.28±4.71
Maternal (caregiver) age (mean, years)	36.7±15.4	37.6±17.9
Maternal (caregiver) education level		
No formal education	2.0%	0.6%
Primary education	67.2%	74.8%
Secondary education	28.2%	22.5%
Other	2.6%	2.1%
Age of assessed children (mean months)	21.32±15.27	21.50±14.74
Gender of assessed children (% female)	49.7%	50.1%
Stunted children on first visit	16.9%	18.7%
Wasted children on first visit	3.5%	2.0%
Diagnosis of MAM on first visit	8.6%	4.8%
Diagnosis of SAM on first visit	2.9%	3.7%

4.3.2 Impact of breastfeeding and child age on food consumption

At enrolment, 49.7% of children were breastfeeding (treatment 48.9%, control 50.4%; p=0.68). As food consumption in the data reflects the transition from a period of breastfeeding as the primary source of nutrition through a period of increased intake of other foods, assessment of this transition to establish age parameters prior to regression analyses was done. In Figure 3, food intake is represented by the sum of servings in the four food groups other than maternal milk (ASF, fruit, vegetables, grains) and plotted relative to breastfeeding and child age. The number of non-breast milk food servings increases at a decreasing rate until approximately 18 months of age, and thereafter remains relatively constant with a slight decline, while the percent of children being breastfed declines from approximately 100% and approaches zero around 40 months of age. These dynamics of breastfeeding and food intake are captured in the four food intake regressions (Table 2) and the fractional multinomial logit regression (Table 3) by the interactions between an age ≤18 months indicator variable or >18 months, a breastfeeding indicator, age, and age squared. Overall, the results associated with these control variables are consistent with the intuitive progression of increasing solid food intake through 18 months and that children not being breastfed tend to eat more solid foods than those breastfeeding. For example, the negative sign on *(18mo&under) X Breastfed* of -0.429 in the Table 2 ASF regression indicates that children ≤18 months of age being breastfed eat approximately 43% less ASF than those in this age category not being breastfed (the base case). The parameters on *(18mo&under) X Age* and *(18mo&under) X Age squared* in the *Ln (Vegetables)* regression of 0.457 and -0.014 show that for those ≤18 months of age, ASF consumption increases at a decreasing rate until about 16 months, consistent with the shape of the Locally Weighted Scatterplot Smoothing (LOWESS) regression line for total servings (Figure 7). Similar results hold for the other food groups, with some statistically insignificant exceptions (Table 2). For children over the age of 18 months, the age effects are not statistically significant except for a slight decline in grains consumption, also consistent with the nearly flat LOWESS curve after 18 months. The fractional multinomial logit regression results in Table 3 highlight substitution between food groups. The statistically significant negative coefficient *(18mo&under) X Breastfed* (-0.400) supports the finding that young children being breastfed are fed less ASF than young children not being breastfed. The large statistically significant parameters on *(Over18mo) X Breastfed* and *(Over18mo) X Not* Breastfed (-1.792 and -1.927 respectively) indicate that older children are fed less ASF relative to grains than younger non-breastfed children (base case), but breastfed older children are fed less ASF relative to grains than older non-breastfed children (p=0.066). This is consistent with a substitution effect in favor of breast milk for both younger and older children.

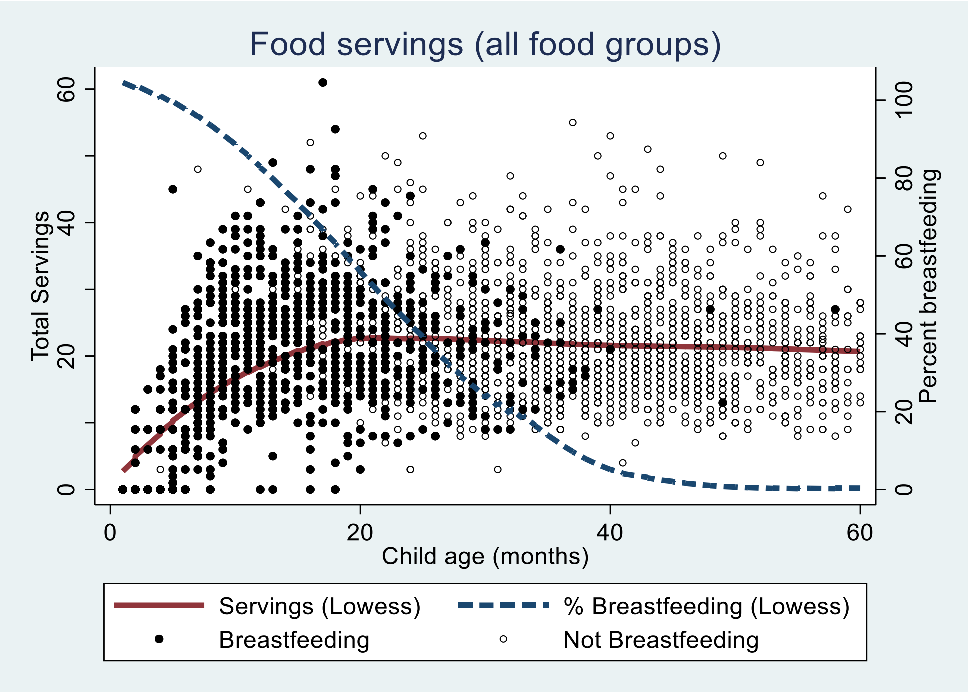

Figure 7: Transition from breastfeeding to solid foods, by child age

The total number of solid food servings increases through about age 1.5 years (18 months) and then remains relatively constant with slight decline, while the fraction of children being breastfed declines to zero.

Table 2: Food Consumption Regressions by Category

Dependent Variable:	Ln (ASF)	Ln (Fruit)	Ln (Vegetables)	Ln (Grains)
Control Group X Time in trial	Base	Base	Base	Base
Treatment Group X Time in trial	0.010 *	-0.003	-0.006	-0.006 ***
Time since first MAM	0.011	-0.006	0.005	0.007
Time since first SAM	0.027 **	0.020	0.006	0.004
Time in trial	-0.006	0.045***	-0.003	0.004*
(18mo&under) X Not Breastfed	Base	Base	Base	Base

(18mo&under) X Breastfed	-0.429***	-0.143	-0.005	-0.029
(Over18mo) X Not Breastfed	0.548	2.704***	3.449***	2.425***
(Over18mo) X Breastfed	0.358	2.481***	3.492***	2.434***
(18mo&under) X Age	0.108*	0.323***	0.457***	0.364***
(18mo&under) X Age squared	-0.005*	-0.010**	-0.014***	-0.013***
(Over18mo) X Age	-0.021	0.003	0.011	-0.012*
(Over18mo) X Age squared	<0.001	<0.001	<0.001	<0.001
Ln (Per Capita Income)	0.014	-0.002	0.000	-0.005
Female	0.066	-0.034	-0.024	-0.018
Mother's Age	0.003**	0.001	0.002*	0.000
Education Level	0.099***	0.080	0.015	0.005
cos(month)	-0.010	0.132	0.107***	0.013
sin(month)	-0.021	-0.174***	-0.079***	0.030***
Constant	0.330	-2.009***	-2.252***	0.305*
var(u)	0.173***	0.208***	0.099***	0.043***
var(v)	4.601***	13.074***	4.810***	19.093***
N	2542	2542	2542	2542

Significance: *** p<0.01, ** p<0.05, * p<0.1 MAM: Moderate Acute Malnutrition (MAM), SAS: Severe Acute Malnutrition

Table 3: Fractional Multinomial Logit Regression of Food Group Consumption Relative to Grains

	ASF	Fruits	Vegetables
Control Group X Time in trial	Base	Base	Base
Treatment Group X Time in trial	0.010**	0.005	-0.002
Time since first MAM	-0.008	-0.018*	<0.001
Time since first SAM	0.018	0.008	-0.012
Time in trial	0.005	0.029***	-0.004
(18mo&under) X Not Breastfed	Base	Base	Base
(18mo&under) X Breastfed	-0.400***	-0.197	-0.211**
(Over18mo) X Not Breastfed	-1.792***	-4.636***	-0.293
(Over18mo) X Breastfed	-1.927***	-4.681***	-0.301
(18mo&under) X Age	-0.337***	-0.003	0.005
(18mo&under) X Age squared	0.013***	0.002	0.003

(Over18mo) X Age	-0.001	0.019	0.016
(Over18mo) X Age squared	0.000	0.000	0.000
(18mo&under) X Ln (Total Servings)	-0.079	-0.387***	-0.347***
(Over18mo) X Ln (Total Servings)	-0.201**	1.166***	-0.025
Ln (Per Capita Income)	0.027***	0.010	0.014**
Female	0.056	0.060	0.036
Mother's Age	0.001	-0.002	0.002**
Education Level	0.122***	0.011	0.031
cos(month)	-0.038	0.088**	0.055***
sin(month)	-0.042	-0.165***	-0.098***
Constant	0.512	-0.948***	-1.118***
N	2542	2542	2542

Significance: *** p<0.01, ** p<0.05, * p<0.1

4.3.3 Impact of household income on food group consumption

Because the dependent variables in the food intake regression (Table 2) and per capita income (household income divided by number of household members) are both in logarithmic form, the parameters associated with per capita income represent elasticities: the percentage change in the number of total food servings in response to a 1% increase in per capita income. There are positive but not statistically significant elasticities for the income effect on ASF and vegetables while elasticities are negative for fruit and grains (Table 2). However, the fractional multinomial Logit model results in Table 3 provide additional perspective on how income affects substitution between food groups. Consumption of ASF and vegetables increased significantly relative to grains (p<0.01 and p<0.1, respectively) as household incomes rose, with the increase in ASF consumption two-thirds greater than that of vegetables (Table 3). This result is consistent with prior findings that ASF and vegetables tend to be more income-responsive than other food groups, while grains are less responsive to income or decline as a share of food expenditures (Colen *et al*., 2018; Desiere *et al*., 2018).

4.3.4 Impact of season on food group consumption

Using the sine and cosine of the scaled month of year to capture seasonality, fruits, grains, and vegetables show significant seasonal intake cycles ($p<0.01$; Table 2), whereas there is little evidence of seasonality for ASF. This is consistent with seasonal production and the limited ability to store fruit, vegetables, and grains at the household level as compared to more consistent availability of ASF. This seasonal relationship for fruits and vegetables but not ASF is also observed in the fractional multinomial logit model where consumption is referenced relative to grains (Table 3).

4.3.5 Impact of household ND vaccination of chickens on food group consumption

First consideration of the effect of being in the ND treatment group was done. The effect of being in the treatment group (a household with quarterly ND vaccination of all chickens) is captured by the parameter associated with the *Treatment Group X Time in trial* $(T \times t)$. A positive parameter estimate means that consumption for that food group increases over the course of the trial faster (and more) in the treatment group *relative to* intake of that food group by children in the control of the study (base case). ASF consumption increased faster in the treatment by 0.01% per month (p=0.082, Table 2), amounting to a 1.2% increase relative to the control over 18 months. In contrast, consumption of grains decreased in the treatment relative to the control over the course of the trial (-0.006, p= 0.010). These results suggest that the vaccination of chickens against ND may have made ASF more available to the point that households substituted children's food toward ASF and away from the other food groups, especially grains. An examination of this relationship further using the fractional multinomial logit model was done (Table 3). The results indicate that the share of ASF relative to grains increases at a significantly faster rate in the treatment (0.10, p=0.019), supporting the conclusion that ND vaccination is inducing or allowing households to substitute toward ASF and away from grains as the trial proceeds. In contrast, there are no significant changes in the shares of consumed fruits and vegetables between the treatment and control (p= 0.254 and p=0.558, respectively).

4.3.6 Impact of food consumption on child growth

For HAZ, higher average ASF consumption over the course of the trial has a relatively large, statistically strong, positive impact (0.124, p=0.002) for children over 18 months of age (Table 4). In contrast, average grain consumption over previous visits had a negative effect on HAZ for older children (-0.135, p=0.053). There were limited effects of current reported consumption on WHZ with only vegetable consumption in older children having a significant positive effect (0.029, p=0.053). The rest of the effects of food intake on older children are negative and/or not significant at conventional test sizes. Consistent with seasonality of fruit and vegetable consumption (Tables 2 and 3), there appears to be seasonal effects on WHZ (Table 4). Note that sin (month) and cos (month) were omitted for HAZ because of the longer time frame of HAZ development.

Table 4:Regression Analysis for Impact on Child Growth: Weight to Height (WHZ) and Height to Age (HAZ)

	WHZ	HAZ
Control Group X Time in trial	Base	Base
Treatment Group X Time in trial	0.009**	0.002
Time in trial	-0.002	0.127***
Time since first MAM	0.005	-0.040***
Time since first SAM	0.001	0.044*
Ln (ASF)[a] X (18mo&under)	-0.010	-0.021
Ln (ASF) X (Over18mo)	-0.003	0.124***
Ln (Fruit) X (18mo&under)	-0.029	0.030
Ln (Fruit) X (Over18mo)	-0.015*	-0.001
Ln (Vegetables)a X (18mo&under)	-0.086***	-0.101***
Ln (Vegetables) X (Over18mo)	0.029*	-0.015
Ln (Grains)a X (18mo&under)	-0.086***	-0.021
Ln (Grains) X (Over18mo)	0.013	-0.135*
Not Breastfed[b] X (18mo&under)	Base	Base
Breastfed X (18mo&under)	0.035	-3.638***
Not Breastfed X (Over18mo)	-0.278*	-0.272
Breastfed X (Over18mo)	-0.398**	-3.315***
Age	-0.020**	-0.170***
Age squared	<0.001**	0.001***
ln (Per Capita Income)	-0.005	0.000
Female	0.114	0.234*
Mother's Age	0.000	-0.001

Education Level	0.064**	-0.026
cos(month)	0.029*	
sin(month)	-0.033**	
Constant	0.011	4.501***
var(u)	0.543***	2.457***
var(v)	0.249***	0.280***
N	2525	2525

Significance: *** p<0.01, ** p<0.05, * p<0.1

Breastfeeding patterns have qualitatively similar effects on WHZ and HAZ (Table 4). Conditional on food consumption, younger children, ≤18 months of age, not being breastfed have higher WHZ than older children not being breastfed, representing a small decline in WHZ in this age category. Younger children being breastfed have higher WHZ than the same age category not being breastfed (conditional on food intake), suggesting, unsurprisingly, that breastfeeding is a positive contributor to WHZ for younger children. In contrast, older children being breastfed have lower WHZ than older children not being breastfed. This may reflect other unobserved differences in the diets of older breastfed children that affect these outcomes. The HAZ results show similar patterns.

Notably, girls had significantly higher HAZ and weakly higher WHZ as compared to boys as the base case (Table 4). The higher HAZ scores as measured directly with the *Female* indicator variable are in addition to the implied HAZ benefits from weakly higher ASF consumption (Table 2) and in ASF consumption relative to grains (Table 3) shown for girls.

[a]For the WHZ regression, *ln (Food Group)* is the logarithm of servings for the current visit. For the HAZ regression, ln (Food) is the logarithm of average servings for that food category reported in household visits to date.

[b]For the WHZ regression, *Breastfed* and *Not Breastfed* indicate whether a child is currently being breastfed. For the HAZ regression, they indicate whether a child has ever been breastfed during a recall period to date.

4.3.7 Direct, indirect, and total impacts of household ND vaccination of chickens on child growth

The increased child consumption of ASF in treatment households, both absolute and relative to grains (Tables 2, 3) represent the effects of ND vaccination mediated through changes in food consumption, denoted as *indirect effects* of being in the treatment group. In addition, the WHZ and HAZ regressions show that there are also methodologically defined *direct effects* of treatment that are captured by this data but are not accrued to the nutritional data collected. These may be treatment group impacts on nutrition not included or accurately measured in the household interviews as well as unidentified behavioral or household management changes linked to being in the treatment of the study. The equations 3 and associated discussion describe how the parameter estimates (Tables 2, 4) are used to calculate the indirect food consumption effects and the total effects of being in a treatment household on child growth. The estimated direct calculated, indirect, and calculated total treatment effects measured at 18 months after initiation of treatment are provided in Table 5. The total estimated impact on a child in the treatment of the trial, including both indirect and direct effects, is an increase in WHZ of 0.158, and an increase in HAZ of 0.075, suggesting positive overall effects of the ND treatment on both scores. Children who had been in the control households had mean WHZ of -0.86±1.23 and a mean HAZ of -0.42±0.92. These results suggest that, controlling for other factors, children in households in the ND vaccination arm had increased WHZ by about 13% of its standard deviation and HAZ by about 8% of its standard deviation (Figure 8). The indirect component, reflecting captured food consumption, of the total effect for HAZ is positive and about equal to the direct effect, suggesting that while the changes in food intake induced by the ND treatment measured by the regressions are positive, there are other impacts of treatment that are not captured by our data on food consumption and these regressions (Figure 9). In contrast, the indirect effects of ND treatment on WHZ through food consumption are negative, though very small, and are more than compensated for by the positive direct effects (Figure 9).

Table 5: Direct, Indirect, and Total Estimated Effects of a Household Receiving Quarterly ND Vaccination on WHZ and HAZ

	α^{f}_{Tt}	β^{WHZ}_{f}	β^{HAZ}_{f}	$\alpha^{f}_{Tt}\beta^{WHZ}_{f}$	$\alpha^{f}_{Tt}\beta^{HAZ}_{f}$
ASF	0.010	-0.003	0.124	-0.000030	0.001240
Fruits	-0.003	-0.015	-0.001	0.000045	0.000003
Vegetables	-0.006	0.029	-0.015	-0.000174	0.000090
Grains	-0.006	0.013	-0.135	-0.000078	0.000810
Indirect effect				-0.000237	0.002143
Direct effect (β_{Tt})				0.009	0.002
Total treatment effect over 18 months				0.158	0.075

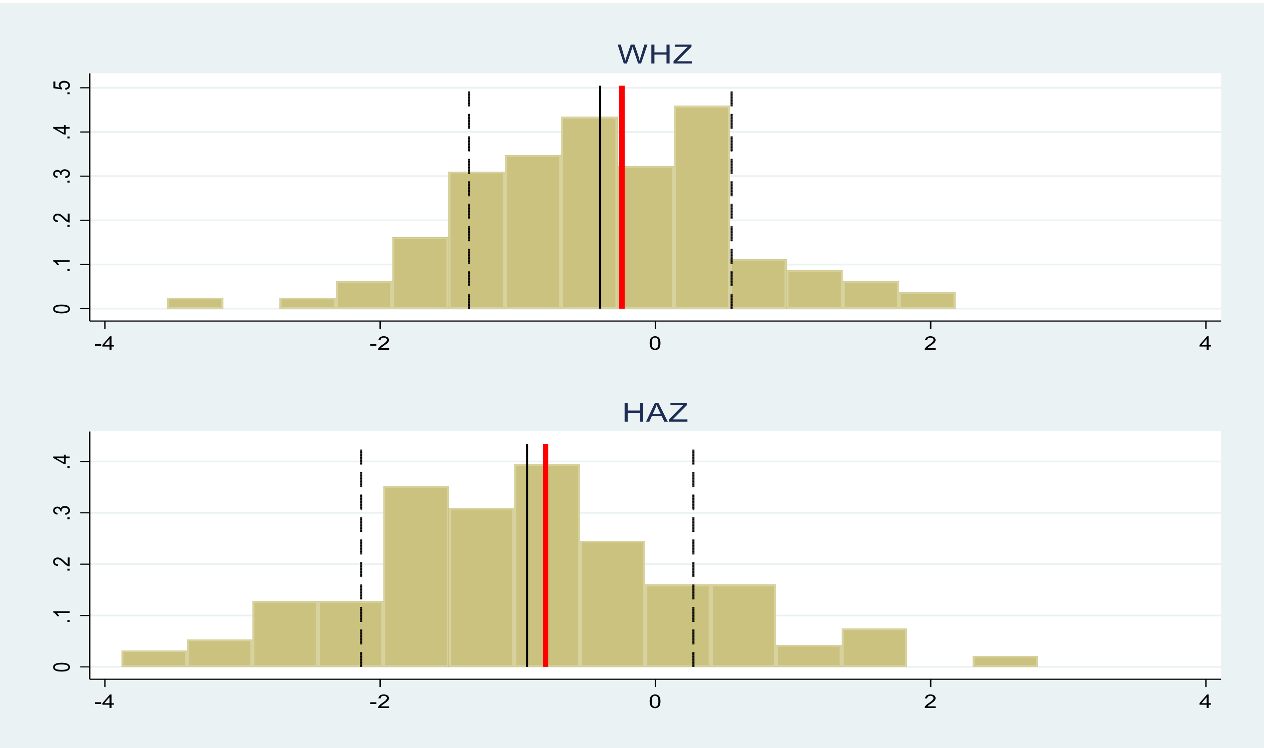

Figure 8: Effect of being in a treatment group household on child growth.

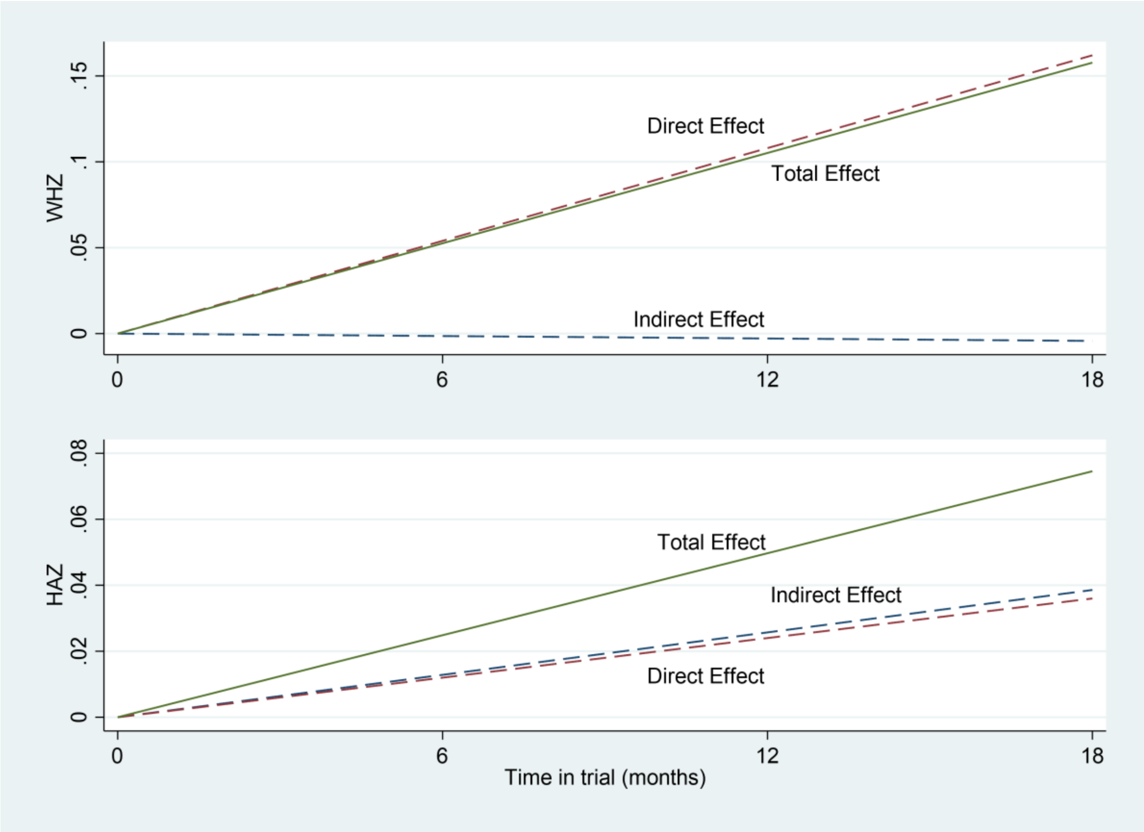

Figure 9: Direct and indirect effects of being in a treatment group household on child growth. Change in WHZ (top plot) and HAZ (bottom plot) from baseline over time.

4.3.8 Impact of nutritional interventions for acute malnutrition

If nutrition counselling after a finding of MAM or counselling and food supplementation after a finding of SAM had positive effects over time on the intake of a food category, then the associated parameters (*Time since first MAM* and *Time since first SAM*, represented in Equations 1 and 2 as $(M_m \times t_m)$) should be positive. Table 2 shows that the signs of the parameters on MAM are mixed and none are statistically significant at conventional levels. However, the MAM parameter in the ASF equation is positive and larger than the others. All SAM parameters are positive, and the ASF parameter is statistically significant and largest (0.027, p=0.036), suggesting that SAM interventions lead to an increase in ASF consumption over the remainder of the trial after a finding of SAM. These results and the decreasing number of SAM and MAM diagnoses over time (Figure 10) suggest counselling effectively promotes ASF consumption within the household as the supplemental feeding during the trial did not have a specified ASF component. As with the ND vaccination treatment effect, the nutritional interventions have both indirect and direct effects. Table 6 summarizes these results for children who were provided nutritional supplements after being identified with SAM. The direct effects of SAM interventions are positive in both the WHZ and HAZ regressions, as is the indirect effect for HAZ. However, the indirect effect of SAM intervention is negative and small for WHZ. Overall, the total direct plus indirect effects of SAM interventions are positive for both growth parameters, though small for WHZ. For HAZ, scores increase by about 0.14 over three months attributable to SAM interventions, or about 15% of one HAZ standard deviation. Over 18 months, HAZ scores would increase by about 1.68, or about 182% of one HAZ standard deviation. This 18-month projection is provided for comparison with the ND vaccination treatment results in Table 5, but because SAM interventions were implemented on a quarterly timescale (between visits), extrapolation of this effect to 18 months is dubious.

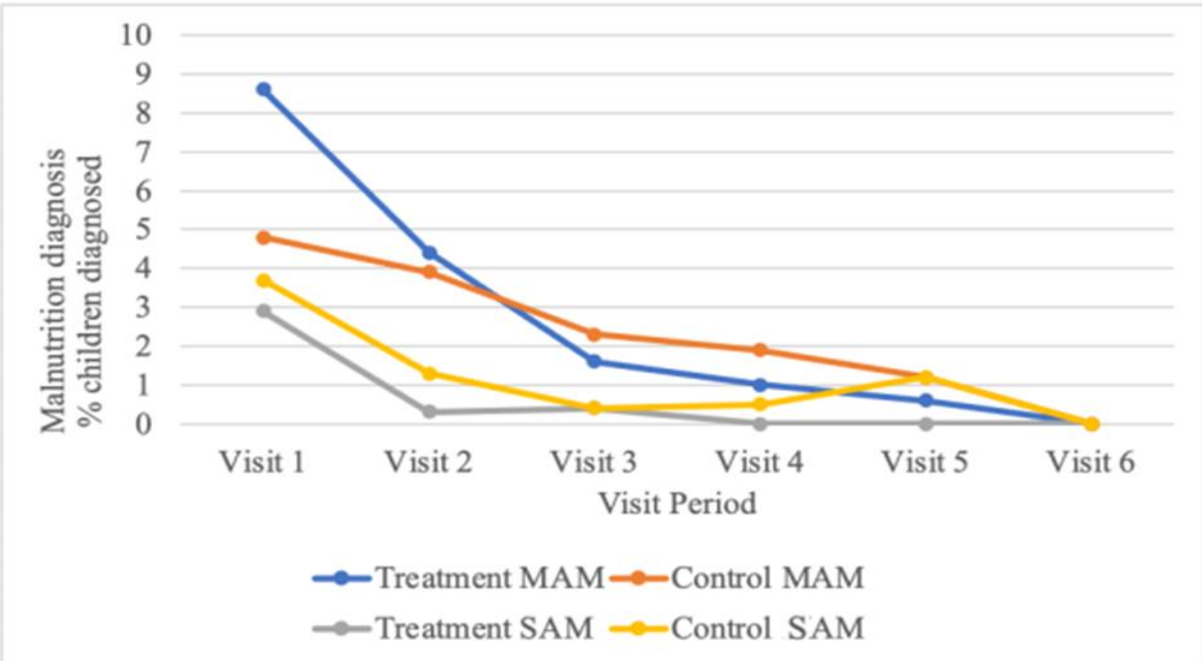

Figure 10: Diagnosis of MAM and SAM during the trial duration

Table 6: Direct, Indirect, and Total Estimated Effects of Interventions for Severe Acute Malnutrition (SAM)

	α^{f}_{Tt}	β^{WHZ}_{f}	β^{HAZ}_{f}	$\alpha^{f}_{Tt}\beta^{WHZ}_{f}$	$\alpha^{f}_{Tt}\beta^{HAZ}_{f}$
ASF	0.027	-0.003	0.124	-0.000081	0.003348
Fruits	0.020	-0.015	-0.001	-0.000300	-0.000020
Vegetables	0.006	0.029	-0.015	0.000174	-0.000090
Grains	0.004	0.013	-0.135	0.000052	-0.000540
Indirect SAM effect				-0.000155	0.002698
Direct SAM effect (β^{z}_{mt})				0.001000	0.044000
Total SAM intervention effect over 3 months				0.002535	0.140094
Total SAM intervention effect over 18 months				0.030420	1.681128

4.3.9 Residual effects of trial participation

Time in trial (months) captures any general effect on food consumption that might result from participating in the trial that is not otherwise captured as a treatment effect, a nutritional counselling or intervention effect, or the effects of other time-varying factors included in the regressions. These residual *Time in Trial* effects on food category intake in Tables 2 and 3 are mixed. These time-varying residual effects are also mixed in the WHZ and HAZ regressions in Table 4. Unexplained changes in WHZ are weak and negative (-0.002), but significantly reducing stunting (HAZ (0.127, p<0.001)).

4.4 Discussion

Despite measurable progress over the past two decades, childhood growth faltering, especially stunting, remains unacceptably high in many countries and communities in Africa (Kinyoki *et al*., 2020; Mosites *et al*., 2016a). Rural communities highly dependent on household food production and limited off-farm income or liquid assets to bridge seasonal food availability are especially vulnerable (FAO, 2015). The study community in Siaya county, Kenya reflects this vulnerability. Focus has been on chickens, a widely held autochthonous resource, managed at the household level by women, that can provide foods high in protein and rich in micronutrients that are critical in preventing stunting (Akinola and Essien, 2011; Allen and Dror, 2011; Krebs *et al.*, 2011; FAO, 2014). Vaccine intervention was initiated recognizing that the pathway from veterinary vaccination to increased chicken productivity through household decision-making to improved nutrition and child growth is complex and impacted by multiple known and unknown factors. Nonetheless, this data shows statistically significant impacts on the intermediate measure of increased ASF consumption and on child growth parameters. This supports concerted ND vaccination of household chickens, for which willingness-to-pay studies indicate strong household interest (Campbell *et al.*, 2018a), as part of a multi-pronged approach to enhance childhood nutrition and reduce stunting in rural communities where chickens are a common household resource. These efforts would shape policies that works to reduce flock losses thereby providing households with more proteins and micronutrients that are critical dietary needs to curb stunting common in rural Kenya and other lower income countries.

The gains measured in treatment households relative to control households very likely underestimate the impact of ND vaccination on ASF consumption and childhood growth due to several inherencies of the trial design. First, for ethical reasons, the control households also received an intervention, medication for parasites for their chickens (as did the treatment households in addition to ND vaccination). This medication, plus any unmeasured veterinary advice provided at the time of treatment, is reflected in the increase in flock size in the control households over baseline and the significant effect of “time in trial” reported here. Second, vaccine was delivered quarterly—this interval is appropriate to maintain immunity in previously vaccinated chickens but misses all chicks hatched in the interim, the age group most susceptible to dying from ND (Miller *et al.*, 2015). This challenge is addressed in the subsequent paragraph. Third, all households received quarterly data on their children's growth and *ad hoc* dietary and poultry management guidance that may have influenced their decisions on both flock management and children's diets. Notably, girls appear to be equal beneficiaries of the substitution towards ASF consumption. Although not linked to a treatment effect, overall the significant increase in both HAZ and WHZ in girls relative to boys is discrepant from prior studies in other regions of Kenya where girls had significantly lower HAZ and WHZ (Ndiku *et al*., 2011). Speculation that this reflects the primary role of women in management of household poultry linked to dietary consumption choices for their children as either parents or caregivers (Nordhagen and Klemm, 2018; Robyn, 2019).

Translating the increase in flock size gained through ND vaccination to a change in a young child's diet is mediated through a household decision, in rural Kenya usually maternal (Ruel and Alderman, 2013; Mosites *et al*., 2016a; b; FAO, 2020). The most significant increase in flock size in treatment households as compared to control households was due to an increase in laying hens. Based on prior studies of indigenous chicken productivity in this region, an increase in 1 hen per flock would result in average production of 6-7 eggs per month, with a potential increase of 6g of protein per egg (Chepkemoi *et al*., 2015; Olwande *et al*., 2010). The impact of increased egg production on child growth is supported by a study demonstrating that each instance of child consumption of an egg during a prior 3-day period was significantly linked to an increase in child height (Mosites *et al*., 2016a). In the present study, the positive and negative signs of the treatment coefficient in the ASF and grains regressions on HAZ and WHZ, respectively, are consistent with the substitution away from high carbohydrate, low protein grains toward high protein ASF, leading a higher protein diet but fewer total calories. This implies that although the treatment provided households with more in-home, accessible protein, there is still a trade-off in resource constrained households: a small drop in WHZ for a larger increase in HAZ. This type of apparent substitution is not inevitable but is a common behavioral response in resource constrained decision making. In contrast to vegetables and fruits, ASF are much less influenced by season (Kristjanson *et al*., 2012) and thus may enhance HAZ to a greater degree due to HAZ reflecting growth over time.

In addition to the effect of being in a treatment household on child growth mediated through food consumption, there were measured effects of nutritional interventions provided to children identified as suffering from moderate or severe acute malnutrition independent of treatment group. While the nutritional counselling approach pursued for moderately acutely malnourished children showed mixed results in terms of effects on WHZ and HAZ, the nutritional supplement program pursued for severely acutely malnourished children resulted in increased ASF consumption and appears to have provided a substantive boost to HAZ scores for those children. This is consistent with impacts of prior ASF supplementation programs conducted in Kenya (Siekmann *et al*., 2003; Neumann *et al*., 2003).

Unidentified effects of being in an ND treatment household on child growth, here denominated as direct treatment effects in the WHZ and HAZ regressions, are captured by the *Treatment Group X Time in trial* interaction term. As child growth measures, collection of data on quality and quantity of consumed foods, and anti-parasite medications were common to both treatment and control groups, the direct effects appear to derive from either household observing vaccination or the increased time that the interviewers and animal health technicians were on the premises due to the additional requirement for vaccination. The latter provided more time for household members to interact with the animal health technicians and potentially receive additional advice regarding poultry and livestock husbandry, crop management, or other issues affecting food production and availability. The direct observation of ND vaccination may also have had an effect. Campbell *et al.* (2018b) identified that knowing a neighbour who vaccinated their chickens had the most significant impact on the decision of a given household to vaccinate. Whether households in the treatment group that routinely observed the vaccination process invested more of their own resources into poultry management was not captured in this study.

Prior studies have established a link between the health of poultry and livestock in rural smallholder farms, and both decreased human disease and enhanced childhood growth (Thumbi *et al*., 2015). While previous studies have documented the impacts of livestock ownership on child nutrition (Rawlins *et al*., 2014; Headey *et al*., 2018), this study provides new evidence on the impact of livestock health interventions. The positive impact of ND vaccination on flock size and its translation into increased ASF consumption and improved child growth provides a compelling rationale for the minimal investments required for widespread vaccination in rural households. The high percentage of chicken ownership in the study site in Siaya county, Kenya is representative of rural households across Africa and in other rural, low-income regions within Asia and South and Central America. A willingness-to-pay study indicated that households, in Tanzania but with similar characteristics as in Siaya county, Kenya, were willing to pay roughly twice the market price for ND vaccines, which are readily available in east Africa (Campbell *et al*., 2018b). However the small number of chickens per household disincentivizes market-based delivery mechanisms and the opportunity cost to individual household members, usually women, to travel and purchase vaccines is a disincentive (Campbell *et al*., 2018a). This additional burden on women is consistent with studies examining the impact of early childhood interventions on mothers (Evans *et al*., 2021). Subsidizing animal health technicians to deliver vaccines to households is proposed to overcome these disincentives and provide a pathway to enhance household poultry productivity with impacts on household well-being and reduced childhood growth faltering.

4.5 Conclusion

Vaccination of chickens against ND has a causal impact on children's consumption of ASF rich in protein and micronutrients relative to a high carbohydrate, grain-based diet. Children in treatment households (chicken vaccination) showed overall increases in Z scores for both HAZ and WHZ relative to control households, benefiting both girls and boys. The findings demonstrate the impact of directing interventions at common on-farm assets managed by women in rural communities and support programs to enhance productivity at the household level.

CHAPTER FIVE: GENERAL DISCUSION, CONCLUSIONS AND RECOMMENDATIONS

5.1. GENERAL DISCUSSION

As poultry represents a widely held economic, nutritional, and sociocultural asset in rural communities worldwide and are frequently managed by women, potential establishments and benefits of larger flocks have not been fully utilized (Wong *et al*., 2017). Average number of chickens kept at the household level is reported to be low with a recent longitudinal study in Siaya county, Kenya reporting a mean number of 10 chickens per household (Otiang *et al*., 2020). Potential increases in flock size is constrained principally by mortality with ND cited as the major cause globally and is responsible in smallholder flocks (Ahlers *et al*., 2009). Routine control measures of ND improves survival, increases ownership and productivity thus improving household welfare (Valdivia, 2001; Kristjanson *et al*., 2012).

Consistent with prior studies, the overall flock size was small but with increases over time. The mean number of chickens owned at monthly census increased by one with significant gains in number of chicks in the vaccinated versus the control flocks. Household reported more gains per month when vaccinated. Gains were balanced by voluntary decreases reflecting decision-making to maximize benefits per unit labor by voluntary reduction of chicken numbers by consumption or sale versus involuntary losses due to mortality, and losses due to predation which were marginally higher in control households (Otiang *et al*., 2020).

With childhood growth faltering remaining unacceptably high in sub-Saharan Africa, rural communities dependent on household food production with limited off-farm income or liquid assets to bridge seasonal food availability are especially vulnerable (Bloss *et al*., 2004; Mosites *et al*., 2016a; b). A cross-sectional survey in Siaya county, Kenya identified 23.5% and 4.8% of children under 5 years of age as stunted and wasted, respectively, using height-for-age Z scores (HAZ) to detect stunting and weight-for-height Z scores (WHZ) for wasting (Mosites *et al*., 2016a). Although these households are classified as living in poverty or extreme poverty with very limited off-farm income, households commonly have on-farm resources that could be developed to improve nutrition. While 95% of these households have chickens and consumption of eggs was shown to increase childhood growth by an average of 5%, the average flock size is small and constrained by high mortality due to infectious disease (Otiang *et al*., 2020).

ND vaccination relieved this constraint and translated into household decisions influencing the diets and growth of children as the intervention demonstrated a causal impact on children's consumption of ASF rich in protein and micronutrients relative to a high carbohydrate, grain-based diet. Children in treatment households showed overall increases in Z scores for both HAZ and WHZ relative to control households, benefiting both girls and boys. This result is consistent with the cumulative importance of protein consumption on HAZ as a growth measure. The findings demonstrated the impact of directing interventions at common on-farm assets managed by women in rural communities and supported programs to enhance productivity at the household level.

This randomized controlled trial demonstrated that relieving a constraint on household nutritional assets, hereby reducing disease of chickens through vaccination, households make dietary choices for young children that increase consumption of protein and micronutrient rich foods and decrease relative consumption of high carbohydrate, low protein grains.

The study provided new causal evidence that this shift in diet results in improved child growth, most notably in height for age but also for weight for height, parameters used to assess childhood stunting and wasting, respectively. Given the high prevalence of childhood growth failure in rural Africa and in Siaya county, Kenya, these results highlight the potential to maximize utility of a common household animal asset to reduce the unacceptably high burden of childhood stunting in these communities.

5.2. CONCLUSIONS

1. ND vaccination and parasiticidal treatment resulted in an increase in flock size.

2. Comparison with previous flock sizes in the study households indicate a more significant benefit of survival of chicken from the combined vaccination and parasiticidal treatment.
3. Vaccination of chickens against ND has a causal impact on children's consumption of ASF rich in protein and micronutrients relative to a high-carbohydrate, grain-based diet.
4. Children in treatment households (chicken vaccination) showed overall increases in scores for both HAZ and WHZ relative to control households, benefiting both girls and boys.

5.3. RECOMMENDATIONS

1. Programs emphasizing comprehensive community approaches such as combined chicken vaccination and parasiticidal treatment on the commonest rural household on-farm animal asset managed by women and majorly constrained by ND, effectively improves flock health and household benefits of production in the smallholder setting, further impacting on household well-being and reduced childhood growth faltering.
2. Parasitic control improves immune responses and helps in protecting other chicken infections.
3. Subsidizing animal health technicians to deliver vaccines to households is proposed as an important aspect in overcoming disincentives that are associated with poor vaccine uptake.

REFERENCES

Abdisa T. and **Tagesu T**. **(2017)**: Review on Newcastle Disease of Poultry and its Public Health Importance. Journal of Veterinary Science & Technology, **8**(3).

Ahlers C., Alders R., Bagnol B., Cambaza A., Bela, Harun M., Mgomezulu R., Msafi H., Pym B., Wegener P., Wethli E. and **Young M.** (**2009**): Village Chicken Production: a manual for field workers and trainers (Issue **139**).

Akinola L. A. F. and **Essien A.** (**2011**): Relevance of rural poultry production in developing countries with special reference to Africa. World's Poultry Science Journal, **67**(4), pp. 697–705. doi: 10.1017/S0043933911000778.

Alders R. and **Spradbrow P.** (**2001**): Controlling Newcastle Disease in Village Chickens: A Field Manual. ACIAR Monograph No. 87; 2002.

Alders R., Anjos F., Bagnol B., Fumo A., Mata B. and **Young M.B. (2002)**: Controlling Newcastle Disease in Village Chickens - A Training Manual.ACIAR Monograph No.117715.

Alders R., House C. and **Bagnol B. (2002**): Controlling Newcastle Disease in Village Chickens - A Training Manual (Issue **May 2014**).

Alexander D. J. (2000): Newcastle disease and other avian paramyxoviruses. OIE Revue Scientifique et Technique, **19**(2), 443–462. https://doi.org/10.20506/rst.19.2.1231.

Allen L. H. and **Dror D. K. (2011**): Effects of Animal Source Foods, with Emphasis on Milk, in the Diet of Children in Low-Income Countries. Milk and Milk Products in Human Nutrition, 113–130.

Amornkul P. N., Vandenhoudt H., Nasokho P., Odhiambo F., Mwaengo D., Hightower A., Buvé A., Misore A., Vulele J., Vitek C., Glynn J., Greenberg A., Slutsker L. and **De Cock K. M. (2009)**: HIV prevalence and associated risk factors among individuals aged 13-34 years in rural Western Kenya. PLoS ONE, **4**(7). https://doi.org/10.1371/journal.pone.0006470.

Apopo A. A., Kariithi H. M., Ateya L. O., Binepal Y. S., Sirya J. H., Dulu T. D., Welch C. N., Hernandez S. M. and **Afonso C. L. (2020)**: A retrospective study of Newcastle disease in Kenya. Tropical Animal Health and Production, **52**(2), 699–710. https://doi.org/10.1007/s11250-019-02059-x.

Baylis M. and **Risley C. (2012)**. Infectious Diseases, Climate Change Effects on Infectious Diseases. In Selected Entries from the Encyclopedia of Sustainability Science and Technology. https://doi.org/10.1007/978-1-4419-0851-3.

Bigogo G., Audi A., Aura B., Aol G., Breiman R. F. and **Feikin D. R. (2010)**: Health-seeking patterns among participants of population-based morbidity surveillance in rural western Kenya: Implications for calculating disease rates. International Journal of Infectious Diseases, **14**(11), e967–e973. https://doi.org/10.1016/j.ijid.2010.05.016.

Bloss E., Wainaina F. and **Bailey R. C. (2004)**: Prevalence and predictors of underweight, stunting, and wasting among children aged 5 and under in Western Kenya. Journal of Tropical Pediatrics, **50**(5), 260–270. https://doi.org/10.1093/tropej/50.5.260.

Campbell Z. A., Otieno L., Shirima G. M., Marsh T. L. and Palmer G. H. (2018a): Drivers of vaccination preferences to protect a low-value livestock resource: Willingness to pay for Newcastle disease vaccines by smallholder households. Vaccine, **11**(58), 11–18. https://doi.org/10.1016/j.vaccine.2018.11.058.

Campbell Z. A., Marsh T. L., Mpolya E. A., Thumbi S. M. and **Palmer G. H. (2018b)**: Newcastle disease vaccine adoption by smallholder households in Tanzania: Identifying determinants and barriers. PLoS ONE, **13**(10), 1–16.

Campbell Z. A., Thumbi S. M., Marsh T. L., Quinlan M. B., Shirima G. M. and **Palmer G. H. (2019)**: Why isn't everyone using the thermotolerant vaccine? Preferences for Newcastle disease vaccines by chicken-owning households in Tanzania. PLoS ONE, **14**(8), 1–15. https://doi.org/10.1371/journal.pone.0220963.

CDC. (2019): CDC https://www.cdc.gov/epiinfo/index.html 2019. Epi Info™, Division of Health Informatics and Surveillance (DHIS), Center for Surveillance, Epidemiology and Laboratory Services (CSELS).

Central Bureau of Statistics (CBS) [Kenya], Ministry of Health (MOH) [Kenya] and ORC Macro. (**2004**): Kenya Demographic and Health Survey 2003. Calverton, Maryland: CBS, MOH, and ORC Macro.

Colen L., Melo P. C., Abdul-Salam Y., Roberts D., Mary S. and **Gomez Y Paloma S. (2018)**: Income elasticities for food, calories and nutrients across Africa: A meta-analysis. Food Policy, **77**(April), 116–132. https://doi.org/10.1016/j.foodpol.2018.04.002.

College Station T. S. L. (2019): StataCorp. 2019. Stata Statistical Software: Release **16.**

Copland J. and **Spradbrow P. (1997)**: Local Production of Thermostable Vaccine To Protect Village Chickens Against Newcastle Disease. Epiderniol. Sante Anim., 31–32.

Copland J. and **Alders R. G. (2005)**: The Australian village poultry development programme in Asia and Africa. World's Poultry Science Journal, **61**(1), 31–38. https://doi.org/10.1079/wps200439.

County Government of Siaya. (2018): County Annual Development Plan 2019-2020.

Desiere S., Hung Y., Verbeke W. and **D'Haese M. (2018)**: Assessing current and future meat and fish consumption in Sub-Sahara Africa: Learnings from FAO Food Balance Sheets and LSMS household survey data. Global Food Security, **16**(June), 116–126. https://doi.org/10.1016/j.gfs.2017.12.004.

Development Initiatives. (2018): Global Nutrition Report: Shining a light to spur action on nutrition. In Bristol, UK: Development Initiatives. https://doi.org/10.1063/1.4759115.

Dwinger R. H. and **Unger H. (2006)**: Improving farmyard poultry production in Africa: Interventions and their economic assessment. In Proceedings of a final research coordination meeting organized by the joint FAO/IEA Division of Nuclear techniques in Food and Agriculture. http://www-pub.iaea.org/mtcd/publications/pdf/te_1489_web.pdf.

Earnshaw J. and **Lewis G. (2008)**: NICE Guide to the Methods of Technology Appraisal Pharmaceutical Industry Perspective. Pharmacoeconomics, **26(**9), 725–727.

Evans D. K., Jakiela P. and **Knauer H. A. (2021)**: The impact of early childhood interventions on mothers. Science, **372**(6544), 794–796. https://doi.org/10.1126/science.abg0132.

Fallou G. E. H. (2000): Women and family poultry production in rural Africa. Development in Practice, **10**(1), 98–102.

FAO, IFAD, UNICEF, WFP and **WHO. (2019)**: Food Security and Nutrition in the World 2019. In Safeguarding against economic slowdowns and downturns. Rome, FAO.

FAO. (2014): Family poultry development-Issues, opportunities and constraints. In Animal Production and Health (Vol. 12).

FAO. (2015): The economic lives of smallholder farmers. http://www.macrothink.org/journal/index.php/rae/article/view/6320%0Ahttp://www.upov.int/edocs/mdocs/upov/en/upov_sym_ge_11/upov_sym_ge_11_10.pdf%0Ahttp://ajae.oxfordjournals.org/cgi/doi/10.2307/1241587%0Awww.iosrjournals.org%0Ahttp://link.springer.com/10.

Feikin D. R., Audi A., Olack B., Bigogo G. M., Polyak C., Burke H., Williamson J. and **Breiman R. F. (2010)**: Evaluation of the optimal recall period for disease symptoms in home-based morbidity surveillance in rural and urban Kenya. International Journal of Epidemiology, **39**(2), 450–458. https://doi.org/10.1093/ije/dyp374.

Fisher R.J. (1993): Social desirability bias and the validity of indirect questioning. Journal of Consumer Research, **20**(2), 303–315. https://doi.org/10.2307/2489277.

Fisher H. (2014): Newcastle disease control in Africa. ACIAR Impact Assessment Series Report No. 87. 62.

Gallili G. E. and **Ben-Nathan D. (1998)**: Newcastle disease vaccines. Biotechnology Advances, **16**(2), 343–366.

Getabalew M., Alemneh T., Akeberegn D., Getahun D. and **Zewdie D. (2019)**: Epidemiology, Diagnosis & Prevention of Newcastle Disease in Poultry. American Journal of Biomedical Science & Research, **3**(1), 50–59. https://doi.org/10.34297/ajbsr.2019.03.000632.

Guèye E. F. (2005): Gender aspects in family poultry management systems in developing countries. World’s Poultry Science Journal, **61**(1), 39–46. https://doi.org/10.1079/WPS200440.

Grosso G., Mateo A., Rangelov N., Buzeti T. and **Birt C. (2020)**: Nutrition in the context of the Sustainable Development Goals. European Journal of Public Health, **30**(1), i19–i23. https://doi.org/10.1093/eurpub/ckaa034.

Headey D., Hirvonen K. and **Hoddinott J. (2018)**: Animal sourced foods and child stunting. American Journal of Agricultural Economics, **100**(5), 1302–1319. https://doi.org/10.1093/ajae/aay053.

Kingori A.M, Wachira A. and **Tuitoek J. (2010)**: Indigenous Chicken Production in Kenya: A Review. International Journal of Poultry Science, **9**(4), 309–316.

Kinyoki D. K., Osgood-Zimmerman A. E., Pickering B. V., Schaeffer L. E., Marczak L. B., Lazzar-Atwood A., Collison M. L., Henry N. J., Abebe Z., Adamu A. A., Adekanmbi V., Ahmadi K., Ajumobi O., Al-Eyadhy A., Al-Raddadi R. M., Alahdab F., Alijanzadeh M., Alipour

V., Altirkawi K., … Hay S. I. (2020): Mapping child growth failure across low- and middle-income countries. Nature, **577**(7789), 231–234. https://doi.org/10.1038/s41586-019-1878-8.

Knueppel D., Cardona C., Msoffe P., Demment M. and **Kaiser L. (2010)**: Impact of vaccination against chicken Newcastle disease on food intake and food security in rural households in Tanzania. Food and Nutrition Bulletin, **31**(3), 436–445. https://doi.org/10.1177/156482651003100306.

Komba E.V., Mbyuzi A.O., and **Mulangila R.C. (2012)**: Adoption of I-2 vaccine in immunization of village chickens against Newcastle Disease Virus in southern Tanzania: Immune status of farmer vaccinated birds. J Agric Sci. 2012; 4(4).

Krebs N. F., Hambidge K. M., Mazariegos M., Westcott J., Goco N., Wright L. L., Koso-Thomas M., Tshefu A., Bose C., Pasha O., Goldenberg R., Chomba E., Carlo W., Kindem M., Das A., Hartwell T. and **McClure E. (2011)**: Complementary feeding: A Global Network cluster randomized controlled trial. BMC Pediatrics, **11**(1), 4. https://doi.org/10.1186/1471-2431-11-4.

Krebs N. F., Mazariegos M., Tshefu A., Bose C., Sami N., Chomba E., Carlo W., Goco N., Kindem M., Wright L. L. and **Hambidge K.M. (2011)**: Meat consumption is associated with less stunting among toddlers in four diverse low-income settings. Food and Nutrition Bulletin, **32**(3), 185–191. https://doi.org/10.1177/156482651103200301.

Kristjanson P., Neufeldt H., Gassner A., Mango J., Kyazze F. B., Desta S., Sayula G., Thiede B., Förch W., Thornton P. K. and **Coe R. (2012)**: Are food insecure smallholder households making changes in their farming practices? Evidence from East Africa. Food Security, **4**(3), 381–397. https://doi.org/10.1007/s12571-012-0194-z.

Kryger K. N., Thomsen K. A., And M. A. W. and **Dissingtion M. (2010)**: Smallholder Poultry Production – Livelihoods, Food Security and Sociocultural Significance. In Smallholder Poultry Production.

Lesnoff M. (2009): Reliability of a Twelve-Month Retrospective Survey Method for Estimating Parturition and Mortality Rates in a Traditional African Livestock Farming System. Revue Élev. Méd. Vét. Pays Trop., **62**(1), 49–5.

Lindahl J. F., Young J., Wyatt A., Young M., Alders R., Bagnol B., Kibaya A. and **Grace D. (2019)**: Do vaccination interventions have effects? A study on how poultry vaccination interventions change smallholder farmer knowledge, attitudes, and practice in villages in Kenya and Tanzania. Tropical Animal Health and Production, **51**(1), 213–220. https://doi.org/10.1007/s11250-018-1679-3.

Lowder S. K., Skoet J. and **Raney T. (2016**): The Number, Size, and Distribution of Farms, Smallholder Farms, and Family Farms Worldwide. World Development, **87**, 16–29.

Maina A. (2005): Prevalence, intensity and lesions associated with gastro-intestinal parasites of indigenous chickens in Kenya. https://doi.org/10.1002/dvdy.24093.

Mapiye C., Mwale M., Mupangwa J. F., Chimonyo M., Foti R. and **Mutenje M. J. (2008)**: A research review of village chicken production constraints and opportunities in Zimbabwe. Asian-Australasian Journal of Animal Sciences, **21**(11), 1680–1688. https://doi.org/10.5713/ajas.2008.r.07.

Marangon S. and **Busani L. (2007)**: The use of vaccination in poultry production. Revue Scientifique et Technique (International Office of Epizootics), **26**(1), 264–274.

Miller P. J, Haddas R., Simanov L., Lublin A., Rehmani S.F., Wajid A., Bibi T., Khan T.A., Yaqub T., Setiyaningsih S. and **Afonso C.L. (2015)**: Identification of new sub-genotypes of virulent Newcastle disease virus with potential panzootic features. Infect Genet Evol., **29**(January), 216–229. https://doi.org/doi: 10.1016/j.meegid.2014.10.032.

MoALF. (2016): Climate Risk Profile for Siaya. Kenya County Climate Risk Profile Series. The Kenya Ministry of Agriculture, Livestock and Fisheries (MoALF), Nairobi, Kenya. (Issue April). https://www.climatelinks.org/sites/default/files/asset/document/2017_Cadmus_Climate-Risk-Profile_Haiti.pdf%0Ahttps://www.climatelinks.org/sites/default/files/asset/document/2017_USAID ATLAS Climate Risk Profile - India.pdf.

Mosites E. M., Rabinowitz P. M., Thumbi S. M., Montgomery J. M., Palmer G. H., May S., Rowhani-Rahbar A., Neuhouse M. L. and **Walson J. L. (2015)**: The relationship between livestock ownership and child stunting in three countries in eastern Africa using national survey data. PLoS ONE, **10**(9), 1–10. https://doi.org/10.1371/journal.pone.0136686.

Mosites E., Thumbi S. M., Otiang E., McElwain T. F., Njenga M., Rabinowitz P. M., Rowhani-Rahbar A., Neuhouse M. L., May S., Palmer G. H. and **Walson J. L. (2016a)**: Relations between Household Livestock Ownership, Livestock Disease, and Young Child Growth. Journal of Nutrition, **146**(5), 1118–1124. https://doi.org/10.3945/jn.115.225961.

Mosites E., Aol G., Otiang E., Bigogo G., Munyua P., Montgomery J. M., Neuhouser M. L., Palmer G. H. and **Thumbi S. M. (2016b)**: Child height gain is associated with consumption of animal-source foods in livestock-owning households in Western Kenya. Public Health Nutrition, **20**(2), 336–345. https://doi.org/10.1017/S136898001600210X.

Msoffe P. L. M., Bunn D., Muhairwa A. P., Mtambo M. M. A., Mwamhehe H., Msago A., Mlozi M. R. S. and **Cardona C. J. (2010)**: Implementing poultry vaccination and biosecurity at the village level in Tanzania: A social strategy to promote health in free-range poultry populations. Tropical Animal Health and Production, **42**(2), 253–263. https://doi.org/10.1007/s11250-009-9414-8.

Msoffe P., Minga U., Olsen J., Yongolo M., Juul-Madsen H. R., Gwakisa P. and **Mtambo M. (2001)**: Phenotypes including immunocompetence in scavenging local chicken ecotypes in Tanzania. Tropical Animal Health and Production, **33**(4), 341–354.

Ndiku M., Jaceldo-Siegl K., Singh P. and **Sabaté J. (2011)**: Gender inequality in food intake and nutritional status of children under 5 years old in rural Eastern Kenya. European Journal of Clinical Nutrition, **65**(1), 26–31. https://doi.org/10.1038/ejcn.2010.197.

Nega M., Moges F., Mazengia H., Zeleke G. N. and **Tamir S. (2012)**: Evaluation of i2 thermostable Newcastle disease vaccine on local chickens in selected districts of western Amhara. Online Journal of Animal Feed Res., **2**(3), 244–248.

Neumann C. G., Bwibo N.O., Murphy S.P., Sigman M., Whaley S., Allen L.H., Guthrie D., Weiss R.E. and **Demment M.W. (2003)**: Animal source foods to improve micronutrient nutrition and human function in developing countries: micronutrient deficiencies and cognitive functioning. Journal Nutrition, **133**(11), 3927S–3931S.

Njagi L., Nyaga P., Mbuthia P., Bebora L., Michieka J., Kibe J. and **Minga U.** (**2010**): Prevalence of Newcastle disease virus in village indigenous chickens in varied agro-ecological zones in Kenya. Livestock Research for Rural Development **22**(95).

Nordhagen S. and **Klemm R. (2018)**: Implementing small-scale poultry-for-nutrition projects: Successes and lessons learned. Maternal and Child Nutrition, **14**(July), 1–10. https://doi.org/10.1111/mcn.12676.

Nyaga P. (2007): Poultry Sector Country Review: Kenya. In FAO Poultry Sector Country Review.

Ochieng J., Owuor G. and **Omeda B.B.** (**2012**): Determinants of adoption of management interventions in indigenous chicken production in Kenya. African Journal of Agricultural and Resource Economics **7**(1), pp. 39–50.

Okitoi L.O., Udo H.M.J., Mukisira E.A., De Jong R. and **Kwakkel R. (2006)**: Evaluation of Low – Input Interventions for Productivity of Indigenous Chickens in Western Kenya. Agricultura Tropica Et Subtropica, **39**(3), 179–182.

Olwande P. O., Ogara W. O., Okuthe S. O., Muchemi, G., Okoth E., Odindo M. O. and **Adhiambo R. F. (2010)**: Assessing the productivity of indigenous chickens in an extensive management system in Southern Nyanza, Kenya. Tropical Animal Health and Production, **42**(2), 283–288. https://doi.org/10.1007/s11250-009-9418-4.

Ondwasy H.O., Okitoi L.O, Wesonga H.O. and **Emon'gor R.A.**, (**2006**): Field Experiences on Newcastle Disease Vaccination of Local Poultry in Western Kenya. Kenya Agricultural Research Institute, Nairobi (Kenya). **7**(1), 437–443.

Onis M., Dewey K., Borghi E., Onyango A., Blössner M., Daelmans B., Piwoz E. and **Branca F. (2013)**: The World Health Organization's global target for reducing childhood stunting by 2025: rationale and proposed actions. Maternal and Child Nutrition, **9**.

Onis M. and **Branca F. (2016)**: Review Article Childhood stunting : a global perspective. Maternal and Child Nutrition, **12**(Suppl.1), 12–26. https://doi.org/10.1111/mcn.12231.

Otiang E., Campbell Z. A., Thumbi S. M., Njagi L. W., Nyaga P. N. and **Palmer G. H. (2020)**: Mortality as the primary constraint to enhancing nutritional and financial gains from poultry: A multi-year longitudinal study of smallholder farmers in western Kenya. PLoS ONE, 15(5), 1–11. https://doi.org/10.1371/journal.pone.0233691.

Randolph T. F., Schelling E., Grace D., Nicholson C. F., Leroy J. L., Cole D. C., Demment M. W., Omore A., Zinsstag J. and **Ruel M. (2007)**: Invited Review: Role of livestock in human nutrition and health for poverty reduction in developing countries. Journal of Animal Science, **85**(11), 2788–2800. https://doi.org/10.2527/jas.2007-0467.

Rawlins R., Pimkina S., Barrett C.B., Pedersen S. and **Wydick B. (2014)**: Got milk? The impact of Heifer International's livestock donation programs in Rwanda on nutritional outcomes. Food Policy, **44, 2**02–213.

Robyn W.K. (2019): Do women reap the benefits? Exploring access and social exclusion among village chicken producers in Kenya. https://doi.org/10.1017/CBO9781107415324.004.

Ruel M. T. and **Alderman H. (2013)**: Nutrition-sensitive interventions and programmes: How can they help to accelerate progress in improving maternal and child nutrition? The Lancet, **382**(9891), 536–551. https://doi.org/10.1016/S0140-6736(13)60843-0.

Siekmann J. H., Allen L. H., Bwibo N. O., Demment M. W., Murphy S. P. and **Neumann, C. G. (2003)**: Kenyan School Children Have Multiple Micronutrient Deficiencies, but Increased Plasma

Vitamin B-12 is the Only Detectable Micronutrient Response to Meat or Milk Supplementation. Journal of Nutrition, **133**(11 SUPPL. 2), 3972–3980. https://doi.org/10.1093/jn/133.11.3972s.

Staal S., Poole J., Baltenweck I., Mwacharo J., Notenbaert A., Randolph T., Thorpe W., Nzuma J. and **Herrero M. (2009)**: Targeting strategic investment in livestock development as a vehicle for rural livelihoods (Jamison D.T., Henry M.W., Measham A.R. and Bobadilla J.L.(eds.)). Oxford University Press.

Stewart C. P., Iannotti L., Dewey K. G., Michaelsen K. F. and **Onyango A. W. (2013)**: Contextualizing complementary feeding in a broader framework for stunting prevention. Maternal and Child Nutrition, 9(S2), 27–45. https://doi.org/10.1111/mcn.12088.

Tako E., Ferket P. R. and **Uni Z.** (**2004**): Effects of in ovo feeding of carbohydrates and β-hydroxy-β- methylbutyrate on the development of chicken intestine. Poultry Science **83**(12), pp. 2023–2028. doi: 10.1093/ps/83.12.2023.

Thumbi S. M., Njenga M. K., Marsh T. L., Noh S., Otiang E., Munyua P., Ochieng L., Ogola E., Yoder J., Audi A., Montgomery J. M., Bigogo G., Breiman R. F., Palmer G. H. and **McElwain T. F. (2015)**: Linking human health and livestock health: A "one-health" platform for integrated analysis of human health, livestock health, and economic welfare in livestock dependent communities. PLoS ONE, **10**(3), 1–18. https://doi.org/10.1371/journal.pone.0120761.

UNDP. (2020): Charting pathways out of multidimensional poverty: Achieving the SDGs. http://hdr.undp.org/sites/default/files/2020_mpi_report_en.pdf.

Valdivia C. (**2001**): Gender, Livestock Assets, Resource Management, and Food Security: Lessons from the SR-CRSP. Agriculture and Human Values **18**(1):27-39. doi: 10.1023/A.

Van Boven M., Bouma A., Fabri T. H. F., Katsma E., Hartog L., and **Koch G. (2008)**: Herd immunity to Newcastle disease virus in poultry by vaccination. Avian Pathology, **37**(1), 1–5. https://doi.org/10.1080/03079450701772391.

Wong J. T., de Bruyn J., Bagnol B., Grieve H., Li M., Pym R. and **Alders R. G. (2017)**: Small-scale poultry and food security in resource-poor settings: A review. Global Food Security, **15**(March), 43–52. https://doi.org/10.1016/j.gfs.2017.04.003.

World Health Organization. (2018): Global Nutrition Targets 2025 to improve maternal, infant and young child. In World Health Organization (Vol. 2, Issue **6**). https://doi.org/10.2165/00024677-200302060-00002.

World Organisation for Animal Health (2012): Terrestrial Animal Health Code. OIE, Paris.

Young M., Alders R., Grimes S., Spradbrow P., Dias P., Silva A. and **Lobo Q. (2002)**: Controlling Newcastle Disease in Village Chickens: A Laboratory Manual.

Zeleke A., Sori T., Gelaye E. and **Ayalet G. (2005)**: Newcastle Disease in Village Chickens in the Southern and Rift Valley Districts in Ethiopia. International Journal of Poultry Science, **4**(7), 507–510.

APPENDICES

Appendix 1: Consent form

Impact of ND vaccination on productivity of indigenous chicken and association with child nutrition in Siaya county, Kenya

Investigator: Elkanah Otiang, KEMRI/Centre for Global Health Research (CGHR)

Introduction

We are from the KEMRI/CGHR, and WSU. We are doing a research study on childhood nutrition by targeting Newcastle disease control in chicken. When this disease is controlled, chicken tend to grow faster, multiply better, produce more eggs and your family then has a better chance to eat more chicken meat, eggs, has more chicken to sell and use the money for other family need such as, paying school fees, buying medicines, or going to seek care in hospitals when sick. We would like to try to find out factors that influences uptake of Newcastle disease vaccination, which when done, chicken tend to survive in more numbers and produce, implying a better livelihood for your family especially in nutrition of children, their growth and development. To do this, we will ask questions relating to chicken keeping, what your children feed on, get their weights, heights, and mid-arm circumference as well as their mother's weights and heights. We will enrol all your chicken and children; every month follow up on chicken and after three months on children, collect samples from 3 chicken when they are sick or dying in our visits and ask questions for two years. As this will be happening, socio-economic survey conducted by PBASS teams will be ongoing. Only blood, cloacal and oropharyngeal swabs samples will be collected. The questions, obtaining the measures from children –mothers and sampling might take a bit of your time. Information you give about your household, children and your chicken will be kept private as required by the law. No names will be used on any of the study reports. The choice to enrol your chicken and children in this study is completely voluntary, and you may refuse to join now or withdraw from the study anytime without any consequences on you, your children or chicken.

Benefits from being in the study

Your household will continue receiving free access to veterinary care for cattle, sheep, goats, and chicken from the PBASS and your household occupants' free medical attention at the Lwak Hospital (St. Elizabeth). During our investigation, all your chicken will have their parasites controlled, those in the intervention arm will get regular Newcastle vaccination, while those in the control arm will not but at the end of the study period. Children found to be suffering from malnutrition will get referred to Lwak Hospital (St. Elizabeth) or nearby Government health facility for appropriate intervention. Scientific knowledge gathered will help understand factors that influence Newcastle disease control, improve the control, and reflect significance chicken play in livelihoods of households that keep them.

Risks from being in the study

If you agree to participate in the study, we may ask you or members of your household to help with restraining the chicken during our visits and holding children when taking measurements. This may expose you or your family members to risk of injury from chicken scratch and stress them. Chicken may get stressed and drawing of blood may cause brief pain. Every care will be taken to minimize this stress, pain, bruising and bleeding. Sampling and obtaining children measures may take some time, as will answering the questions about chicken and children nutrition.

We would also like to ask your permission to store the samples from your chicken, and to perhaps carry out other tests on these samples later. These tests might be done in a laboratory here in Kenya or one overseas.

Persons to Contact

Deciding whether to be in the study today is your choice. You can choose not to join, or to drop out at any stage. If you want to discuss this study with a veterinarian not involved in the study, contact Dr Mark Otieno, Sub-County veterinary officer, Rarieda on 0713-704-965 or a medical doctor, Dr Eric Osoro, MOH, Zoonotic Disease Unit (ZDU) in Nairobi on 0722-216-391. Should any more questions arise or if you feel like you, your family or your chicken might have been harmed by being in the study, or if you have any questions about this study or later decide that you don't want your chicken's samples stored or have questions about the research, please contact Dr Elkanah Otiang at the Integrated Human Animal Health Program (IHAHP) office in Lwak on 0724-430-387. If you have any questions about your rights as research participants, please contact the secretary, KEMRI/National Emergency Response Committee (NERC) (tel. 0202722541 or 0722205901 or 0733400003); **P.O Box 54840-00200, Nairobi-Kenya, E-mail: ERCAdmin@kemri.org**

The consent form has been explained to me and I agree for my household and chicken to take part in the study. I have been told that I am free to choose not to take part in this study at any time and that saying "NO" will have no effect on my family or me and will not affect my participation in other studies.

Head of household	Name………………….	Signature: …………………	**Date**□□/□□/□□
Witness*	Name………………….	Signature: ……...................	**Date**□□/□□/□□

* Subject may sign or provide verbal consent and thumbprint in the presence of a witness. The witness (by his/her signature) verifies that the consent form has been accurately read to the subject and this is the subject's signature or that he/she has provided verbal consent.

Appendix 2: Household Recruitment and monthly visit questionnaires

Household information

Compound ID code ________/________/________

Household ID code _______/________/_________

Interviewer Date _______________

Head of Compound Name (First, middle and last)

__________/______________/__________

Respondent Name (First, middle and last)

_______________/______________/____________

Household eligibility

Is the Household Eligible?

- ☐ Yes
- ☐ No

If NO, Reason for ineligibility ________

- ☐ Refused consent
- ☐ No Adult Occupier > 16 years
- ☐ No longer in International Emerging Infections Program (IEIP)
- ☐ Withdrawal
- ☐ No local chicken at recruitment
- ☐ No child between 6 months-3 years at recruitment/ below 5 years for follow up
- ☐ Other Reason (specify)_____________

Is the respondent the Compound Head?

- ☐ Yes
- ☐ No

If NO, what is the relationship of the respondent to the household head?

- ☐ Spouse
- ☐ Son
- ☐ Daughter
- ☐ Brother
- ☐ Sister
- ☐ Uncle
- ☐ Aunt
- ☐ Nephew
- ☐ Niece
- ☐ Other (specify)_____________

Is the respondent the primary chicken caretaker?

- ☐ Yes
- ☐ No

Primary chicken caretaker's sex

- ☐ Male
- ☐ Female

Chicken Ownership

Do you have indigenous chicken today?

- ☐ Yes
- ☐ No

If Yes, break down as.

Chicken category	Number Owned
☐ Chicks (<3 months)	_____________
☐ Growers (3-9 months)	______________
▪ Hens	_____________

- Cockerels ____________

☐ Adults (>9 months)

- Hens ____________
- Cockerels ____________

☐ **Total__________**

If No for Monthly follow up visits, but indigenous chicken available at enrolment/beginning of the study, what happened and how many?

	<3 months	3-9 months	<9 months	Total
<u>Categories</u>		Hens/Cockerels (separate boxes)	Hens/cockerels (separate boxes)	
☐ Died of disease				
☐ Sold				
☐ Slaughtered				
☐ Given out as gifts				
☐ Killed by animals				
☐ Others? Specify______				
Total				

number **OUT**				

Do you keep any other poultry type?

☐ Yes ☐ No

If Yes, which one and how many?

☐ Improved kienyeji______

☐ Layers_________

☐ Broilers______

☐ Ducks________

☐ Pigeons________

☐ Geese______

☐ Turkey_____

☐ Quill________

☐ Guinea fowls_______

☐ Other (specify) ________, how many? ______

Chicken Husbandry and Management

How are your chicken managed during the day?

☐ Free ranging

☐ In human house

☐ In a coop/cage

☐ Other (specify)________

How are your chicken managed at night?

☐ No specific housing

☐ In resided human house

☐ In non-resided human housing e.g., kitchen

- ☐ In a coop/cage
- ☐ Other (specify)__________

In case of mixed poultry keeping: Do you house all your poultry together?

- ☐ Yes
- ☐ No

How do you feed your indigenous chicken?

- ☐ Free ranging only
- ☐ Feed supplementation only
- ☐ Free ranging and feed supplementation
- ☐ Other(specify) __________

(If they provide supplements). What supplements do you provide?

- ☐ Commercialized feeds
- ☐ Crop residues e.g., grains
- ☐ Fish meal
- ☐ Minerals
- ☐ Concentrates
- ☐ Fermented brew
- ☐ Ants/termites
- ☐ Other (specify)___________

How does your chicken access drinking water??

- ☐ Chicken obtaining from free raging
- ☐ Provided at the household in container
- ☐ Any other(specify)___________

Have you accessed chicken veterinary services in the last 1month?

- ☐ Yes

☐ No

Are there any preventive treatments/vaccinations on chicken carried out in your household in the last 1 month?

☐ Deworming?

What drugs?

1) ______________________

2) ______________________

3) ______________________

4) Not known

☐ Dusting for ectoparasite control beside the study's one?

What drug?

1) ______________________

2) ______________________

3) ______________________

4) Not known

☐ Vaccination beside the study's one?

Against what infections?

1) ______________________

2) ________________________

3) ________________________

4) Not known

☐ Drugs given to prevent infections (Prophylactic treatment)?

- Antibiotics
- Antifungal
- Antiviral

- Antiprotozoans
- Antinflammatory
- Herbal
- Any other ?

Specify ___________?

1) _________________

2) _________________

3) _________________

Chicken Production and Survival Details

Do you have any chicken of laying age?

☐ Yes

☐ No

If Yes, how many? __________

Do you have active laying chicken in your flock?

☐ Yes

☐ No

If Yes, how many chickens? _________Eggs/week? __________

Eggs/month (populate)? ___________

What is the age of a chicken at first laying? __Months

☐ 4

☐ 5

☐ 6

☐ 7

☐ 8

☐ 9

- ☐ 10
- ☐ 11
- ☐ 12
- ☐ Over 12

How long would it take a chicken to lay from hatching? __ Months

- ☐ Less than 1 month
- ☐ 2-3
- ☐ 4-5
- ☐ 6-7
- ☐ 8-9
- ☐ 10-11
- ☐ Over 12
- ☐ D/K

Have any of your chicken fallen sick in the last one month?

- ☐ Yes
- ☐ No
- ☐ Don’t know

If Yes, how old is/was the chicken? And how many?

- ☐ Chicks(<3months) ________
- ☐ Growers (3-9months)
- ▪ Hens ________
- ▪ Cockerels______
- ☐ Adults(>9months) ______
- ▪ Hens ________
- ▪ Cockerels_____

Total_________

What clinical signs are/were the chicken(s) exhibiting?

- ☐ Stargazing (looking skyward) ______(Yes/No)
- ☐ Torticollis _______(Yes/No)
- ☐ Staggering(ataxia)______(Yes/No)
- ☐ Inability to walk (Paralysis) _______(Yes/No)
- ☐ Head swelling______(Yes/No)
- ☐ Discoloration of the head_______(Yes/No)
- ☐ Diarrhoea_______(Yes/No)
- ☐ Nasal discharge_______(Yes/No)
- ☐ Oral discharge_______(Yes/No)
- ☐ Coughing_______(Yes/No)
- ☐ Difficulty breathing______(Yes/No)
- ☐ Pox lesions_______(Yes/No)
- ☐ Decrease in egg production______(Yes/No)
- ☐ Cessation of egg production_______(Yes/No)
- ☐ Depression_______(Yes/No)
- ☐ Loss of appetite_______(Yes/No)
- ☐ Ballooning_______(Yes/No)
- ☐ Loss of feathers_______(Yes/No)
- ☐ Cloacal blockage_________ (Yes/No)

Has any of your chicken died in the last 1 month?

- ☐ Yes
- ☐ No
- ☐ Don't know

How old is/was the chicken? And how many?

- ☐ Chicks (<3months) _______
- ☐ Growers (3-9months)
  - Hens_______
  - Cockerels_______
- ☐ Adults(>9months)
  - Hens_____
  - Cockerels_____

Total_________

Did the chicken(s) exhibit any signs prior to death?

- ☐ Yes
- ☐ No
- ☐ Don't know

If Yes, what signs?

- ☐ Stargazing (looking skyward) ______(Yes/No)
- ☐ Torticollis _______(Yes/No)
- ☐ Staggering(ataxia)______(Yes/No)
- ☐ Inability to walk (Paralysis) ______(Yes/No)
- ☐ Head swelling______(Yes/No)
- ☐ Discoloration of the head______(Yes/No)
- ☐ Diarrhoea______(Yes/No)
- ☐ Nasal discharge_______(Yes/No)
- ☐ Oral discharge_______(Yes/No)
- ☐ Coughing_______(Yes/No)
- ☐ Difficulty breathing_______(Yes/No)

- ☐ Pox lesions_______(Yes/No)
- ☐ Decrease in egg production________(Yes/No)
- ☐ Cessation of egg production_______(Yes/No)
- ☐ Depression_______(Yes/No)
- ☐ Loss of appetite_______(Yes/No)
- ☐ Ballooning_______(Yes/No)
- ☐ Loss of feathers_______(Yes/No)
- ☐ Cloacal blockage_________ (Yes/No)

Did you have chicken that were sick but didn't die?

- ☐ Yes
- ☐ No
- ☐ Don't know

How old is/was the chicken? And how many?

- ☐ Chicks(<3months) ________
- ☐ Growers (3-9months)
  - Hens________
  - Cockerels_______
- ☐ Adults(>9months)
  - Hens_______
  - Cockerels_______

Total_________

In the last one month, fill in the changes in chicken ownership by the household for different age categories.

	<3 months	3-9 months	<9 months	Total
<u>Categories</u>		Hens/Cockerels	Hens/cockerels	

		(separate boxes)	(separate boxes)	
Hatched in HH				
Bought				
Received as gifts				
Total number IN				
Slaughtered and consumed				
Sold				
Died of disease and consumed				
Died of disease and not consumed				
Total number OUT				

How do you normally dispose dead chicken?

- ☐ Eaten by family members
- ☐ Eaten by dogs
- ☐ Buried/thrown in pit latrine
- ☐ Left out to rot
- ☐ Sold
- ☐ Others(specify)_________

Appendix 3: Clinical and Post-mortem visit questionnaires

NB. For flock mortalities and morbidities, a maximum of 3 chickens will be sampled (2 live and 1 dead). No randomization for picking. Pick from each age category.

Household Information

Village code____

Compound ID code ______/______/______

Household ID code _____/______/_______

Interviewer Date __________

Chicken Sickness and Death Information

What is the cause of response?

☐ Sickness

☐ Death

☐ Sickness and death

If Sickness, how old is/was the chicken? And how many?

☐ Chicks (<3months) ______

☐ Growers (3-9months)

▪ Hens

▪ Cockerels______

☐ Adults (>9months) ______

▪ Hens _______

▪ Cockerels_______

Total________

Did the chicken(s) exhibit any signs prior to death?

- ☐ Yes
- ☐ No
- ☐ Don't know

If Yes, what clinical signs are/were the chicken(s) exhibiting?

- ☐ Stargazing (looking skyward) _____(Yes/No)
- ☐ Staggering(ataxia)_______ (Yes/No)
- ☐ Torticollis _______(Yes/No)
- ☐ Inability to walk (Paralysis) ______(Yes/No)
- ☐ Head swelling_______(Yes/No)
- ☐ Discoloration of the head______(Yes/No)
- ☐ Diarrhoea______(Yes/No)
- ☐ Nasal discharge______(Yes/No)
- ☐ Oral discharge_______(Yes/No)
- ☐ Coughing_____(Yes/No)
- ☐ Difficulty breathing______(Yes/No)
- ☐ Pox lesions______(Yes/No)
- ☐ Decrease in egg production______(Yes/No)
- ☐ Cessation of egg production_______(Yes/No)
- ☐ Depression_______(Yes/No)
- ☐ Loss of appetite_______(Yes/No)
- ☐ Ballooning_______(Yes/No)
- ☐ Loss of feathers_______(Yes/No)
- ☐ Cloacal blockage_________ (Yes/No)

If dead, how old is/was the chicken? And how many?

- ☐ Chicks (<3months) ______

- ☐ Growers (3-9months)
  - ▪ Hens_____
  - ▪ Cockerels______
- ☐ Adults (>9months)
  - ▪ Hens_____
  - ▪ Cockerels_____

Total______

Did the chicken(s) exhibit any signs prior to death?

- ☐ Yes
- ☐ No
- ☐ Don’t know

If Yes, what signs?

- ☐ Stargazing (looking skyward) _____(Yes/No)
- ☐ Staggering(ataxia)_______ (Yes/No)
- ☐ Torticollis _______(Yes/No)
- ☐ Inability to walk (Paralysis) ______(Yes/No)
- ☐ Head swelling_______(Yes/No)
- ☐ Discoloration of the head______(Yes/No)
- ☐ Diarrhoea______(Yes/No)
- ☐ Nasal discharge______(Yes/No)
- ☐ Oral discharge_______(Yes/No)
- ☐ Coughing_____(Yes/No)
- ☐ Difficulty breathing______(Yes/No)
- ☐ Pox lesions______(Yes/No)
- ☐ Decrease in egg production______(Yes/No)

- ☐ Cessation of egg production_______(Yes/No)
- ☐ Depression_______(Yes/No)
- ☐ Loss of appetite_______(Yes/No)
- ☐ Ballooning_______(Yes/No)
- ☐ Loss of feathers_______(Yes/No)
- ☐ Cloacal blockage_________ (Yes/No)

Is post-mortem being done?

- ☐ Yes
- ☐ No

If No, what are the reasons?

- ☐ Reported after 24 hours after it occurred
- ☐ Chicken not available
- ☐ Carcass not available
- ☐ Carcass interference
- ☐ Traumatic injury
- ☐ Other, specify______

Post-mortem Form

Event ID________________

Part I: General Observation

Date/time of exam: ___________________________

Date/time of death: ___________________________

Hours since death: ____________________________

Flock information

Number of chickens in flock: _________________

Number dead: _______________________________

Number sick: ________________________________

Number healthy: _____________________________

Other poultry type in flock:

Ducks () Pigeons () Geese () Turkey () Quills () Layers () Broilers () Improved Kienyeji ()

Guinea fowls () Other: ___________________________________

Clinical History and Observation

History of disease

- ☐ Yes
- ☐ No

If Yes,

- ☐ Less than a day
- ☐ 1-3 days
- ☐ 4-7 days

Do you suspect poisoning?

- ☐ Yes
- ☐ No

State of carcass

- ☐ Normal in size and weight
- ☐ Thin
- ☐ Dehydrated
- ☐ Ectoparasites present
- ☐ Exudates from openings
- ☐ Traumatic wounds
- ☐ Others, specify___________

Part 2: Gross findings per system

a) Integumentary ______ (YES/NO)

☐ Feather loss

☐ Follicular enlargement

☐ Bruises/Wounds

☐ Pox lesions

☐ Ectoparasites

☐ Others________________________________

b) Respiratory (YES/NO)

☐ Tracheal froth

☐ Tracheal haemorrhages

☐ Bronchi froth

☐ Lung emphysema

☐ Lung oedema

☐ Lung haemorrhages

☐ Worms

☐ Other___________

c) Digestive ______ (YES/NO)

☐ Esophageal haemorrhages

☐ Crop haemorrhages

☐ Proventriculus haemorrhages

☐ Mucus in proventriculus

☐ Gizzard haemorrhages

☐ Intestinal haemorrhages

☐ Hepatomegaly

☐ Caecal haemorrhages

☐ Pancreatitis

☐ Cloacal haemorrhages

☐ Worms

☐ Other____________________________

d) Lymphatic_____ (YES/NO)

☐ Splenomegaly

☐ Inflammation of bursa of *Fabricius*

☐ Mesenteries lymphadenopathy

☐ Other____________________________

e) Urogenital_____ (YES/NO)

☐ Nephritis

☐ Other____________________________

f) Cardiovascular_____ (YES/NO)

☐ Heart hemorrhages

☐ Other______________________

g) Musculoskeletal_____ (YES/NO)

☐ Hemorrhages in breast muscles

☐ Hemorrhages in thigh muscles

☐ Excess fluid accumulation in joints

☐ Other_________________________

h) Nervous _____ (YES/NO)

☐ Inflammation of sciatic nerve

☐ Inflammation of neck and abdominal nerves

☐ Other_________________________

Part 3: Tentative diagnoses

- ☐ Newcastle disease
- ☐ Infectious bursal disease (Gumboro disease)
- ☐ Fowl pox
- ☐ Pullorum
- ☐ Fowl typhoid
- ☐ Infectious bronchitis
- ☐ Coccidiosis
- ☐ Infectious coryza
- ☐ Environmentally acquired Pneumonia
- ☐ Starvation
- ☐ Non-classified septicaemia/bacteraemia
- ☐ Parasitism
- ☐ Poisoning
- ☐ Mycoplasmosis
- ☐ Other

1) ________________________

2) ________________________

3) ________________________

Appendix 4: Nutritional visit questionnaires

Demographics

Household ID: _______________________ Individual ID: ___________________

Name of parent/guardian: (First, Middle and Last) ____/____/________

Education level of the parent/guardian?

☐ No formal Education

☐ Primary school

▪ Primary education not completed

▪ Primary education completed

☐ Secondary school

▪ Secondary education not completed

▪ Secondary education completed

☐ College-graduate

☐ Other

☐ Don't Know

Name of child1: (First, Middle and Last) _________/_____/______

Age of child: _____________(months)

Name of child2: (First, Middle and Last) _________/_____/______

Age of child: _____________(months)

Interview date: ______________________

Visit number: _________________ Visit attempt number: ____________

For Children under 5:

<u>Allow for up to two children</u>

1) Is this child breastfeeding?

☐ Yes

☐ No

If no, skip to 3.

2) Does, the child feed on:

2a) Breast milk only? _________

☐ Yes (if yes, END)

☐ No

2b) Breast milk how often per day? __________ (numeric 1-20)

2c) Baby formula only instead of breast milk? _______

☐ Yes

☐ No (if yes go to 2d if no go to 2e)

2d) How often per day? __________ (numeric 1-20)

2e) Both breast milk and baby formula _________

☐ Yes

☐ No (if yes go to 2f if no go to 3)

2f) How often per day? ___________ (numeric 1-20)

In the last 3 days, what have you fed this child: If check "yes" for any, prompt to answer "How many times": numeric 1-100?

Group 1

☐ Eggs, frequency__? How many (number of eggs) ____?

☐ Of consumed, how many were from?

- Chicken____
- Other poultry___

☐ Milk only, frequency__? How many litres (example cups;1, 2, 3, 4, 5) ____?

- Milk in tea taken____
- Milk in porridge taken____

- Milk in other forms____

- ☐ Poultry meat, frequency__? What amount (pieces; gm) __?
- ☐ Fish, frequency___? What amount (size; a sample of piece of wood size for big fish and tablespoon for small fish gm) ___?
- ☐ Meat from other domestic animals, frequency__? What amount (pieces gm) ___?
- ☐ Plant proteins (Beans/peas/green grams), frequency___?
- ☐ Others (specify) _________, frequency? (Allow for options of 3 in order of frequency

Group 2

- ☐ Porridge, frequency___?
- ☐ Ugali/maize, frequency___?
- ☐ Potatoes, frequency____? Bread/Chapati/Mandazi, frequency__?
- ☐ Others (specify) ___________ frequency? (Allow for options of 3 in order of frequency)

Group 3

- ☐ Greens, frequency____?
- ☐ Squash, frequency____?
- ☐ Pumpkin, frequency___?
- ☐ Cabbage, frequency___?
- ☐ Tomatoes, frequency___?
- ☐ Carrots, frequency___?
- ☐ Others (specify) ___________ frequency? (Allow for options of 3 in order of frequency)

Group 4

- ☐ Mangoes, frequency____?
- ☐ Bananas, frequency____?
- ☐ Oranges, frequency____?
- ☐ Guavas, frequency____?

- ☐ Pawpaw, frequency____?
- ☐ Avocado frequency____?
- ☐ Others (specify) __________ frequency___? (Allow for options of 3 in order of frequency)

Measurements

In the clinical signs section: For child (children)

Child weight: in kg (1-30kgs) ________

Child mid-upper arm circumference: in cm (6-26) ________

- ☐ Green
- ☐ Yellow
- ☐ Red

Child length: in cm (30-120cm) _______

OR

Child height: in cm (30-120cm) _______

In the clinical signs section: For mother (mothers)

Mother’s height: in cm (120-200cm) ______

Mother’s weight: in kg (40-110kgs) ______

Child mid-upper arm circumference: colour drop down menu; in cm (20-40)

- ☐ Green
- ☐ Yellow
- ☐ Red

Household Feeding Patterns

Who decides what your household eats?

- ☐ Father
- ☐ Mother

☐ Son

☐ Daughter

☐ Uncle

☐ Aunt

☐ Nephew

☐ Niece

☐ Daughter-in-law

☐ Other (specify)__________

Who routinely prepares what the household eats?

☐ Father

☐ Mother

☐ Son

☐ Daughter

☐ Uncle

☐ Aunt

☐ Nephew

☐ Niece

☐ Daughter-in-law

☐ Other (specify)__________

What influences the entire family feeding patterns?

☐ Availability of a particular food in the household

☐ Availability of a particular food in the market

☐ Sickness of a family member

☐ Food offering a balanced diet

☐ Cost

- ☐ Season
- ☐ Random decision
- ☐ Other (specify)________

What influences what the child feeds on?

- ☐ Availability of a particular food in the household
- ☐ Availability of a particular food in the market
- ☐ Sickness of the child
- ☐ Food offering a balanced diet
- ☐ Age of the child
- ☐ Cost
- ☐ Season
- ☐ Random decision
- ☐ Other (specify)________

Appendix 5: Socio-Economic surveillance questionnaire

Visit Number: ___________

Household Information

Compound ID code ___/___/___

Household ID code ___/___/___

Interviewer Date ___________

Head of Compound Name (First, middle and last) __/____/_____

Respondent Name (First, middle and last) ___/_____/________

Is the respondent the Compound Head?

☐ Yes

☐ No

What is the relationship of the respondent to the household head?

☐ Head

☐ Spouse

☐ Son

☐ Daughter

☐ Brother

☐ Sister

☐ Uncle

☐ Aunt

☐ Nephew

☐ Niece

☐ Other (specify)__________

Primary chicken caretaker's sex

☐ Male

☐ Female

Primary chicken caretaker's name (First, middle and last) ___/____/___

Is the respondent the primary animal caretaker?

☐ Yes

☐ No

If no, specify relationship of the caretaker with the household

☐ Family member

☐ Employee/

☐ Other (Specify) ______________________

Household eligibility

Is the Household Eligible?

☐ Yes

☐ No

If NO, Reason for ineligibility _________

☐ Refused consent

☐ No Adult Occupier > 16 years

☐ No longer in IEIP

☐ Withdrawal

☐ No local chicken

☐ No child below 5 years

☐ Other Reason (specify)_________

Household Demographics

a) How many members greater than 10 years of age currently live in the household __(numeric)

b) For each of the first 5 members provide details below:

I. Member 1- First, second and last name ___/___/___

II. Member 1- Date of birth ___/___/__ __ Member Age ___ (autofill from date of birth)

III. Member 1- Gender _____(Male/Female)

IV. Member 1- Education level _______ (No formal Education/Primary school/Secondary school/College-graduate/Other/Don't Know)

V. Marital status _______ (Single/Married/Polygamous/Divorced/Separated/Widow(er))

VI. Family role _________ (Household head/Spouse/Son or daughter/Sibling/Father or Mother/Nephew or niece/Grandson or daughter/Other)

VII. Village role ________ (Chief/Asst. Chief/Elder/Village Reporter/None/Other)

VIII. Primary Occupation________ (Employed full time on farm/Employed part time on farm/Self-employed off farm/Employed off farm Agriculture/Salaried off farm/Other)

IX. Secondary Occupation __________ (Employed part time on farm/Self-employed off farm/Employed off farm Agriculture/Salaried off farm/Other)

c) What is the total number of male children below 5 years in this household? ______

d) How many of these male children are below 3 years? ___________

e) What is the total number of female children below 5 years in this household? ______

f) How many of these female children are below 3 years? ___________

g) What is the total number of male children between 5 – 10 years in the household? ___

h) What is the total number of female children between 5 – 10 years in the household? _

i) Total number of household members ___________ (autofill)

Household Assets Information

Does the household currently own a plough or other farm implements?

☐ Yes

☐ No

☐ Don't know

If YES,

i) Number of draft implements (plough) in usable conditions __________

ii) What market value would you expect if you bought or sold these draft implements today?

________ (Value in Kshs.)

iii) Number of hand implements (pangas/jembes/rakes/slashes/) in usable conditions___

iv) What market value would you expect if you bought or sold these hand implements today?

________ (Value in Kshs.)

Does the household currently own Bicycle(s)?

☐ Yes

☐ No

☐ Don't Know

If YES,

i) Number of bicycles in usable condition__________

ii) What market value would you expect if you bought or sold all these bicycle(s) today?

________ (value in Kshs.)

Does the household currently own Vehicle(s)?

☐ Yes

☐ No

☐ Don't Know

If YES,

i) Number of Vehicles in usable condition __________

ii) What market value would you expect if you bought or sold all these Vehicle(s) today?

________ (value in Kshs.)

Does the household currently own Radio(s)?

☐ Yes

☐ No

☐ Don't Know)

If YES, i) Number of Radios in usable condition _________

ii) What market value would you expect if you bought or sold the Radio (s) today? _______

(value in Kshs.)

Does the household currently own Tractor(s)?

☐ Yes

☐ No

☐ Don't Know

If YES,

i) Number of Tractors in usable condition _________

ii) What market value would you expect if you bought or sold the Tractor(s) today? _______

(value in Kshs.)

Does the household currently own Mobile Phone(s)?

☐ Yes

☐ No

☐ Don't Know

If YES,

i) Number of Mobile Phones in usable condition _________

ii) What market value would you expect if you bought or sold the Mobile Phone(s) today?

_______ (value in Kshs.)

Does the household currently own Motorcycle(s)?

☐ Yes

☐ No

☐ Don’t Know

If YES,

i) Number of Motorcycle(s) in usable condition _________

ii) What market value would you expect if you bought or sold the Motorcycle(s) today? _______ (value in Kshs.)

Does the household currently own Television(s)?

☐ Yes

☐ No

☐ Don’t Know

If YES,

i) Number of Televisions in usable condition _________

ii) What market value would you expect if you bought or sold the Television(s) today? _______ (value in Kshs.)

Does the household currently own Computer(s)?

☐ Yes

☐ No

☐ Don’t Know

If YES,

i) Number of Computer (s) in usable condition _________

ii) What market value would you expect if you bought or sold the computer(s) today? _______ (value in Kshs.)

Does the household currently own Television(s)?

☐ Yes

☐ No

☐ Don't Know)

If YES,

i) Number of Televisions in usable condition _________

ii) What market value would you expect if you bought or sold the Television(s) today? _______ (value in Kshs.)

Does the household currently own other electronics e.g., fridges, microwaves?

☐ Yes

☐ No

☐ Don't Know)

If YES,

i) Number of units in usable condition _________

ii) What market value would you expect if you bought or sold these electronics today? _______ (value in Kshs.)

Number of mud wall grass thatched buildings __________

Number of mud Walls and Iron roof buildings __________

Number of mud Walls plastered Iron roof buildings __________

Number of stones, brick, or concrete wall buildings__________

Number of other types of buildings _____________

Latrine ___________

☐ Indoor

☐ Outdoor

☐ Both

☐ None

Electricity __________

☐ Yes

- [ ] No

What is the primary source of drinking water ______?

- [ ] Communal Borehole
- [ ] Communal Dam or Borehole
- [ ] Rainwater or seasonal river
- [ ] Private Borehole
- [ ] Public or private tap water
- [ ] Lake
- [ ] Well
- [ ] Spring

How much time per day per household is spent collecting water?

- [ ] Tap at household
- [ ] Less than 1 hour
- [ ] 1-2 hours
- [ ] More than 2 hours

How many times per day does a household member collect water? ______________

What is the primary source of cooking fuel/energy?

- [ ] Electricity
- [ ] Natural Gas
- [ ] Kerosene
- [ ] Firewood
- [ ] Charcoal
- [ ] Cow Dung
- [ ] Other(specify)____

If you are using firewood, where do you get it?

- ☐ Buy
- ☐ Collect
- ☐ Don’t Know

How much time per day per household is spent collecting firewood?

- ☐ Less than 1 hour
- ☐ 1-2 hours
- ☐ More than 2 hours

Which Household Member(s) Collect Firewood?

- ☐ Father
- ☐ Mother
- ☐ Children
- ☐ Relatives
- ☐ Not collected
- ☐ Others(specify) ____________

For each of the first 5 members, provide information on the following:

- ☐ Off-Farm Net Income over the last 3 months ________ (Kshs.)
- ☐ Time spent per week earning this income ________ (average hours/week over last 3 months)
- ☐ Time spent per week working off the farm in trade for goods hours/week over last 3 months)
- ☐ Sum of off-compound labour income for the last 3 months from all children 10 and younger: _________(Kshs/3months)
- ☐ Sum of time spent by all children 10 and younger working off-compound for cash income___ (average hours/week over last 3 months)
- ☐ Sum of time spent by all children 10 and younger working off-compound in trade for goods ________ (average hours/week over last 3 months)

For each child aged 6-20, is this child enrolled in school?

- ☐ Yes
- ☐ No

If yes, what grade does this child attend?

- ☐ standard 1 to 5
- ☐ standard 6 to 8
- ☐ Secondary level

If not, why is this child not enrolled in school?

- ☐ The child does not like it
- ☐ The child is not performing well
- ☐ The child has behavioural problems
- ☐ The child is required to work
- ☐ The family cannot afford fees
- ☐ The child is sick
- ☐ Other(specify) __________

Household total off-compound labour income over the last 3 months __________ (Kshs.)

Household total time spent per week for cash income off-compound _______________ (average hours/week over last 3 months)

Household total time spent per week working off-compound in trade for goods and services ________ (average hours/week over last 3 months)

Does any household member maintain savings Account/Mpesa/Airtel Money/MShwari/Table banking, commercial bank, or any mobile money transfer account?

- ☐ Yes
- ☐ No
- ☐ Don't Know

If Yes,

What is the current Total Household saving balance ___________?

- ☐ < Kshs 7,000
- ☐ 7000 -17, 500
- ☐ 17,500 – 35,000
- ☐ > 35,000
- ☐ Don't Know

Has any household member obtained a loan in the last 3 months?

- ☐ Yes
- ☐ No
- ☐ Don't Know

If Yes,

Why was the loan taken? _________

☐Business

☐School expenses

☐Health expenses

☐Household purchase

☐Livestock

☐Other (specify) ___

☐Don't know

Household Consumption and Expenditure

Average Household food consumption over the last seven days

i) Poultry Eggs consumed?

- ☐ Yes
- ☐ No

If Yes,

a) Quantity produced home (Number/week) _______

b) Quantity purchased (Number/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Number/week) ______

ii) Poultry Meat consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

iii) Cow milk consumed?

☐ Yes

☐ No

If Yes,

a) Quantity of consumed milk that was produced home (Litres/week) ____

b) Quantity purchased (Litres/week) _______

c) Amount spent (Kshs/Litre) ________

d) Total quantity consumed (Litres/week) ______

iv) Goat milk consumed? _________

☐ Yes

☐ No

If Yes,

a) Quantity produced home (Litres/week) _______

b) Quantity purchased (Litres/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Litres/week) ______

v) Beef consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

vi) Sheep and Goat meat consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

vii) Fish/Omena consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

viii) Maize/Maize Meal consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

ix) Cassava (Fresh/Dry/Flour) consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

x) Millet/Sorghum?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xi) Banana (Including Plantains) consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xii) Peas/Beans/Lentils/Other pulses consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xiii) Onions/Tomatoes/Carrots/Green peppers/other vegetables (Viungo) consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xiv) Leafy Greens: Spinach, Cabbage, etc. consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xv) Sweet potatoes?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xvi) Irish Potatoes consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xvii) Cooking Fats consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Kg/week) _______

b) Quantity purchased (Kg/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Kg/week) ______

xviii) Cooking Oil consumed?

☐ Yes

☐ No

If Yes,

a) Quantity produced at home (Litres/week) _______

b) Quantity purchased (Litres/week) _______

c) Amount spent (Kshs.) ________

d) Total quantity consumed (Litres/week) ______

Has the Household received any food aid from any source in the last 3 months?

☐ Yes

☐ No

Other household expenditures

Item Amount Purchased (Kshs.)

i) Cooking Fuel ____ (Yes/No) _________

ii) Clothes ____ (Yes/No) _________

iii) Health Care ____ (Yes/No) _________

☐ Vaccinations

☐ De-wormers

□ Consultations

□ Medications (drugs/herbs)

□ Transport cost

□ Others

iv) Education ____ (Yes/No) __________

v) Other ____ (Yes/No) __________

Comments ____

Household Livestock Ownership

Report current inventory of livestock (i. e, in each cell, enter the Number of Livestock Owned by the household, on and off the compound)

Species Yes/No Number on compound Number off compound Total

- □ Cattle
  - Calves (Currently < 12 months) _____ ____________ ________ _________
  - Heifers (Currently 1-2 years) ____ ________________ _____________ _______
  - Bullocks (Currently 1-2 years) ____ ____
  - Adult bulls (Currently > 2 years) ____ ___
  - Adult cows (Currently > 2 years) _________________________ ____________________
- □ Goats _______ ____________________ _______________
- □ Sheep _______ ____________________ ___________________
- □ Poultry
  - Improved kienyeji_____________________ ______________________ ____________
  - Layers_______________________________ ______________________ ____________
  - Broilers___________________________ ______________________ ____________
  - Ducks______________________________ ______________________ ____________
  - Pigeons______________________________ ______________________ ____________

- Geese________________________ ____________________ ___________
- Turkey______________________ ____________________ ___________
- Quill___________________________ ____________________ __________
- Guinea fowls__________________________ ____________________
- Other (specify)

- ☐ Donkeys ______ ____________ __________________
- ☐ Pigs
- ☐ Rabbits
- ☐ Others (specify)___

		During last 3 months							
		Births	Deaths	Sold (live or slaughtered)	Slaughtered for home consumption	Gifts received	Gifts given away	Purchased	Lost or stolen
Cattle									
	calves (<12 months)								
	▪ Heifers (1-2 yrs.)								
	▪ Bullocks (1-2 yrs.)								
	▪ Adult bulls (>2yrs) ▪ Adult cows								

	(>2yrs)							
Goats	Total							
Sheep	Total							
Poultry	Total ▪ Improved kienyeji ▪ Layers ▪ Ducks ▪ Pigeons ▪ Geese ▪ Turkey ▪ Quill ▪ Guinea fowls ▪ Other (specify) Pigs Rabbits Others (specify)							

Do any livestock sleep in the buildings where humans sleep or cook?

Yes- check box:

☐ Cattle

☐ Sheep

☐ Goats

☐ Chickens

☐ No

☐ Unknown

Household Livestock Inventory

Report changes in Inventory in the last 3 months

How many male cattle that you own?

i) Predominantly Zebu? _______

ii) Zebu/Exotic cross? ________

iii) Other? ___________

How many female cattle that you own?

i) Predominantly Zebu? _______

ii) Zebu/Exotic cross? ________

iii) Other? ___________

What is the estimated value of livestock owned by you that were lost during the last 3 months due to drought, floods, wild animals, fire, or similar factors? (Kshs.) ________

Do you manage cattle for someone else? _________ (No/Yes)

How many cattle do you currently manage for someone else? _______

Why are you keeping these cattle? ___________

☐ On loan for breeding

☐ On loan for milk

☐ Disease management

☐ Better grazing where managed

☐ Favour provided to Family/Friends/Neighbours

☐ Other, specify ____________

Do you have cattle managed by someone else _____________ (Yes/No)?

How many cattle that you own are currently managed by someone else _______

Why are these cattle managed by someone else? ________

- ☐ On loan for breeding
- ☐ On loan for milk
- ☐ Disease management
- ☐ Better grazing where managed
- ☐ Favour provided to Family/Friends/Neighbours
- ☐ Other, specify ___________

Comments ____________________

Select any 2 adult females from your herd with at least 2 calves

i) For Cow 1, when was the last calf born? __________ (Date)

ii) For Cow 1, when was the last calving before the one mentioned above___ (Date)

iii) Estimated calving interval ____________ (Months)

iv) For Cow 2, when was the last calf born? __________(Date)

v) For Cow 2, when was the last calving before the one mentioned above _________ (Date)

vi) Estimated calving interval ____________(Months)

Income from Livestock

Enter values of on-household livestock income from the last 3 months [note the Numbers of Animals sold as in animal inventory]

Species	# sold	Amount in Kshs
a) Cattle		
i) Calves (< 12 months)	_______	______________
ii) Heifers (1-2 years)	_______	______________
iii) Bullocks (1-2 years)	_______	______________
iv) Adults bulls (> 2 years)	______	______________

v) Adults cows (> 2 years) ________ ________________

b) Goats ________ ________________

c) Sheep ________ ________________

d) Donkeys ________ ________________

e) Poultry

- Improved kienyeji
- Layers
- Ducks
- Pigeons
- Geese
- Turkey
- Quill
- Guinea fowls
- Other (specify) ________ ________________

f) Other domestic animals (specify) ________ ________________

2. Livestock products produced and sold over the last 3 months?

☐ Yes

☐ No

If YES,

a) Cattle

i) Average amount of milk produced (litres/day/herd) _________

ii) Average amount sold __________

iii) Total value sold (Kshs/day) ___________

iv) Number of cows currently producing __________

b) Goats (Milk)

i) Average amount of milk produced (litres/day/herd) _________

ii) Average amount sold _________

iii) Total value sold (Kshs/day) __________

iv) Number of shoats currently producing _________

c) Poultry (Eggs/week)

i) Average amount produced _________ (Eggs/week/flock)

ii) Average amount sold _________ (Eggs/week/flock)

iii) Total value sold (Kshs/week/flock) __________

iv) Number of poultry currently producing eggs_________

d) Other

- ☐ Yes
- ☐ No

If Yes, specify ________

i) Average amount produced _________

ii) Average amount sold _________

iii) Total value sold (Kshs/week) __________

iv) Number of currently producing _________

On-farm Expenses on Livestock

A. Livestock purchases for on-household use over the last 3 months (*Quantities listed in livestock inventory*)

Species	Yes/No	Amount in Kshs
a) Cattle		
i) Calves (< 12 months)	_______	______________
ii) Heifers (1-2 years)	_______	______________

iii) Bullocks (1-2 years) ________ ________________

iv) Adults bulls (> 2 years) ________ ________________

v) Adults cows (> 2 years) ________ ________________

b) Goats ________ ________________

c) Sheep ________ ________________

d) Donkeys ________ ________________

e) Poultry

- Improved Kienyeji
- Layers
- Ducks
- Pigeons
- Geese
- Turkey
- Quill
- Guinea fowls
- Other (specify)

f) Other domestic animals (specify) ________ ________________

B. Total Production cost for owned and managed livestock on compound in the last 3 months (Kshs/3months)

a) Expenditures for cattle management?

☐ Yes

☐ No

If YES,

i) Hired labour (herding/milking etc) __________

ii) Supplements (Salts or minerals) __________

iii) Feeds/Fodder ___________

iv) Health and vet services __________

- Acaricides
- Deworming
- Treatment e.g (Antibiotics)
- Vaccinations __________
- Lumpy skin disease
- Black quarter and anthrax
- Foot and Mouth
- Other (specify) _____________

v) Other Production Costs __________

b) Expenditures for goats/Sheep management?

- Yes
- No

If YES,

i) Hired labour (herding/milking etc) __________

ii) Supplements (Salts or minerals) __________

iii) Feeds/Fodder ___________

iv) Health and vet services __________

- Acaricides
- Deworming
- Treatment e.g. (Antibiotics)
- Vaccination
- Others

v) Other Production Costs __________

c) Expenditures for donkey management?

- ☐ Yes
- ☐ No

If YES,

i) Hired labour (herding etc) _________

ii) Supplements (Salts or minerals) _________

iii) Feeds/Fodder __________

iv) Health and vet services _________

- ☐ Acaricides
- ☐ Deworming
- ☐ Treatment e.g. (Antibiotics)
- ☐ Vaccinations
- ☐ Others

v) Other Production Costs _________

d) Expenditures for poultry management?

- ☐ Yes
- ☐ No

If YES,

i) Hired labour _________

ii) Supplements _________

iii) Feeds __________

iv) Health and vet services _________

- ☐ Deworming
- ☐ Treatment e.g. (Antibiotics)
- ☐ Vaccination

☐ Others

v) Other Production Costs __________

C. How many acres of land does the household own and not share with other livestock holders for grazing? ________

D. Does the household have access to common grazing lands?

☐ Yes

☐ No

E. If your household had better access to more grazing land, would you choose to increase your cattle herd size?

☐ Yes

☐ No

☐ Not sure

Land and Crop Inventory

Did you plant any crop(s) during the most recent crop production season?

☐ Yes

☐ No

If yes:

a. How many acres did you plant in crops for the household during the most recent crop production season? _______
b. How many of the acres are owned by you/your household? _____________
c. Is part of this land rented?

    ☐ Yes

    ☐ No

d. How many of these acres are rented? __________
e. If land is rented, what is the mode of payment

- ☐ Cash
- ☐ Crop shared
- ☐ Both
- ☐ Other

f. If cash rented, how much per (Kshs)/year? ___________

2. Have you lost any household crops due to drought, floods, wild animals, fire or any other thing in the last 3 months?

☐ Yes

☐ No

If YES, what is the estimated value of loss (Kshs.) ___________

3. Crop production cost in the last 3 months

a) Maize production?

☐ Yes

☐ No

i) Area planted (Acre) _________

ii) Date planted _________

iii) Manure used _________ (Yes/No)

iv) Any inputs purchases _________ (Yes/No)

If input purchases YES, check box if inputs purchased

- ☐ Seeds
- ☐ Fertilizer
- ☐ Herbicide
- ☐ Pesticide
- ☐ Other, Specify _______________

v) Total cost of non-household labour (similar questions for the following crops)

a. Sweet potato

b. Sorghum

c. Cassava

d. Groundnuts

e. Beans

f. Others (specify) ___________

4. On-farm Stocks: crops. Enter information on household crops over the last 3 months

a) Maize produced?

☐ Yes

☐ No

i) Acres harvested _________

ii) Date harvest completed _________

iii) Produced (Kg) _________

iv) Carryover (Kg) _________

v) Purchased (Kg) _________

vi) Gifts Received (Kg) _________

vii) Gifts Out (Kg) _________

viii) Relief Aid (Kg) _________

iv) Any inputs purchased? _________ (Yes/No)

If input purchases YES, check box if inputs purchased

☐ Seeds

☐ Fertilizer

☐ Herbicide

☐ Pesticide

☐ Other (specify) ________________

A. Total cost of non-household labour inputs (Kshs/3 months)

B. Total cost of non-household labour for crop production (Kshs/3 months) (similar questions for the following crops)

a. Sweet potato

b. Sorghum

c. Cassava

d. Groundnuts

e. Beans

f. Others (specify)

5. On-Farm Income: Crops.

(Enter values for on-farm crop production and income for the last 3 months)

Crop	Yes/No	Quantity sold (kg)	Value (Kshs/total)
Maize			
Potato			
Sorghum			
Cassava			
Beans			
Groundnuts			
Other (specify)			

Cattle Health and Care

Tick treatment

a) What method do you usually use to control ticks?

- ☐ Acaricide dip

□ Spray

□ No tick controls

□ Other (specify) ____

b) How far do you move your cattle for dipping/spraying? (Km) _____________

c) How many ticks control treatments (e.g., spraying or dipping) do you apply per month? _____ (0/1/2/3/4)

d) What is the total cost of tick control treatment (e.g., Spraying or dipping) per month for the entire cattle herd _____ (Kshs/herd/month?)

e) Have any of your animals been vaccinated against one or more of the major diseases in the last 3 months?

□ Yes

□ No

Human Health Characteristics

a) Has any household member been sick (could not work or attend school) in the last 3 months?

□ Yes

□ No

□ Don't Know

Was the household member sick a child (children below 5)?

□ Yes

□ No

If Yes, what sickness was/were they?

□ ?______

□ ?______

□ ?______

☐?______

☐ Don’t know

b) How many days of on-compound work have been missed due to illness of family members in the last 3 months (*sum for whole household)* _____________ (days)

c) How many days of off-compound work have been missed due to illness of family members in the last 3 months (*sum for whole household)* _____________

d) Has any household member visited a health clinic or hospital in the last 3 months?

☐ Yes

☐ No

e) How many visits to a health clinic or hospital were made by household members in the last 3 months ______ (total for household?)

f) How much time does it take to travel to the health canter or hospital that you use? _______(hours)

g) Are children under 5 years old in the household currently vaccinated against one or more of the major diseases?

☐ Yes

☐ No

Appendix 6: KEMRI Scientific and Ethics Review Unit (SERU) Approval

KENYA MEDICAL RESEARCH INSTITUTE

P.O. Box 54840 - 00200 NAIROBI - Kenya
Tel: (254) (020) 2722541, 254 (020) 2713349, 0722-205901, 0733-400003 Fax (254) (020) 2720030
Email: director@kemri.org info@kemri.org Website:www.kemri.org

KEMRI/RES/7/3/1 **January 27, 2016**

TO: **DR. THUMBI MWANGI,**
PRINCIPAL INVESTIGATOR

THROUGH: **DR. STEPHEN MUNGA,**
THE DIRECTOR, CGHR,
KISUMU

FORWARDED
DIRECTOR
CENTRE FOR GLOBAL HEALTH RESEARCH

Dear Sir,

RE: PROTOCOL NO. KEMRI/SERU/CGHR/026/3159 (*RESUBMISSION OF INITIAL SUBMISSION*): ENHANCING CHILDHOOD NUTRITION GROWTH THROUGH TARGETED LIVESTOCK INTERVENTIONS

Reference is made to your letter dated 7th January 2016. KEMRI/Scientific and Ethics Review Unit (SERU) acknowledges receipt of the revised protocol on the 19th January 2016.

This is to inform you that the Committee notes that the issues raised during the 245th committee A meeting of the KEMRI/Scientific and Ethics Review Unit (SERU) held on November 10, 2015 have been adequately addressed.

Consequently, the study is granted approval for implementation effective this day **27th day of January 2016** for a period of one year. Please note that authorization to conduct this study will automatically expire on **26th January, 2017.** If you plan to continue data collection or analysis beyond this date, please submit an application for continuation approval to SERU by **15th December 2016**.

You are required to submit any proposed changes to this study to SERU for review and the changes should not be initiated until written approval from SERU is received. Please note that any unanticipated problems resulting from the implementation of this study should be brought to the attention of SERU and you should advise SERU when the study is completed or discontinued.

You may embark on the study.

Yours faithfully,

EAB

PROF. ELIZABETH BUKUSI,
ACTING HEAD,
KEMRI/SCIENTIFIC AND ETHICS REVIEW UNIT

In Search of Better Health

Appendix 7: KEMRI Animal Care and Use Committee (ACUC)Approval

KENYA MEDICAL RESEARCH INSTITUTE

Centre for Virus Research, P.O. Box 54628 - 00200 NAIROBI - Kenya
Tel: (254) (020) 2722541, 254 02 2713349, 0722-205901, 0733-400003 Fax (254) (020) 2726115, Email: cvr@kemri.org

KEMRI/ACUC/ 02.08.16 **5th August 2016**

Dr Thumbi Mwangi,
CGHR, KEMRI

Dr. Thumbi,

RE: <u>Animal use approval for "Enhancing childhood nutrition and growth through targeted livestock interventions" protocol</u>

The KEMRI ACUC committee acknowledges the resubmission of the above mentioned protocol. It has been confirmed that all the issues raised earlier have been addressed appropriately. It has also been noted that all the poultry handling will be done in conjunction with the Department of Veterinary Services as stated in the letter MOALF/SDL/VEEU/DSE.INV/VOL.1/ (10).

Approval is granted for a period of two years starting from when the SERU approval will be obtained. If you still intend to handle poultry after the period covered by this initial approval, you are required to submit an application for continuing approval to the ACUC 1 month prior to the expiry of the initial SERU approval. In addition, the committee expects the study to provide an annual report on the progress of animal use simultaneously with the annual continuing review report to SERU.

The committee expects you to adhere to all the poultry handling procedures as described in the protocol.

The committee wishes you all the best in your work.

Yours sincerely,

Dr. Konongoi Limbaso
Chairperson KEMRI ACUC

KENYA MEDICAL RESEARCH INSTITUTE
★ 5 AUG 2016 ★
ANIMAL CARE AND USE COMMITTEE
Signature: ....

Appendix 8: University of Nairobi, Biosafety, Animal Use and Ethics (BAUEC) Approval

UNIVERSITY OF NAIROBI

FACULTY OF VETERINARY MEDICINE

DEPARTMENT OF VETERINARY ANATOMY AND PHYSIOLOGY

P.O. Box 30197,
00100 Nairobi,
Kenya.

Tel: 4449004/4442014/ 6
Ext. 2300
Direct Line. 4448648

Dr Elkanah Otiang
Dept.Vet Pathol. Microbiol & Parasitology

REF:FVM BAUEC/2018/154

09/07/2018

Dear Dr Otiang,

RE: Approval of Proposal by Biosafety, Animal use and Ethics committee

Impact of Newcastle disease vaccination on survival and productivity of indeginous chicken and subsequent child nitrition and growth in associated households in Western Kenya.

By Elkanah Otiang 52193/2015

We refer to the above PhD proposal that you submitted to our committee for review. We have now reviewed the proposal and have noted the following:

1. That your work involves vaccination of Chicken for Newcastle disease and collection of data on chicken growth and survival and also human data.
2. That your research proposal has been approved by the KEMRI Scientific Ethics and Review Unit and separately by the Animal care and Use committee in KEMRI.
3. That the actual collection of samples and handling of chicken will be carried out by qualified personel supervised by a Veterinarian.

We hereby approve your work as per the proposal you submitted.

Rodi O. Ojoo BVM M.Sc Ph.D
Chairman, Biosafety, Animal Use and Ethics Committee,
Faculty of Veterinary Medicine

Appendix 9: Publication in referred journals

Appendix 9.1: Mortality as the primary constraint to enhancing nutritional and financial gains from poultry: A multi-year longitudinal study of smallholder farmers in western Kenya

PLOS ONE

RESEARCH ARTICLE

Mortality as the primary constraint to enhancing nutritional and financial gains from poultry: A multi-year longitudinal study of smallholder farmers in western Kenya

Elkanah Otiang[1,2,3], Zoë A. Campbell[4,5], Samuel M. Thumbi[1,2,3,4], Lucy W. Njagi[1], Philip N. Nyaga[1], Guy H. Palmer[1,3,4]*

1 University of Nairobi, Nairobi, Kenya, **2** Kenya Medical Research Centre, Kisumu, Kenya, **3** Washington State University Global Health-Kenya, Nairobi, Kenya, **4** Paul G. Allen School for Global Animal Health, Washington State University, Pullman, Washington, United States of America, **5** International Livestock Research Institute, Nairobi, Kenya

* gpalmer@wsu.edu

Check for updates

OPEN ACCESS

Citation: Otiang E, Campbell ZA, Thumbi SM, Njagi LW, Nyaga PN, Palmer GH (2020) Mortality as the primary constraint to enhancing nutritional and financial gains from poultry: A multi-year longitudinal study of smallholder farmers in western Kenya. PLoS ONE 15(5): e0233691. https://doi.org/10.1371/journal.pone.0233691

Editor: Roman R. Ganta, Kansas State University, UNITED STATES

Received: March 6, 2020

Accepted: May 10, 2020

Published: May 29, 2020

Peer Review History: PLOS recognizes the benefits of transparency in the peer review process; therefore, we enable the publication of all of the content of peer review and author responses alongside final, published articles. The editorial history of this article is available here: https://doi.org/10.1371/journal.pone.0233691

Data Availability Statement: The full data set is provided in Open Science Framework at https://osf.io/9u4fn/.

Abstract

Background

Chickens are a widely held economic and nutritional asset in rural Africa and are frequently managed by women. Despite potential benefits of larger flock sizes, the average number of chickens kept at the household level is reported to be low. Whether this reflects decision-making to maximize benefits per unit labor by voluntary reduction of chicken numbers by consumption or sale versus involuntary losses due to mortality is a significant gap in knowledge relevant to improving smallholder household welfare.

Methods

In a 4-year longitudinal study of 1,908 smallholder households in rural western Kenya, the number of chickens owned by quarterly census at each household was determined. Households reported gains and losses of chicken over the immediate previous quarter. Gains were classified as on-farm or off-farm; losses were classified as voluntary (sales, gifts, consumption) or involuntary (mortality, unclassified loss).

Results

The mean number of chickens owned over the 16 quarters was 10, consistent with prior cross-sectional data. Involuntary losses represented 70% of total off-take, while voluntary off-take represented the remaining 30%. Mortality composed 60% of total reported off-take and accounted for most of the involuntary losses. Household consumption, sales, and gifts represented 18%, 9%, and 3% of off-take, respectively.

Funding: Funding was provided by the Paul G. Allen School for Global Animal Health Washington State University. There is no specific grant number. https://globalhealth.wsu.edu Neither the late Paul G. Allen or his Foundation had any role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing interests: The authors have declared that no competing interests exist.

Conclusion

The overwhelming majority of off-take can be classified as involuntary off-take, principally due to mortality, that does not reflect the owner's decision to maximize value through nutritional gain, income, or social capital. This strongly suggests that there is substantial opportunity to enhance the value of chickens as an asset, both nutritional and income generating, for smallholder households living at poverty level. Our findings suggest that programs emphasizing community level poultry vaccination and feed supplementation are much more likely to be effective than those solely focused on providing chickens.

Introduction

Improving small scale poultry production has been targeted as a pathway to improve nutritional and economic gains for rural African families affected by high rates of childhood stunting and poverty. Chickens kept by smallholder farmers serve as a source of food rich in protein and micronutrients (eggs and meat), household income through sales, investment, and savings [1–4]. In addition, chickens have an important cultural role in societal and familial life [2]. Chickens are frequently managed by women, providing opportunity to enhance gender equality in resource management [5–8]. This, in turn, may result in healthier households as women are more likely to prioritize spending on health care, nutrition, and education [8]. The commitment of organizations such as the Bill & Melinda Gates Foundation, Heifer International, the U.N. Food and Agriculture Organization, and others in encouraging chicken-keeping and improving smallholder poultry productivity in sub-Saharan Africa illustrates the potential for achieving broader health and development goals [3,9].

Despite these known and potential benefits, smallholder flock sizes remain small. In a cross-sectional survey of smallholder households in Tanzania, mean ownership was 11 chickens in a 2007 study [10] and 13 chickens as reported in two independent studies from 2010 and 2018 [3,11]. Similarly, in Kenya, smallholder flock sizes ranged between 10 and 20 chickens [12–15]. Whether this consistent small flock size reflects deliberate management choices or involuntary losses is a significant gap in knowledge relevant to ongoing programs encouraging smallholder poultry ownership. Although smallholder flocks typically scavenge during the day for a significant proportion of their feed and are often housed only at night, even this minimal management has an opportunity cost for the household, with the greatest cost to women. Production priorities are likely to differ between men and women when production increases do not allow women to benefit in proportion to their labor contributions [6]. Increasing the number of chickens beyond the current flock sizes may impact time available for other activities that fall predominantly on women such as gathering fuel and water, cooking, and child-care. A large proportion of voluntary off-take suggests a household is actively managing flock size and optimizing benefits per available unit of labor. Examples of voluntary off-take include sales, giving chickens as gifts, and household consumption of chickens. In contrast, when a large proportion of loss is involuntary, such as death from disease, starvation, or predation, it suggests the household is missing out on potential nutritional and economic benefits per unit labor.

We address this question of why flock sizes remain small using a longitudinal survey between 2013 and 2017 of approximately 1,900 households in ten villages in rural western Kenya. A 2013–2014 study reported that 80% of these households depend on mixed-crop and livestock agriculture and 88% kept chickens [15]. In the current longitudinal study, a census of chicken ownership was conducted quarterly for each household, and included recall of increases due to purchases, gifts, and on-farm hatching as well as decreases due to sale, consumption, gifts, death, and unknown losses such as predation. Here, we present the findings of the study, and discuss the results in the context of understanding the factors that drive small flock sizes in smallholder production systems with the goal of enhancing benefits of smallholder chicken ownership.

Materials and methods

Ethical approval

The research was approved by the Ethical and Animal Care and Use Committees (SSC Protocol no. 2250) of the Kenya Medical Research Institute to actively conduct animal disease surveillance and economic impact of domestic animal ownership, morbidity, and mortality.

Study population

A total of 1,908 rural households with a total population of approximately 6,400 individuals in Rarieda sub-county, Siaya County, Kenya were enrolled and followed longitudinally over 16 quarterly visits starting in February, 2013. The mean age of household head was 53 years with a third of households having at least a child <3 years of age and 42% with a child < 5 years old (Table 1). A cross-sectional study in 2015 found that 23.5% of children <5 years in these households were physically stunted, underscoring the importance of nutrition in this population [16]. Households practice small-scale mixed livestock and crop agriculture for livelihoods. At study initiation, 93% of households reported owning at least one species of livestock, and 88% reported owning chickens. The median number of ruminant livestock was nine per household, which included cattle (55% of households), goats (41%), and sheep (19%). Primary crops were maize (produced by 91% of households), beans (35%), potatoes (8%), sorghum (5%), and cassava (4%) [15]. All households were included in the study even if they did not own chickens at study initiation as chicken ownership may fluctuate by season or be affected by external factors such as periodic off-farm employment opportunities.

Table 1. Demographic characteristics of the study population.

Age of Primary Respondent	53.3±17.1 years
Gender	
Male	50.1%
Female	49.9%
Education Level	
No formal education	14.5%
Primary education	65.4%
Secondary education	16.6%
Tertiary education	3.6%
Primary Occupation	
Employed full-time on farm	56.6%
Employed part time on farm	9.0%
Self-employed off farm	16.3%
Salaried off farm	5.3%
Other	12.8%
Households with ≥ 1 child	
Under 3 years	33%
Under 5 years	42.3%
Under 10 years	56.3%

https://doi.org/10.1371/journal.pone.0233691.t001

Data collection

Data on the number of chickens owned on the interview day and recall of gains and losses over the previous three months were collected from each participating household quarterly. These data were collected by community enumerators in the local language, entered onto an electronic data capture tool, downloaded, and stored in a Microsoft Access database®. Increases in chickens over the last quarter were reported by an adult household member and categorized as hatched on premises, purchased, or acquired as a gift. Decreases in chickens were reported as sold, died, lost due to undetermined cause, or given as gifts. For sales, the price per chicken was reported. For income (earnings and sales) and expenses, Kenya shillings were converted to U.S. dollars using 2013 year's average as an exchange rate reference (1 USD = 85.52 Shillings) and discounted at an annual rate of 3.5% [17]. All datasets underwent validation and consistency checks to identify and resolve errors. The full data set is provided in Open Science Framework at https://osf.io/9u4fn/.

Data analysis

Analysis of flock size and increases and decreases over time were analyzed using STATA (Stata, 2013). Decreases in chicken numbers were categorized as voluntary (consumption, sale, or gifts) or involuntary (death or unclassified loss such as predation or theft).

Results and discussion

Chicken ownership over time

The overwhelming majority of households (1,805/1,908; 94.6%) reported owning at least one chicken during at least one of the 16 quarterly visits. This is in broad agreement with prior data from this community that reported 88% of households owning chickens [15]. The average flock size determined by quarterly on-farm census was approximately 10 chickens and was relatively constant over multiple years (Table 2). This longitudinal data supports previous cross-sectional data of chicken ownership in rural east Africa [3,10–15].

Sources of increases in chicken ownership

Households reported mean gains of 6.38±8.26 chickens per quarter (Table 3). Chicks hatched at the household represented the overwhelming majority of reported increases (97%), with purchases and received gifts representing only 2% and 1%, respectively (Fig 1). The relatively constant flock size suggests that gains through hatching are balanced by off-take.

Sources of decreases in chicken ownership

Households reported mean off-take of 5.35±7.68 chickens per quarter (Table 4). Involuntary losses represented 70% of total off-take, while voluntary off-take represented the remaining 30%. Mortality composed 60% of total reported off-take and accounted for most of the involuntary losses (Fig 2). Household consumption, sales, and gifts represented 18%, 9%, and 3% of off-take, respectively. Household visits that took place within rainy versus dry seasons were analyzed for an impact of seasonality: there was no significant effect on either gains and decreases in chicken numbers by season nor a significant effect on the source of gains or decreases. In aggregate, the overwhelming majority of off-take can be classified as involuntary, off-take that does not reflect the owner's decision to maximize value through nutritional gain, income, or social capital.

Table 2. Longitudinal census of household chicken ownership in Rarieda, Kenya.

Visit Quarter	Mean # of Chickens/ Household	95% confidence interval
1	11.65	11.14–12.15
2	9.78	9.36–10.21
3	10.45	9.97–10.92
4	9.98	9.50–10.46
5	8.31	7.90–8.72
6	11.10	10.59–11.61
7	9.92	9.46–10.39
8	9.70	9.28–10.12
9	10.65	10.19–11.11
10	11.26	10.80–11.71
11	11.45	10.99–11.91
12	9.34	8.94–9.73
13	8.10	7.74–8.47
14	7.44	7.12–7.77
15	9.51	9.11–9.91
16	9.94	9.54–10.35
Cumulative	**9.91**	**9.48–10.35**

https://doi.org/10.1371/journal.pone.0233691.t002

Evidence of bias in self-reported gains and losses

There is a slight but notable inconsistency in self-reported quarterly gains and losses as compared to the census data (Fig 3). The recalled gains are on average higher than recalled losses, suggesting the numbers of chickens should marginally increase over time. This is not

Table 3. Longitudinal household reporting of chicken gains in Rarieda, Kenya.

Visit Quarter	Total Gains Mean (95% C. I.)	On-farm Gains[1] Mean (95% C. I.)	Off-farm Gains[2] Mean (95% C. I.)
1	7.51 (7.14–7.89)	7. 34 (6.97–7.71)	0.17 (0.13–0.21)
2	5.71 (5.41–6.020	5.57 (5.27–5.87)	0.15 (0.12–0.18)
3	7.23 (6.84–7.63)	7.05 (6.65–7.45)	0.18 (0.14–0.23)
4	7.67 (7.24–8.10)	7.41 (7.00–7.82)	0.26 (0.16–0.36)
5	5.85 (5.44–6.25)	5.61 (5.24–5.98)	0.24 (0.13–0.34)
6	6.89 (6.46–7.33)	6.66 (6.24–7.08)	0.23 (0.11–0.36)
7	6.57 (6.15–7.00)	6.34 (5.93–6.76)	0.23 (0.12–0.34)
8	6.01 (5.65–6.37)	5.83 (5.47–6.19)	0.18 (0.14–0.22)
9	6.33 (5.97–6.70)	6.18 (5.83–6.53)	0.16 (0.05–0.26)
10	7.38 (6.98–7.80)	7.14 (6.73–7.54)	0.24 (0.16–0.32)
11	7.62 (7.22–8.02)	7.50 (7.10–7.90)	0.12 (0.09–0.14)
12	6.44 (6.05–6.84)	6.32 (5.93–6.71)	0.13 (0.10–0.15)
13	4.85 (4.53–5.17)	4.68 (4.38–4.98)	0.17 (0.07–0.28)
14	4.10 (3.84–4.36)	3.97 (3.71–4.22)	0.14 (0.10–0.17)
15	5.90 (5.57–6.23)	5.79 (5.46–6.12)	0.11 (0.09–0.14)
16	5.95 (6.01–6.75)	5.81 (5.50–6.13)	0.14 (0.10–0.17)
Cumulative	**6.38 (6.01–6.75)**	**6.20 (5.84–6.56)**	**0.18 (0.11–0.24)**

[1]Hatching on premises

[2]Purchases and received gifts to the household

https://doi.org/10.1371/journal.pone.0233691.t003

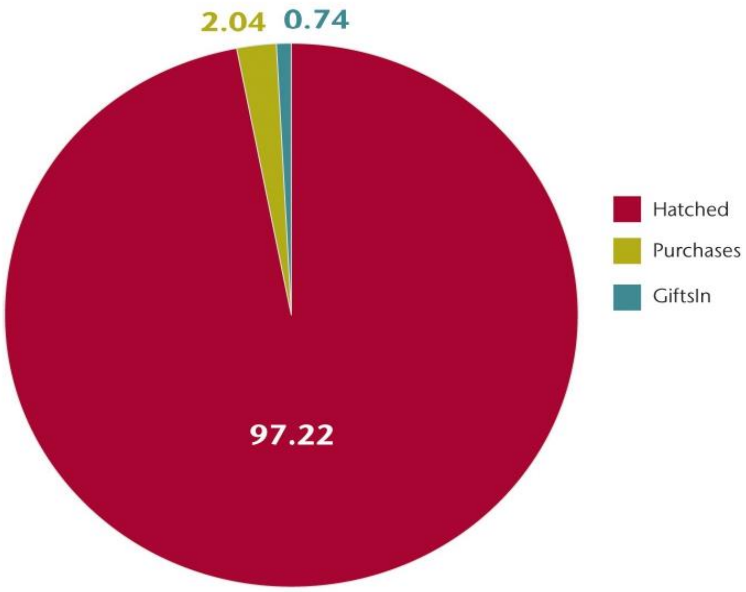

Fig 1. Source of reported gains in chicken ownership (percentages).

https://doi.org/10.1371/journal.pone.0233691.g001

consistent with the census, which shows flock size remaining stable over time. A notable difference is that the self-reported gains and losses require recall over the prior quarter while the census is based on an actual count. Two possible explanations for the discrepancy are recall bias and social desirability bias. Recall bias, or systematic errors in remembering past events or

Table 4. Longitudinal household reporting of chicken off-take in Rarieda, Kenya.

Visit Quarter	Total Off-take Mean (95% C.I.)	Voluntary Off-take Mean (95% C.I.)	Involuntary Losses Mean (95% C.I.)
1	6.95 (6.49–7.40)	1.97 (1.82–2.12)	4.97 (4.59–5.35)
2	6.00 (5.59–6.39)	1.64 (1.52–1.77)	4.35 (4.00–4.70)
3	6.62 (6.21–7.04)	1.54 (1.43–1.65)	5.08 (4.71–5.45)
4	7.60 (7.16–8.03)	2.18 (2.04–2.32)	5.41 (5.03–5.79)
5	5.04 (4.72–5.87)	1.41 (1.30–1.53)	3.63 (3.35–3.90)
6	4.37 (4.07–4.67)	1.35 (1.22–1.48)	3.02 (2.78–3.26)
7	5.50 (5.12–5.87)	1.65 (1.50–1.81)	3.84 (3.52–4.16)
8	4.94 (4.62–5.26)	1.69(1.54–1.84)	3.25 (2.99–3.51)
9	4.10 (3.84–4.36)	1.23 (1.13–1.33)	2.87 (2.65–3.08)
10	5.89 (5.52–6.26)	1.79 (1.61–1.96)	4.10 (3.80–4.40)
11	5.92 (5.53–6.32)	1.79 (1.65–1.93)	4.13 (3.81–4.45)
12	5.69 (5.31–6.06)	1.31 (1.26–1.41)	4.37 (4.04–4.70)
13	5.29 (4.90–5.63)	1.65 (1.48–1.82)	3.62 (3.33–3.91)
14	3.91 (3.65–4.17)	1.23 (1.11–1.35)	2.68 (2.47–2.89)
15	3.52 (3.30–3.74)	1.10 (1.01–1.19)	2.41 (2.23–2.60)
16	4.39 (4.14–4.64)	1.34 (1.24–1.45)	3.05 (2.84–3.26)
Cumulative	**5.35 (5.01–5.70)**	**1.56 (1.43–1.69)**	**3.08 (3.51–4.09)**

https://doi.org/10.1371/journal.pone.0233691.t004

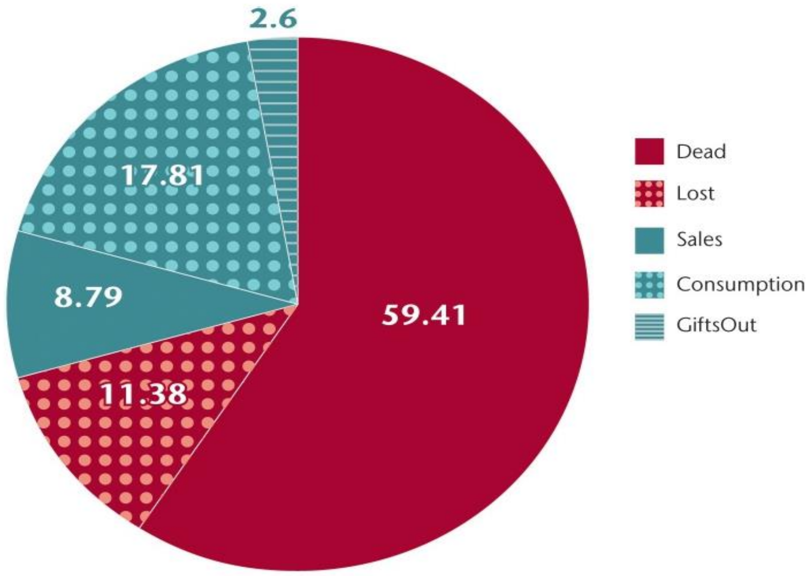

Fig 2. Source of reported off-take in chickens (percentages).

https://doi.org/10.1371/journal.pone.0233691.g002

experiences, has been investigated in epidemiologic and medical research [18], where it has found to be related to factors including length of recall period, frequency of experiencing reported events, and respondent characteristics such as age. While retrospective surveys are often used in agricultural and livestock surveys, recall bias is rarely quantified because of the expense [19]. This study design is well placed to identify recall bias because it includes a respondent recall of chicken gains and losses as well as a longitudinal census. In this study, respondents are systematically over-reporting gains relative to losses of chickens. This may reflect a bias towards matching reported gains to the number of chickens at the time of the census and/or a tendency to discount prior, especially temporally distant, losses. The age of the chicken may also be a factor in recall bias. In scavenge-based and free-range systems, an estimated 70 percent of chicks die before they reach the age of six weeks [3]. While we do not have data on the proportions of chick losses relative to older chickens, one explanation is that prior losses of chicks may be discounted relative to adult chickens and thus lead to recall bias on losses. Another potential explanation is social desirability bias, whereby the desire to present oneself in a positive light or to give a socially desirable response can lead to systematic underreporting of negative outcomes [20].

Involuntary loss and opportunity cost

The hypothesis that chicken numbers are maintained at a low, relatively constant level due to a decision to maximize value while controlling opportunity cost linked to chicken management can be rejected. Both sources of involuntary loss, observed mortality and loss/predation, reflect, at least in part, lack of investment in chicken husbandry. Observed mortality is due to infectious disease, poor nutrition available by scavenging, and frequently a combination of the two [4,14,21]. Loss/predation reflects the lack of secure housing, especially during the day

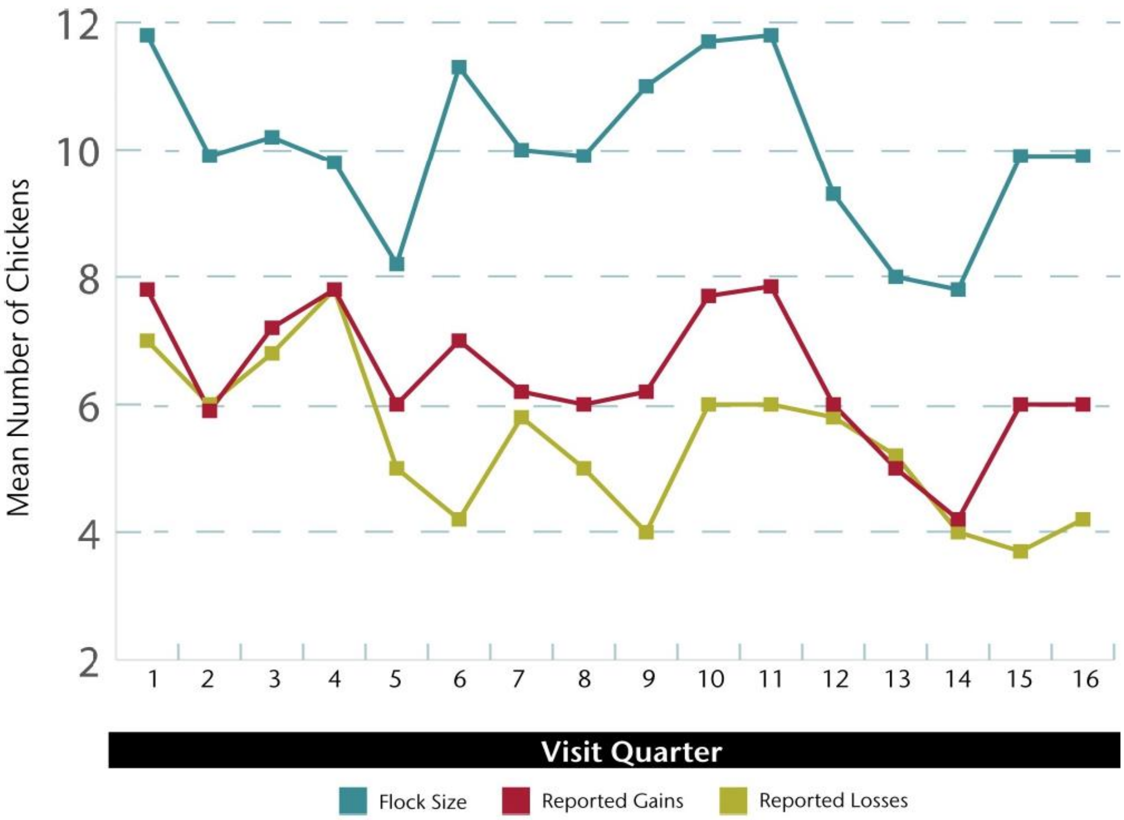

Fig 3. Mean household flock size, reported gains and losses.

https://doi.org/10.1371/journal.pone.0233691.g003

when most chickens are freely scavenging. In our study, 98% of households reported that chickens free range scavenged during the day; 93% maintained chickens within the human household at night with only 7% in a dedicated coop or enclosure. Similarly, only 12% of households reported receiving some form of treatment, vaccination, or de-worming at some point during the 16 quarterly visits. This is supported by a survey of a subset of 533 households in which 5.65% reported veterinary service with vaccination at 1.35%, ectoparasite control at 1.26%, and de-worming at 0.42%.This lack of investment may reflect lack of capital available for vaccine and drug purchases and materials for chicken housing, the opportunity cost given other household responsibilities, or both. Accessible capital is limited within the households: mean assets for these households were reported in 2015 as $100 USD (median of $50 USD/household) [15] and in the current study mean daily income was $1.17 USD (S1 Table). Similarly, time for chicken management may be markedly limited as 91% of households collect firewood daily for cooking and 60% of households devote more than an hour to daily water collection. Vaccination against Newcastle disease, an episodic and highly virulent disease responsible for the greatest mortality in small flocks [22,23], requires both capital and time [24,25]. Consequently, vaccinations and treatment for Newcastle disease and other poultry diseases may be difficult for smallholder households to routinely access [26].

Maximizing value of smallholder poultry production

Chickens are the most commonly held livestock species in this study population [15], broadly consistent with their role in rural smallholder communities [2,27]. As a commonly held asset, there is potential to enhance their value to the household. Over 800 children were tracked for a research project that overlapped temporally and spatially with the current study [16]. Recent egg consumption was associated with an average 30% increase in children's height gain, and egg consumption, unsurprisingly, was linked to the number of chickens owned by the household [16]. In addition to direct nutritional benefits, the mean selling price for a chicken in the current study was $1.97 USD ($1.57–2.39 USD) (S2 Table), representing a potential gain for increasing voluntary off-take even if the same total number of chickens was maintained. Given a mean daily income of $1.17 USD, this represents an opportunity based on a currently widely held resource. To the degree that our study population is representative of rural African households in poverty, our findings suggest that initiatives solely focused on providing chickens or on improved breeds of chickens are unlikely to be successful in the absence of improved husbandry. While selected breeds have increased productivity in more intensively managed production, "local African chickens" are of composed of ecotypes with high genetic diversity and heterogenicity, a result of natural selection within the resource limitations of smallholder farmers [28]. As mortality represents the greatest source of involuntary loss, programs to reduce the capital and opportunity costs of vaccination and supplemental feed for the local chicken ecotypes are most likely to be effective in resource optimization. Comparative data from a recent study of smallholder chicken owners in Tanzania indicates that a substantial majority (81%) are aware of Newcastle Disease vaccines, even though only 26% had used these in the past three months [24]. A willingness to pay analysis from the same study population indicated that households were prepared to pay twice the actual cost of vaccine, reflecting recognition of the benefits of vaccination [11]. However, the opportunity cost to acquire vaccines and the need to share vaccine among multiple households to reduce the cost per dose represent barriers to routine vaccination [11,25]. Vaccine sharing, requiring leadership and coordination within the community, is enhanced through education; if the household decision-maker for chickens had completed primary school or secondary school compared to having no formal education, the household was 2.7 and 4.4 times, respectively, more likely to share vaccine [25]. Given that in the present study communities, 14% had no formal education and 65% had only primary school education (Table 1), this suggests a barrier to routine vaccination that could be overcome programmatically. While we do not have similar data on feed supplementation, similar capital limitations and opportunity cost barriers likely exist for smallholder households.

Conclusion

This study strongly suggests that there is substantial opportunity to enhance the value of chickens as an asset, both nutritional and income generating, for smallholder households living at poverty level. Our findings suggest that programs emphasizing community level vaccination and feed supplementation are much more likely to be effective than those solely focused on providing chickens

Supporting information

S1 Table. Summary of quarterly household incomes (USD).
(DOCX)

S2 Table. Summary of quarterly income and on-farm expenses (USD).
(DOCX)

Acknowledgments

We thank the contributions of the following individuals, who made the research possible. Field team supervisor, James Oigo; Animal Health Technicians, Samwel Asembo, Geoffrey Odima, Bob Miseda, Beryl Oyoo and Austin Ochung'; Community Health Interviewers: Joseph Onyango, Daniel Odongo, Rosemary Warinda, Bridon Kojo, Thomas Wachiaya, Fredrick Ong'wen, Meresia Owuor; Martin Aundi, Fredrick Adhiambo, Benedine Warinda, Jane Okal, Fred Ojode; Data Programmer and Manager, Linus Otieno; Data Clerk, Judith Oduol.

Author Contributions

Conceptualization: Guy H. Palmer.

Data curation: Zoë A. Campbell, Samuel M. Thumbi.

Formal analysis: Elkanah Otiang, Samuel M. Thumbi, Guy H. Palmer.

Funding acquisition: Guy H. Palmer.

Investigation: Elkanah Otiang, Samuel M. Thumbi.

Methodology: Elkanah Otiang, Zoë A. Campbell, Samuel M. Thumbi, Guy H. Palmer.

Project administration: Guy H. Palmer.

Resources: Guy H. Palmer.

Supervision: Samuel M. Thumbi, Lucy W. Njagi, Philip N. Nyaga, Guy H. Palmer.

Validation: Elkanah Otiang, Guy H. Palmer.

Writing – original draft: Elkanah Otiang, Guy H. Palmer.

Writing – review & editing: Elkanah Otiang, Zoë A. Campbell, Samuel M. Thumbi, Lucy W. Njagi, Philip N. Nyaga, Guy H. Palmer.

References

1. Randolph TF, Schelling E, Grace D, Nicholson CF, Leroy JL, Cole DC, et al. Role of livestock in human nutrition and health for poverty reduction in developing countries. J Animal Sci. 2007; 85:2788–2800. https://doi.org/10.2527/jas.2007-0467.
2. Alders RG, Pym RAE. Village poultry: Still important to millions, eight thousand years after domestication. World's Poultry Science J. 2009; 65:181–190.
3. Kryger KN, Thomsen KA, Whyte MAW, Dissing M. Smallholder Poultry Production–Livelihoods, Food Security and Sociocultural Significance. Smallholder Poultry Production Paper No. 4. Rome: Food and Agriculture Organization; 2010.
4. Magothe TM, Okeno TO, Muhuyi WB, Kahi AK. Indigenous chicken production in Kenya: I. Current status. World's Poultry Science J. 2012; 68:119–132. https://doi.org/10.1017/S0043933912000128.
5. Kingori A, Wachira A, Tuitoek J. Indigenous chicken production in Kenya: A review. *Internat J Poultry Sci.* 2010; 9:309–316.
6. Kristjanson P, Neufeldt H, Gassner A, Mango J, Kyazze FB, Desta S et al. Are food insecure smallholder households making changes in their farming practices? Evidence from East Africa. Food Security. 2012; 4:381–397. https://doi.org/10.1007/s12571-012-0194-z.
7. Wilson KR. Do women reap the benefits? Exploring access and social exclusion among village chicken producers in Kenya. PhD Dissertation, Ohio State University. 2019. Available from https://doi.org/10.1017/CBO9781107415324.004
8. Valdivia C. Gender, livestock assets, resource management, and food security: lessons from the SR-CRSP. Agr Human Values, 2001; 18: 27–39. https://doi.org/10.1023/A.
9. Farrell D. The role of poultry in human nutrition, In: Poultry Development Review. Rome: Food and Agriculture Organization; 2013. pp. 2–9.

10. Msami H. Poultry Sector Country Review: Tanzania. In FAO Poultry Sector Country Review. Rome: Food and Agriculture Organization; 2007. https://doi.org/10.1093/acprof:oso/9780190498511.003.0001

11. Campbell ZA, Otieno L, Shirima GM, Marsh TL, Palmer GH. Drivers of vaccination preferences to protect a low-value livestock resource: Willingness to pay for Newcastle disease vaccines by smallholder households. Vaccine. 2018a; 11:11–18. https://doi.org/10.1016/j.vaccine.2018.11.058.

12. Nyaga P. (2008). Poultry sector country review: Kenya. In FAO Poultry Sector Country Review. Rome: Food and Agriculture Organization; 2007. http://agris.fao.org/agris-search/search.do?recordID=XF2006446635.

13. Okitoi LO, Udo HMJ, Mukisira EA, De Jong R, Kwakkel RP. (2006). Evaluation of low input interventions for productivity of indigenous chickens in western Kenya. Agricultura Trop Et Subtrop. 2006; 39:179–182.

14. Olwande PO, Ogara WO, Okuthe SO, Muchemi G, Okoth E, Odindo MO, et al. Assessing the productivity of indigenous chickens in an extensive management system in Southern Nyanza, Kenya. Trop Animal Health Prod. 2010; 42:283–288. https://doi.org/10.1007/s11250-009-9418-4.

15. Thumbi SM, Njenga MK, Marsh TL, Noh S, Otiang E, Munyua P, et al. Linking human health and livestock health: A "one-health" platform for integrated analysis of human health, livestock health, and economic welfare in livestock dependent communities. PLoS ONE 2015; https://doi.org/10.1371/journal.pone.0120761.

16. Mosites E, Thumbi SM, Otiang E, McElwain TF, Njenga M, Rabinowitz PM et al. Relations between household livestock ownership, livestock disease, and young child growth. J Nutr. 2016; 146:1118–1124. https://doi.org/10.3945/jn.115.225961.

17. Earnshaw J, Lewis G. (2008). NICE Guide to the methods of technology appraisal: pharmaceutical industry perspective. Pharmacoeconomics. 2008; 26: 725–727.

18. Althubaiti A. (2016). Information bias in health research: Definition, pitfalls, and adjustment methods. J Multidisciplinary Healthcare. 2016; 9:211–217. https://doi.org/10.2147/JMDH.S104807.

19. Lesnoff M. Reliability of a twelve-month retrospective survey method for estimating parturition and mortality rates in a traditional African livestock farming system. Revue Élev. Méd. Vét. Pays Trop. 2009; 62:49–57.

20. Fisher RJ. Social desirability bias and the validity of indirect questioning. J Consumer Res. 1993; 20:303–315. https://doi.org/10.2307/2489277.

21. Kitalyi AJ. Village chicken production systems in rural Africa: Household food security and gender issues. Rome: Food and Agriculture Organization; 1998.

22. Alders R, dos Anjos F, Bagnol B, Fumo A, Mata B, Young M. Controlling Newcastle Disease in Village Chickens: A Training Manual. ACIAR Monograph No. 87; 2002.

23. Fisher, H. Newcastle disease control in Africa. ACIAR Impact Assessment Series Report No. 87. Canberra: Australian Centre for International Agricultural Research; 2014. pp. 1–62.

24. Campbell ZA, Marsh TL, Mpolya EA, Thumbi SM, Palmer GH. Newcastle disease vaccine adoption by smallholder households in Tanzania: Identifying determinants and barriers. PLoS ONE. 2018b: 13:1–16. https://doi.org/10.1371/journal.pone.0206058.

25. Campbell ZA, Thumbi SM, Marsh TL, Quinlan MB, Shirima GM, Palmer GH. Why isn't everyone using the thermotolerant vaccine? Preferences for Newcastle disease vaccines by chicken-owning households in Tanzania. PLoS ONE. 2019; 14:1–15. https://doi.org/10.1371/journal.pone.0220963.

26. Lindahl JF, Young J, Wyatt A, Young M, Alders R, Bagnol B, et al. Do vaccination interventions have effects? A study on how poultry vaccination interventions change smallholder farmer knowledge, attitudes, and practice in villages in Kenya and Tanzania. Trop Animal Health Prod. 2019; 51:213–220. https://doi.org/10.1007/s11250-018-1679-3.

27. Thornton PK, Kristjanson PM, Kruska RL, Reid RS. Mapping livestock and poverty: a tool for targeting research and development. BSAP Occasional Pub. 2004; 33:37–50. https://doi.org/10.1017/s1463981500041649.

28. Mpenda FN, Schilling MA, Campbell ZA, Mngumi EB, Buza J. The genetic diversity of local African chickens: A potential for selection of chickens resistant to viral infections. J App Poultry Res. 2019; 28:1–12. https://doi.org/10.3382/japr/pfy063.

Appendix 9.2: Impact of routine ND vaccination on chicken flock size in smallholder farms in western Kenya

PLOS ONE

RESEARCH ARTICLE

Impact of routine Newcastle disease vaccination on chicken flock size in smallholder farms in western Kenya

Elkanah Otiang[1,2,3], Samuel M. Thumbi[1,3,4], Zoë A. Campbell[5], Lucy W. Njagi[1], Philip N. Nyaga[1], Guy H. Palmer[3,4,6]*

1 College of Agriculture and Veterinary Sciences, University of Nairobi, Nairobi, Kenya, **2** Center for Global Health Research, Kenya Medical Research Institute, Kisumu, Kenya, **3** Washington State University Global Health-Kenya, Nairobi, Kenya, **4** Paul G. Allen School for Global Health, Washington State University, Pullman, Washington, United States of America, **5** International Livestock Research Institute, Nairobi, Kenya, **6** College of Health Sciences, University of Nairobi, Nairobi, Kenya

* gpalmer@wsu.edu

Check for updates

Citation: Otiang E, Thumbi SM, Campbell ZA, Njagi LW, Nyaga PN, Palmer GH (2021) Impact of routine Newcastle disease vaccination on chicken flock size in smallholder farms in western Kenya. PLoS ONE 16(3): e0248596. https://doi.org/10.1371/journal.pone.0248596

Editor: Eric Fèvre, University of Liverpool, KENYA

Received: January 18, 2021

Accepted: March 2, 2021

Published: March 18, 2021

Peer Review History: PLOS recognizes the benefits of transparency in the peer review process; therefore, we enable the publication of all of the content of peer review and author responses alongside final, published articles. The editorial history of this article is available here: https://doi.org/10.1371/journal.pone.0248596

Data Availability Statement: Data can be found at https://osf.io/yn7fk/ (DOI: 10.17605/OSF.IO/YN7FK).

Funding: The study was supported by a gift of Paul G. Allen to Washington State University. The

Abstract

Background

Poultry represent a widely held economic, nutritional, and sociocultural asset in rural communities worldwide. In a recent longitudinal study in western Kenya, the reported mean number of chickens per household was 10, with increases in flock size constrained principally by mortality. Newcastle disease virus is a major cause of chicken mortality globally and hypothesized to be responsible for a large part of mortality in smallholder flocks. Our goal was to determine the impact of routine Newcastle disease virus (NDV) vaccination on flock size and use this data to guide programs to improve small flock productivity.

Methods

We conducted a factorial randomized controlled trial in 537 households: in 254 households all chickens were vaccinated every 3 months with I-2 NDV vaccine while chickens in 283 households served as unvaccinated controls. In both arms of the trial, all chickens were treated with endo- and ecto parasiticides every 3 months. Data on household chicken numbers and reported gains and losses were collected monthly for 18 months.

Results

Consistent with prior studies, the overall flock size was small but with increases in both arms of the study over time. The mean number of chickens owned at monthly census was 13.06 ±0.29 in the vaccinated households versus 12.06±0.20 in the control households ($p = 0.0026$) with significant gains in number of chicks ($p = 0.06$), growers ($p = 0.09$), and adults ($p = 0.03$) in the vaccinated flocks versus the controls. Household reported gains were 4.50 ±0.12 total chickens per month when vaccinated versus 4.15±0.11 in the non-vaccinated controls ($p = 0.03$). Gains were balanced by voluntary decreases, reflecting household decision-making for sales or household consumption, which were marginally higher, but not

funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing interests: The authors have declared that no competing interests exist.

statistically significant, in vaccinated households and by involuntary losses, including mortality and loss due to predation, which were marginally higher in control households.

Conclusion

Quarterly NDV vaccination and parasiticidal treatment resulted in an increase in flock size by a mean of one bird per household as compared to households where the flock received only parasiticidal treatment. While results suggest that the preventable fraction of mortality attributable to Newcastle disease is comparatively small relatively to all-cause mortality in smallholder households, there was a significant benefit to vaccination in terms of flock size. Comparison with previous flock sizes in the study households indicate a more significant benefit from the combined vaccination and parasiticidal treatment, supporting a comprehensive approach to improving flock health and improving household benefits of production in the smallholder setting.

Introduction

Poverty and undernutrition, including both wasting and stunting, are major global challenges as illuminated in their importance to achieving the United Nations' Sustainable Development Goals [1]. Rural households in Africa, Asia, and Latin America disproportionately suffer from poverty and malnutrition [2, 3]. The Food and Agriculture Organization estimates that there are 500 million smallholder farms worldwide, which provide all or part of the household welfare for many of the 9% of the global population that living on less than US$2 per day [4–6]. Smallholder farms typically generate food and income from mixed crop agriculture, variably combined with small scale livestock production [7]. Chickens are the most commonly held livestock resource of smallholder households and represent an opportunity to provide eggs and meat to the household and potentially generate income from local sale [8, 9]. Furthermore, household chicken flocks represent an important economic and nutritional asset most commonly managed by women in rural households and can reflect their priorities for familial well-being [7, 8, 10].

Despite the global representation of chickens as the primary smallholder animal asset, small flock sizes and overall low productivity limit maximizing potential benefits [8, 11, 12]. Importantly, the low flock sizes do not appear to represent an economic based decision to optimize labour input while maximizing gain from household consumption or sale but rather a high level of involuntary losses due to mortality and predation [13]. A recent 4-year longitudinal study of 1,908 households in western Kenya found that the mean flock size was approximately 10 and highly stable over time, reflecting a balance of new chicks hatched on premises and losses, 60% of which were due to mortality [13]. Inputs into smallholder flocks were minimal: 98% of households reported that chickens scavenge for all or most of their feed during the day and 93% house chickens within the family dwelling at night [13]. Vaccination, supplemental nutrition, and treatment of endo- and ecto-parasites that would be expected to reduce morbidity and mortality [14] were uncommon [13].

Newcastle disease virus (NDV) is a highly transmissible and globally distributed infection of poultry [15, 16]. While high levels of biosecurity combined with vaccination are commonly used to prevent NDV outbreaks in commercial poultry, the reliance on free range scavenging for chickens in smallholder households results in ease of transmission between and within flocks [17]. Consequently, NDV is widely considered to be the leading constraint to efficient smallholder poultry productivity in Africa [8]. While vaccination is highly effective under controlled conditions, its efficacy under smallholder conditions may be much more variable depending on the underlying health and age composition of the flock [9, 12, 16, 17].

To determine whether regularly scheduled NDV vaccination resulted in increases in flock sizes over time, we conducted a factorial randomized controlled trial in 537 households where all chickens in 254 households were vaccinated every 3 months with I-2 NDV vaccine while chickens in 283 households served as unvaccinated controls. Here we report the change in monthly flock census over an 18 months period and discuss the results in the context of improving flock productivity and household well-being.

Materials and methods

Ethical approval

The study was approved by the Ethical and Animal Care and Use Committees (SSC Protocol no. 3159) of the Kenya Medical Research Institute.

Study population

The study took take place in Rarieda Sub-county of Siaya County in western Kenya within a health and demographic surveillance system (HDSS) site run by the Kenya Medical Research Institute and the United States Centres for Disease Control and Prevention (CDC) [18]. Livestock ownership is common in the area's households at 93%; 95% of households with an average flock size of ten chickens with chicken mortality accounting for over half of all reported animal death cases in participating households [13, 19].

Study design

The field study was an 18-month factorial randomized controlled trial with 537 households enrolled and followed upon meeting an inclusion criteria of chicken ownership and grouped into two arms (vaccinated n = 254 and control n = 283). The sample size calculation used the assumption that Newcastle disease vaccination would decrease flock mortality by $\geq$10%, assuming a probability of type 1 error set at 0.05 and 80% detection power and further assuming 10% participant loss (household loss from study). This was based on average flock size per household of 10 chickens (range 4–60), accounting for unequal cluster sizes. The vaccinated group routinely received immunization of two drops of Newcastle disease virus (NDV) AVIVAX I-2 thermostable vaccine ($10^{9.7}$ egg infectious doses/ml) intranasally or intraocularly depending on chicken's age at recruitment and every three months thereafter. All vaccines were diluted by an animal health technician on the morning of the vaccination, maintained in a cool box at 4˚C, and delivered prior to noon. Chickens in the control arm were not vaccinated. The same animal health technician dusted ecto-parasiticides of the group carbaryl (Sevin® powder) on the chickens and administered oral deworming using piperazine citrate (Ascarex-D®) in drinking water at recruitment and then every three months for chickens in both arms of the study.

Data collection and analysis

Flock size and age composition of the flock was enumerated at each monthly visit by an animal health assistant blinded to the treatment groups. In addition, the individual responsible for flock or the household head was interviewed in the local language using a semi-structured questionnaire to collect recall data on increases and decreases to the flock during the prior three months. The data were collected using a mobile phone based application CommCare®

and data maintained through a Microsoft Access database®. The data were cleaned and analysed using STATA (Stata, 2013). The full data set and data dictionary are provided in Open Science Framework https://osf.io/yn7fk/

Results

Study population

A total of 537 rural households in Rarieda Sub-county of Siaya County in western Kenya were enrolled and followed longitudinally over 18 monthly visits starting in December, 2016. By random household distribution, 254 were grouped into the vaccination arm while 283 served as controls. The primary respondents to the questionnaire were most often the individuals who managed the chickens (89%) or the head of the compound (11%). Of the individuals responsible for management of the flock, 93% were women.

Longitudinal monthly flock census

The mean flock sizes on visit 1, at the time of the first vaccination but prior to any possible effects of vaccination, were 11.63±0.70 for the 254 households in the vaccination arm of the study and 11.13±0.67 for the 283 control households. Enrolled households maintained flocks throughout the study period with less than 2% of visits recording no chickens at the monthly census. Over 18 monthly visits, the flock sizes increased in both arms but the total flock sizes were significantly greater in the vaccinated households: there was a cumulative mean of 13.06 ±0.29 chickens in the vaccinated households versus 12.06±0.20 in the control households ($p = 0.0026$) (Fig 1). The increases occurred across all age categories (Fig 1): the mean number of chicks in vaccinated households was 6.59±0.20 as compared to 6.20±0.13 in controls ($p = 0.06$), mean number of growers was 3.84±0.08 versus to 3.63±0.09 ($p = 0.09$), and mean number of adults was 3.32±0.19 as compared to 2.93±0.05 versus ($p = 0.03$). The increase was sustained throughout the study, whether analysed by the best-fit over the 18 visits (Fig 2) or by a best-fit tethered to the flock size at the visit 1 and then a best-fit determined (Fig 3).

Household reported gains and losses

During each monthly visit, the household respondent was asked to self-report gains and losses during the prior month. Households that received vaccination reported gains of 4.50±0.12

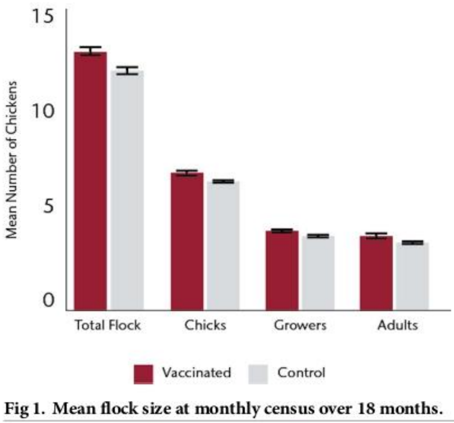

Fig 1. Mean flock size at monthly census over 18 months.

https://doi.org/10.1371/journal.pone.0248596.g001

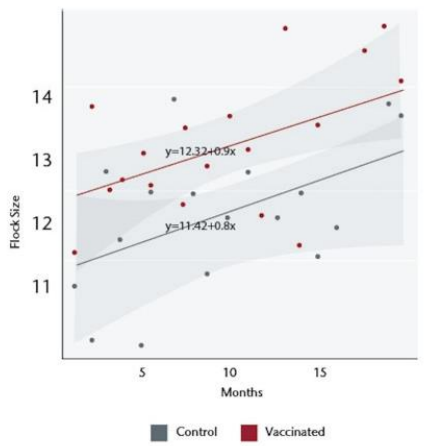

Fig 2. Flock size dynamic over time (best-fit of all data points).

https://doi.org/10.1371/journal.pone.0248596.g002

chickens per month as compared to 4.15±0.11 in the non-vaccinated control households ($p = 0.03$). Vaccination households reported total decreases of 2.50±0.09 chickens per month versus 2.43±0.09 in the control households ($p = 0.56$). Reported voluntary decreases in flock size, reflecting household decision-making for sales or household consumption, were marginally greater in vaccinated households, 1.10±0.05, as compared to 1.03±0.04 in control households ($p = 0.19$), representing 44% and 42% of the monthly decreases in vaccinated and control households, respectively (Fig 4). Involuntary losses, including mortality and both unspecified loss and loss to predation, were reported to be marginally higher in control households, 1.4±0.08, as compared to 1.3±0.08 in vaccinated households ($p = 0.39$), with mortality representing the greatest reported source of loss in both groups (Fig 4).

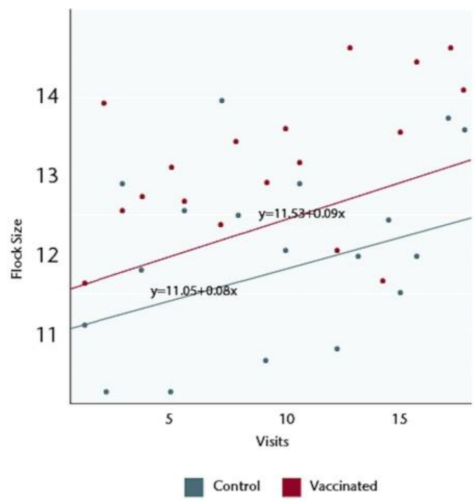

Fig 3. Flock size dynamic over time (best-fit of data points tethered to initial flock size).

https://doi.org/10.1371/journal.pone.0248596.g003

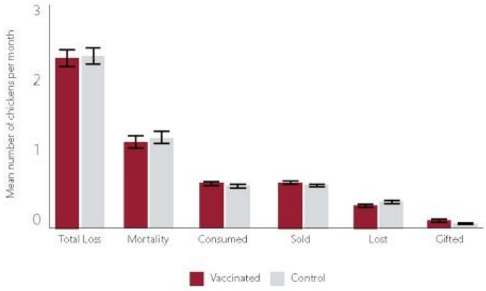

Fig 4. Source of reported monthly off-take in chickens.

https://doi.org/10.1371/journal.pone.0248596.g004

Discussion

Newcastle disease is endemic at the village level in Kenya and specifically in this region of western Kenya [20], where greater than 50% of chicken mortality is attributed to NDV. The use of a relatively large factorial randomized controlled trial allowed determination of the impact of NDV vaccination on flock size. Quarterly NDV vaccination of all chickens resulted in a mean gain of one chicken per household. While this gain seems modest, it represents an 8–10% increase in flock size from vaccination against a single viral pathogen. Importantly, the increase was sustained throughout the 18-month study, which included both wet and dry seasons to account for seasonal variation in transmission [20]. This suggests the opportunity for increased gains over time and an accumulating impact of routine NDV vaccination. If these gains were utilized for either household nutrition or income from sale there would be measurable benefits to the family [21, 22]. Consumption of either eggs or chicken meat have been shown to reduce childhood stunting [22], which remains at a high level in the study region and throughout much of rural sub-Saharan Africa. Two prior studies in this population support the impact of larger flock size on nutritional gains. The first, a study of 1500 households, found that an increase in number of chickens per household associated with a 28% likelihood of childhood consumption of eggs in the prior 3 days, holding other household factors constant [19]. The second study of 1800 households showed that poultry ownership was linked to a significant increase in both egg and chicken consumption (adjusted incidence rate ratio of 1.3). Furthermore, consumption was associated with a significant increase in monthly child height gain for children over the age of 6 months [22]. Similarly, routine vaccination has potential to increase household income. In a recent study of smallholder households in rural Tanzania, the market price for vaccine to inoculate 10 chickens was US$1.20 while the local market price for an adult chicken was US$3.12 [23]. Notably, respondents in that study were willing to pay twice the market price for vaccine, reflecting that households valued vaccination and perceived a favorable return on investment [23]. As 93% of the individuals in this study that managed the flocks were women and women have been shown to devote a much greater proportion of income into family nutrition and health care, this relatively modest increase in income can have a significant impact on familial well-being [7, 10, 11].

The current study allowed an estimation of the preventable fraction of mortality due to NDV. Vaccination was carried out by qualified animal health technicians supervised by a licensed veterinarian and records were kept on the storage and delivery of the I-2 vaccines. While NDV I-2 vaccination has been shown in numerous experimental and field studies to be highly effective [24–26], the preventable fraction of mortality due to a single vaccine reflects the overall causes of mortality and varies depending on the specifics of poultry management at the household level [17, 23]. The most reliable measure of vaccine impact from this study would be the 8–10% increase in total flock size at monthly census. In contrast to commercial poultry vaccination programs, in this study chickens were not uniformly vaccinated at a given age but on a quarterly schedule when all chickens on the premises were vaccinated regardless of prior vaccination history. This variation in vaccination history and immune status of individual birds affects the level of population immunity and would be likely to significantly diminish the flock level efficacy of vaccination. However, this variation in age structure for vaccination is reflective of smallholder household flock management.

The self-reported mortality by the household respondents indicated no significant difference in mortality between the vaccinated and control flocks (Fig 4). This discrepancy between independent census data and self-reported data has been previously observed among households in the study region [13]. In the prior study, individuals consistently overestimated gains and underestimated losses relative to actual census data [13]. This pattern is observed in the current study: as an example, the self-reported total monthly gains in vaccinated households were reported as 4.5 with overall monthly decreases of 2.5, inconsistent with census data that indicates a smaller monthly increase. Recall bias, representing systemic errors in remembering past events, and social desirability bias are two possible explanations for the discrepancy [27]. Households in the study region do not maintain written records on flock size, gains, and losses, thus losses that occurred earlier in the month may be discounted relative to gains that are still represented in the flock. This may be especially true for young chicks, which have a high mortality rate from NDV as well as other infectious and non-infectious causes [13, 15, 28, 29]. Social desirability bias, the tendency for survey respondents to answer questions in a way that will be viewed favourably by others, may also have an impact as the household may want to be seen by the interviewers and animal health team as being a responsible member of the community and thus overstate gains and understate involuntary losses, including mortality [30].

Notably, there were sustained gains in flock size in households that received vaccination and parasiticidal treatment and the control households that received only parasite control. While a control group with no treatment was not included (as participation required time commitment by the respondents), comparison with both the flock sizes at enrolment and the historical mean flock size of 10 in this study site [13, 19] suggest that parasiticidal treatment had a significant effect alone, which was further enhanced by NDV vaccination. Furthermore, quarterly visitation by an animal health technician provided the opportunity to seek *ad hoc* advice on flock management. This opportunity and the impact of external interest in a household's flock size may also have improved management independent of or interacting with vaccination and parasiticidal treatment. This is consistent with prior studies showing the impact of combined interventions and emphasizes that a comprehensive approach to improved poultry management at the smallholder level is needed [8]. Integrating supplemental nutrition would highly likely increase the efficacy of vaccination as well as maximize benefits from parasite control. The lack of routine vaccination for smallholder flocks does not appear to reflect a lack of knowledge regarding the importance of vaccination as indicated by willingness to pay studies in which respondents were willing to pay more than the actual cost of NDV vaccine [23]. Rather, the primary barrier to improved management appears to be at the level of services delivery. At a household level the incentives for effective delivery of more comprehensive poultry health and husbandry services are too small for commercial investment. However, at a community level this may provide a larger integrated market that would attract commercial engagement, especially if incentivized by government support for rural communities.

Finally, whether increased flock sizes are desirable from a labor management perspective is important, especially given that women, who most commonly have primary responsibility for flock husbandry management in this region, have multiple other demands on their effort. In a prior study [13], we assessed whether poultry owning households in this region maintained relatively small flock sizes as a deliberate decision to maximize benefits per unit labor by voluntary reduction of chicken numbers by consumption or sale versus involuntary losses due to mortality, predation or theft. The overwhelming majority of off-take was involuntary, principally due to mortality, that does not reflect the owner's decision to maximize value through nutritional gain, income, or social capital. This strongly suggests that there is substantial opportunity to enhance the value of chickens as an asset, both nutritional and income generating, for smallholder households.

Conclusion

This study demonstrates a significant impact of NDV vaccination on overall flock size that is maintained over time and is enhanced by parasite control. This is consistent with the need for integrated control of infectious diseases of poultry with substantial opportunity to improve nutritional and economic security for rural smallholder households.

Acknowledgments

We thank the contributions of the following individuals, who made the research possible. Field team supervisor, James Oigo; Animal Health Technicians, Samwel Asembo, Geoffrey Odima, Bob Miseda, Beryl Oyoo and Austin Ochung'; Community Health Interviewers: Joseph Onyango, Daniel Odongo, Rosemary Warinda, Bridon Kojo, Thomas Wachiaya, Fredrick Ong'wen, Meresia Owuor; Martin Aundi, Fredrick Adhiambo, Benedine Warinda, Jane Okal, Fred Ojode; Data Programmer and Manager, Linus Otieno; Data Clerk, Judith Oduol.

Author Contributions

Conceptualization: Zoë A. Campbell, Guy H. Palmer.

Data curation: Elkanah Otiang.

Formal analysis: Elkanah Otiang, Samuel M. Thumbi, Zoë A. Campbell, Guy H. Palmer.

Investigation: Elkanah Otiang, Samuel M. Thumbi.

Methodology: Guy H. Palmer.

Project administration: Guy H. Palmer.

Resources: Guy H. Palmer.

Supervision: Samuel M. Thumbi, Lucy W. Njagi, Philip N. Nyaga.

Visualization: Guy H. Palmer.

Writing – original draft: Elkanah Otiang, Guy H. Palmer.

Writing – review & editing: Elkanah Otiang, Samuel M. Thumbi, Zoë A. Campbell, Lucy W. Njagi, Philip N. Nyaga, Guy H. Palmer.

References

1. United Nations Sustainable Development Goals. https://sdgs.un.org/goals
2. Van de Poel E, Hosseinpoor AR, Speybroeck N, van Outi T, Vega J. Socioeconomic inequality in malnutrition in developing countries. Bull WHO. 2008; 86:282–291, 2008. https://doi.org/10.2471/blt.07.044800 PMID: 18438517
3. SDG Atlas 2017. https://datatopics.worldbank.org/sdgatlas/archive/2017/SDG-02-zero-hunger.html

4. Lowder SK, Skoet J, Raney T. The number, size, and distribution of farms, smallholder farms, and family farms worldwide. World Dev. 2016; 87:16–29. https://doi.org/10.1016/j.worlddev.2015.10.041.
5. World Bank. 2020. "Poverty and Shared Prosperity 2020: Reversals of Fortune." https://openknowledge.worldbank.org/bitstream/handle/10986/34496/9781464816024.pdf6.
6. UNDP. Charting pathways out of multidimensional poverty: Achieving the SDGs. 2020; http://hdr.undp.org/sites/default/files/2020_mpi_report_en.pdf.
7. Wong JT, de Bruyn J, Bagnol B, Grieve H, Li M, Pym R, et al. Small-scale poultry and food security in resource-poor settings: A review. Glob Food Sec. 2017; 15:43–52.
8. Kryger KN, Thomsen KA, Whyte MAW, Dissing M. Smallholder Poultry Production–Livelihoods, Food Security and Sociocultural Significance. Smallholder Poultry Production Paper No. 4. Rome: Food and Agriculture Organization; 2010.
9. Kingori A, Wachira A, Tuitoek J. Indigenous chicken production in Kenya: A review. Internat J Poultry Sci. 2010; 9:309–316.
10. Valdivia C. Gender, livestock assets, resource management, and food security: lessons from the SRCRSP. Agr Human Values. 2001; 18: 27–39. https://doi.org/10.1023/A.
11. Kristjanson P, Neufeldt H, Gassner A, Mango J, Kyazze FB, Desta S, et al. Are food insecure smallholder households making changes in their farming practices? Evidence from East Africa. Food Secur. 2012; 4(3):381–97.
12. Campbell ZA, Marsh TL, Mpolya EA, Thumbi SM, Palmer GH. Newcastle disease vaccine adoption by smallholder households in Tanzania: Identifying determinants and barriers. PLoS One. 2018; 13(10):1–16. https://doi.org/10.1371/journal.pone.0206058 PMID: 30356260
13. Otiang E, Campbell ZA, Thumbi SM, Njagi LW, Nyaga PN, Palmer GH. Mortality as the primary constraint to enhancing nutritional and financial gains from poultry: A multi-year longitudinal study of smallholder farmers in western Kenya. PLoS One. 2020; 15(5):1–11. https://doi.org/10.1371/journal.pone.0233691 PMID: 32470070
14. Dwinger RH, Unger H. Improving farmyard poultry production in Africa: Interventions and their economic assessment [Internet]. Proceedings of a final research coordination meeting organised by the joint FAO/IEA Division of Nuclear techniques in Food and Agriculture. 2006. http://www-pub.iaea.org/mtcd/publications/pdf/te_1489_web.pdf
15. Alexander DJ. Newcastle disease and other avian paramyxoviruses. OIE Rev Sci Tech. 2000; 19 (2):443–62. https://doi.org/10.20506/rst.19.2.1231 PMID: 10935273
16. FAO. Family poultry development-Issues, opportunities and constraints. Vol. 12, Animal Production and Health. 2014.
17. Alders R, dos Anjos F, Bagnol B, Fumo A, Mata B, Young M. Controlling Newcastle Disease in Village Chickens: A Training Manual. ACIAR Monograph No. 87; 2002.
18. Feikin DR, Audi A, Olack B, Bigogo GM, Polyak C, Burke H, et al. Evaluation of the optimal recall period for disease symptoms in home-based morbidity surveillance in rural and urban Kenya. Int J Epidemiol. 2010; 39(2):450–8. https://doi.org/10.1093/ije/dyp374 PMID: 20089695
19. Thumbi SM, Njenga MK, Marsh TL, Noh S, Otiang E, Munyua P, et al. Linking human health and livestock health: A "one-health" platform for integrated analysis of human health, livestock health, and economic welfare in livestock dependent communities. PLoS One. 2015; 10(3):1–18.
20. Apopo AA, Kariithi HM, Ateya LO, Binepal YS, Sirya JH, Dulu TD, et al. A retrospective study of Newcastle disease in Kenya. Trop Anim Health Prod. 2020; 52:699–710. https://doi.org/10.1007/s11250-019-02059-x PMID: 31501991
21. Mosites E, Thumbi SM, Otiang E, McElwain TF, Njenga M, Rabinowitz PM, et al. Relations between household livestock ownership, livestock disease, and young child growth. J Nutr. 2016; 146(5):1118–1124. http://jn.nutrition.org/cgi/doi/10.3945/jn.115.225961 PMID: 27075911
22. Mosites E, Aol G, Otiang E, Bigogo G, Munyua P, Montgomery JM, et al. Child height gain is associated with consumption of animal-source foods in livestock-owning households in Western Kenya. Public Health Nutr. 2017; 20(2):336–345. https://doi.org/10.1017/S136898001600210X PMID: 27515059
23. Campbell ZA, Otieno L, Shirima GM, Marsh TL, Palmer GH. Drivers of vaccination preferences to protect a low-value livestock resource: Willingness to pay for Newcastle disease vaccines by smallholder households. Vaccine. 2019; 37(1):118. https://doi.org/10.1016/j.vaccine.2018.11.058 PMID: 30478006
24. Marangon S, Busani L. The use of vaccination in poultry production. Rev Sci Tech. 2007; 26(1):264–74. PMID: 17633308
25. Komba EV, Mbyuzi AO, Mulangila RC. Adoption of I-2 vaccine in immunization of village chickens against Newcastle Disease Virus in southern Tanzania: Immune status of farmer vaccinated birds. J Agric Sci. 2012; 4(4).

26. Nega M, Moges F, Mazengia H, Zeleke GN, Tamir S. Evaluation of I-2 thermostable Newcastle Disease vaccine on local chickens in selected districts of western Amhara. Online J Anim Feed Res. 2012; 2 (3):244–248.

27. Lesnoff M. Reliability of a twelve-month retrospective survey method for estimating parturition and mortality rates in a traditional African livestock farming system. Rev Élev Méd Vét Pays Trop 2009; 62:49–57.

28. Nyaga P. (2008). Poultry sector country review: Kenya. In FAO Poultry Sector Country Review. Rome: Food and Agriculture Organization; 2007. http://agris.fao.org/agris-search/search.do?recordID=XF2006446635.

29. Olwande PO, Ogara WO, Okuthe SO, Muchemi G, Okoth E, Odindo MO, et al. Assessing the productivity of indigenous chickens in an extensive management system in Southern Nyanza, Kenya. Trop Anim Health Prod. 2010; 42(2):283–8. https://doi.org/10.1007/s11250-009-9418-4 PMID: 19680773

30. Fisher RJ. Social desirability bias and the validity of indirect questioning. J Consum Res. 1993; 20 (2):303–315. https://doi.org/10.2307/2489277

Appendix 9.3: Vaccination of household chicken results in a shift in young children's diet and improves child growth in rural Kenya

PNAS RESEARCH ARTICLE | ECONOMIC SCIENCES AGRICULTURAL SCIENCES

OPEN ACCESS

Vaccination of household chickens results in a shift in young children's diet and improves child growth in rural Kenya

Elkanah Otiang[a,b,c,1], Jonathan Yoder[d,e], Shanthi Manian[d,e], Zoë A. Campbell[f], Samuel M. Thumbi[a,b,c,d], Lucy W. Njagi[a], Philip N. Nyaga[a], and Guy H. Palmer[c,d,g,1]

Edited by Xiang-Jin Meng, Virginia Polytechnic Institute and State University, Blacksburg, VA; received December 23, 2021; accepted April 1, 2022

Childhood growth faltering remains unacceptably high in sub-Saharan Africa. Rural communities dependent on household food production with limited off-farm income or liquid assets to bridge seasonal food availability are especially vulnerable. A cross-sectional survey in Siaya County, Kenya identified 23.5 and 4.8% of children under 5 y of age as stunted and wasted, respectively, using height-for-age *Z* (HAZ) scores to detect stunting and weight-for-height *Z* (WHZ) scores for wasting. Although these households are classified as living in poverty or extreme poverty with very limited off-farm income, households commonly have on-farm resources that could be developed to improve nutrition. While 95% of these households have chickens and consumption of eggs was shown to increase childhood growth by an average of 5%, the average flock size is small and constrained by high mortality due to infectious disease. We hypothesized that interventions to relieve this constraint would translate into household decisions influencing the diets and growth of children. Here, we show that vaccination of chickens against Newcastle disease has a causal impact on children's consumption of animal source foods rich in protein and micronutrients relative to a high-carbohydrate, grain-based diet. Children in treatment households (chicken vaccination) showed overall increases in scores for both HAZ and WHZ relative to control households, benefiting both girls and boys. The findings demonstrate the impact of directing interventions at common on-farm assets managed by women in rural communities and support programs to enhance productivity at the household level.

child growth | nutrition | household decisions | animal source foods

Child growth failure (stunting, wasting, and underweight in children under the age of 5) remains a major burden on the development of individuals, families, communities, and nations (1). Dramatic reductions in child growth failure are required to meet the World Health Organization's Global Nutrition Targets of a reduction in stunting by 40% and wasting to less than 5% by 2025 (2, 3). Similarly, meeting the United Nations' Sustainable Development Goals is dependent on these reductions as nutrition underlies 12 of the individual goals (4).

Growth failure, especially stunting and wasting, remains a major health and development challenge in rural western Kenya (5–7). A 2014 cross-sectional survey of 597 households in Siaya County identified that 23.5 and 4.8% of children under 5 y of age were stunted and wasted, respectively, using analyses of height-for-age *Z* (HAZ) scores to detect stunting and weight-for-height *Z* (WHZ) scores for wasting (6). Although the majority of these households are classified as living in poverty or extreme poverty with very limited off-farm income, households commonly have on-farm resources that could be developed to improve nutrition and growth outcomes (8, 9). While 95% of these households have chickens and consumption of eggs was shown to increase childhood growth by a mean of 5%, the average flock size is small (∼10, only half of which are potential sources of eggs or meat; the rest are young chicks), and only 16% of children over the age of 6 mo were reported to have consumed eggs in the 3 d prior to the study survey (7, 9). In a follow-up longitudinal census of chickens and decision-making in 1,908 households within the same community, mortality was identified as the primary constraint on flock size, representing 60% of all chicken losses, as opposed to a voluntary household decision to sell, consume, or gift chickens to meet household needs or to maintain a desired flock size based on optimizing the input cost of household labor or other management resources (10). The high mortality of chicks constrained growth of the flock both in total numbers and in composition, as fewer chicks survived to maturity and productivity. We hypothesized that interventions to relieve this constraint of involuntary loss due to infectious disease would translate into increased consumption of protein-rich diets by children and ideally, into improved growth outcomes.

Significance

This randomized, controlled trial demonstrates that by relieving a constraint on household nutritional assets, here through reducing chicken mortality through vaccination, households make dietary choices for young children that increase consumption of protein- and micronutrient-rich foods and decrease relative consumption of high-carbohydrate, low-protein grains. The study provides causal evidence that this shift in diet results in improved height for age, a key measure of childhood stunting. Given the high prevalence of childhood growth failure in rural Africa, these results highlight the potential to increase the utility of a common household animal asset to reduce the burden of childhood stunting in these communities.

Author contributions: E.O., Z.A.C., S.M.T., L.W.N., P.N.N., and G.H.P. designed research; E.O., S.M.T., and G.H.P. performed research; E.O., J.Y., S.M., S.M.T., and G.H.P. analyzed data; J.Y. and G.H.P. wrote the paper; and L.W.N. and P.N.N. edited the paper.

The authors declare no competing interest.

This article is a PNAS Direct Submission.

[1]To whom correspondence may be addressed. Email: elotiang@gmail.com or gpalmer@wsu.edu.

This article contains supporting information online at http://www.pnas.org/lookup/suppl/doi:10.1073/pnas.2122389119/-/DCSupplemental.

Published June 6, 2022.

In a two-arm randomized, controlled trial, we first determined whether vaccination against Newcastle disease virus (NDV), widely considered to be the predominant infectious cause of mortality in free range scavenging chickens globally (11, 12), would result in increased flock size. Quarterly vaccination over an 18-mo period resulted in an increase in average flock size from 11.63 ± 0.70 chickens at enrollment to 13.06 ± 0.29 chickens, a significantly greater increase ($P = 0.0026$) as compared with the unvaccinated arm of the trial (13). Based on this intervention, we analyzed whether NDV vaccination translated to increased children's consumption of animal source foods (ASFs) rich in protein and micronutrients relative to a high-carbohydrate, low-protein, grain-based diet. Furthermore, we determined whether a shift in consumption affected childhood growth. Herein, we present the results of this analysis and discuss the results in the context of maximizing the utility of a commonly held household resource in addressing childhood growth failure in rural Africa.

Results

Household Participation. Fig. 1 provides detail on household selection and allocation to the treatment or control arms of the study, participation at baseline, and follow-up during the course of the trial. Not all households provided food consumption data and anthropometric measurements at every quarter, and some are not represented in the final quarter of the 18-mo trial (*SI Appendix*, Fig. S1, Supplementary Text S1, and Table S1) due to respondent or child absence, illness, or conflicting household activities following three attempts within the scheduled week. Of six planned data collection visits per child, the average number of completed visits per child is 4.24 in the treatment arm (1,307 records total) and 4.32 in the control arm (1,380 records total). Statistical analyses indicated no systematic sample selection affecting the results; nonetheless, inverse probability weighting was applied to minimize potential selectivity bias in estimation results (*Materials and Methods* and *SI Appendix*, Supplementary Text S1 and Tables S2 and S3). Final datal cleaning defines the number of records used for analysis (Fig. 1); each record is linked to an individual child within a household as some households had more than one enrolled child.

Demographics and Nutritional Status of Children at Baseline. Household demographics and nutritional status at baseline are provided in Table 1. Based on HAZ and WHZ determination, stunting and wasting were present in 18.25 and 2.72% of the children, respectively. There were no significant differences between the treatment and control households at baseline (stunting: treatment households, 17.8%; control households, 18.6%; $P = 0.793$; wasting: treatment households, 3.5%; control households, 2.0%; $P = 0.24$). Using middle upper arm circumference (MUAC) measurements collected at enrollment, 8.6% of children suffered acute malnutrition: 2.8% with severe acute malnutrition (SAM) and 5.8% with moderate acute malnutrition (MAM).

Impact of Breastfeeding and Child Age on Food Consumption. At baseline, 50.1% of children were breastfeeding (treatment households, 49.6%; control households, 50.7%; $P = 0.76$). In Fig. 2, food intake is represented by the sum of servings in the

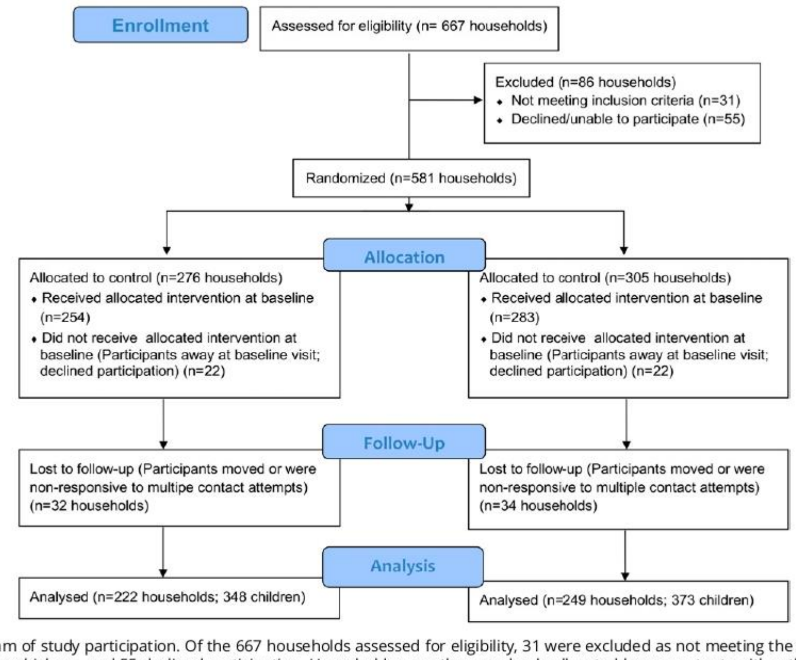

Fig. 1. Consort diagram of study participation. Of the 667 households assessed for eligibility, 31 were excluded as not meeting the criteria of having a child <3 y of age and keeping chickens, and 55 declined participation. Households were then randomly allocated by computer to either the treatment arm of the study (vaccination of chickens against NDV plus parasite control) or the control arm (parasite control only). Households not present at baseline or that declined at baseline were excluded from the study, resulting in 254 treatment households and 283 control households entering the study. Households that did not participate in any subsequent quarters after three contact attempts were excluded from analysis, resulting in 222 treatment households with 348 children and 249 control households with 373 children for analysis. All households received the intervention (NDV vaccination and parasite control or parasite control alone) every quarter. Households that were assessed for child diet and growth on all quarterly visits and on multiple but not all quarterly visits were retained in the study (*SI Appendix*, Fig. S1, Supplementary Text S1, and Table S1). The procedures used to detect and control for any bias in intermittent participation are detailed in *Materials and Methods* and in *SI Appendix*, Supplementary Text S1 and Tables S2 and S3.

Table 2. Consumption of ASF, fruits, vegetables, and grains

Independent variable†	Food category consumption (dependent variable)‡			
	ln(A)	ln(F)	ln(V)	ln(G)
Treatment × Months in Trial	0.013*	0.013	0.000	−0.006
Treatment	−0.038	−0.140	−0.144*	0.003
Time in Trial (mo)	0.015***	0.034***	0.000	0.002
MAM @ one or more visits	−0.119	−0.404**	−0.186	0.067
SAM @ one or more visits	−0.596****	−0.598*	−0.291	−1.186**
Time since MAM	0.000	0.008	0.009	0.004
Time since SAM	0.053****	0.040	0.011	0.107**
>18 mo × Breastfeeding	−0.179*	−0.253*	−0.113****	−0.092*
≤18 mo × Not breastfeeding	−1.664***	−3.541***	−4.586***	−6.240***
≤18 mo × Breastfeeding	−2.461***	−4.037***	−5.095***	−6.542***
>18 mo × Child Age	0.007	−0.027	0.002	−0.003
≤18 mo × Child Age	0.234***	0.445***	0.556***	1.054***
>18 mo × Child Age sq.	0.000	0.000	0.000	0.000
≤18 mo × Child Age sq.	−0.007**	−0.016***	−0.016***	−0.040***
ln(Per capita income)	0.016****	0.002	0.008	−0.004
Female Child	0.016	−0.001	0.024	−0.039
Mother's (Caregiver§) Age	0.001	−0.006**	0.004**	0.002****
Mother's (Caregiver§) Education Level	0.185***	0.126****	0.031	0.062**
cosmonth	0.019	0.314***	0.073**	0.049**
sinmonth	0.035	−0.238***	−0.093***	0.017
Intercept	0.001	0.810	1.063***	2.321***

The number of observations for all equations is 2,549. The estimation method is the structural equation model with random effects at the child level. Food intake regressions are estimated simultaneously with the HAZ and WHZ regressions presented in Table 3 using the Stata 17 gsem routine with random effects at the individual child level. *Statistically significant at $P < 0.1$; **statistically significant at $P < 0.05$; ***statistically significant at $P < 0.01$; ****statistically significant at $P < 0.15$.

†Independent variables include the NDV vaccination treatment group (the control group is the base case), time since first diagnosis of MAM (MUAC of 115 to 124 mm) or SAM (MUAC of <115 mm), time in the trial in months, child age and breastfeeding status (18 mo+ × not breastfeeding is the base case), logarithm of per capita income, gender of the child, mother's age, mother's education level, and month of the year (to reflect seasonality). The use of 18 mo as an age reference is based on our data that the transition from breastfeeding as the primary source of child nutrition through a period of increased intake of other food sources occurs up to month 18 (Fig. 2). × represents the interaction between two variables.

‡Dependent variables are the natural logarithm of the number of servings for each food consumption category (ASF [A], fruits [F], vegetables [V], grains [G]).

§Principal respondent and responsible for child care.

older children, and breastfeeding children consume relatively less ASF than nonbreastfeeding children of the same age group (*SI Appendix*, Table S4).

Impact of Household Income on Food Group Consumption. Because the dependent variables in the food intake regression (Table 2) and per capita income (household income divided by number of household members) are both in logarithmic form, the parameters associated with per capita income represent elasticities: the percentage change in the number of total food servings in response to a 1% increase in per capita income. Elasticity is positive for ASF, fruit, and vegetables, while the elasticity is negative for grains (Table 2).

Additional perspective on how income affects substitution between food groups is provided from a fractional multinomial logit model presented in *SI Appendix*, Supplementary Text S2 and Table S4. Consumption of ASF and vegetables is significantly higher relative to grains ($P < 0.068$ and $P < 0.031$, respectively) for households with higher incomes, with the increase in ASF consumption higher than that of vegetables (*SI Appendix*, Table S4).

Impact of Season on Food Group Consumption. Using the sine and cosine of the scaled month of year to capture seasonality, consumption of fruits, grains, and vegetables shows significant seasonal intake cycles ($P < 0.01$) (Table 2), whereas there is little evidence of seasonality for ASF. This is consistent with seasonal production and the limited ability to store fruit, vegetables, and to a lesser extent, grains at the household level as compared with more consistent availability of ASF. This seasonal relationship for fruits and vegetables but not ASF is also observed in the fractional multinomial logit model, where consumption is referenced relative to grains (*SI Appendix*, Table S4).

Impact of Household NDV Vaccination of Chickens on Food Group Consumption. The effect of being in the treatment group (a household with quarterly NDV vaccination of all chickens) is captured by the parameter associated with the *Treatment Group* × *Time in trial* ($T \times t$). A positive parameter estimate means that consumption for that food group increases over the course of the trial faster (and more) in the treatment group relative to intake of that food group by children in the control arm of the study (base case). ASF consumption increased faster in the treatment group by 1.3% per month (based on a parameter estimate of 0.013; $P = 0.089$) (Table 2). This implies a 24% increase in ASF consumption relative to the control group by the end of the trial. In contrast, estimated consumption of grains decreased in the treatment group relative to the control group over the course of the trial. These results suggest that the vaccination of chickens against NDV may have made ASF more available to the point that households substituted children's food toward ASF and away from the other food groups, especially grains.

We examined this substitution effect associated with NDV treatment using the fractional multinomial logit model as well (*SI Appendix*, Supplementary Text S2 and Table S4). The results indicate that the share of ASF relative to grains increases at a significantly faster rate in the treatment group ($P = 0.081$), supporting the conclusion that NDV vaccination is inducing or allowing households to substitute toward ASF and away from grains as the trial proceeds. In contrast, there are no significant changes in the shares of consumed fruits and vegetables between the treatment and control groups ($P = 0.450$ and $P = 0.645$, respectively) (*SI Appendix*, Table S4).

Impact of Food Consumption on Child Growth. For HAZ, higher average ASF consumption over the course of the trial has a relatively large, statistically strong, positive impact (0.165; $P = 0.014$) for children over 18 mo of age (Table 3). This result is consistent with the cumulative importance of protein consumption on HAZ as a growth measure (14). In contrast, average grain consumption over previous visits had a negative effect on HAZ for older children (−0.234; $P = 0.095$). For WHZ, the food consumption effects were mixed. Most statistically significant effects were negative, although the effect of vegetable consumption for older children was positive and significant (0.037; $P = 0.027$). The rest of the effects of food intake on older children are negative and/or are not significant at conventional test sizes. The coefficients on *Time since MAM* and *Time since SAM* are positive, suggesting that the intervention in response to MAM and SAM positively affected WHZ but not HAZ. Consistent with the seasonality of fruit and vegetable consumption (Table 2 and *SI Appendix*, Table S4), there appear to be seasonal effects on WHZ (Table 3). Note that sin(*month*) and cos(*month*) were omitted for HAZ because of the longer time frame of HAZ development.

Breastfeeding patterns have qualitatively similar effects on WHZ and HAZ (Table 3). The base case for comparison is children >18 mo of age not ever breastfed during the study period for HAZ and not currently breastfed for WHZ. Conditional on food consumption, older children being breastfed have lower HAZ and WHZ than older children not being breastfed. Younger children being breastfed have higher estimated WHZ than younger children not being breastfed, conditional on food intake. In contrast, older children being breastfed have lower WHZ than older children not being breastfed (−0.102; $P = 0.085$). These differences may reflect other unobserved differences in the diets of older breastfed children that affect these outcomes. Surprisingly, younger children never breastfed during the study have higher HAZ than younger children who have been breastfed [0.644 − (−0.685) = 1.329; $P = 0.001$]. This unintuitive result could be due to the coarseness of the variable "Has breastfed" (i.e., it may not capture either breastfeeding in the "Never breastfed" children that

Table 3. Impacts on child growth: WHZ and HAZ

	Child *Z* score (dependent variable)	
Independent variable[†]	HAZ	WHZ
Treatment × Month in Trial	0.007	0.006
Treatment	−0.205*	0.108
Time in Trial (mo)	0.023***	0.006****
MAM @ one or more visits	−1.217***	−0.777***
SAM @ one or more visits	−0.569*	−0.900***
Time since MAM	−0.009	0.020****
Time since SAM	−0.013	0.049**
>18 mo × ln([Avg]A)[‡]	0.165**	−0.019
≤18 mo × ln([Avg]A)	−0.029	−0.017
>18 mo × ln([Avg]F)	0.070	−0.022**
≤18 mo × ln([Avg]F)	0.000	−0.029
>18 mo × ln([Avg]V)	0.081	0.037**
≤18 mo × ln([Avg]V)	−0.036	−0.092***
>18 mo × ln([Avg]G)	−0.234*	−0.003
≤18 mo × ln([Avg]G)	−0.061	−0.111***
>18 mo × Breast(fed) [feeding][§]	−0.338**	−0.102*
≤18 mo × Not breast(fed) [feeding]	0.644	0.349**
≤18 mo × Breast(fed) [feeding]	−0.685****	0.434***
Child Age	−0.064***	−0.022**
Child Age sq.	0.001**	0.000*
ln(Per capita income)	0.028**	−0.007
Female Child	0.154****	0.076
Mother's (Caregiver's) Age	0.004	0.000
Mother's (Caregiver's) Education Level	0.096	0.050*
cosmonth		0.052***
sinmonth		−0.045***
Intercept	0.095	−0.179

WHZ and HAZ scores are the dependent variables. The number of observations for all equations is 2,549. The estimation method is the structural equation model with random effects at the child level. Regressions are estimated (simultaneously with food category regressions in Table 2) using the gsem routine in Stata 17 with random effects at the individual child level. *Statistically significant at $P < 0.1$; **statistically significant at $P < 0.05$; ***statistically significant at $P < 0.01$; ****statistically significant at $P < 0.15$.

†Independent variables include the NDV vaccination treatment group (the control group is the base case), time since first diagnosis of MAM (MUAC of 115 to 124 mm) or SAM (MUAC of <115 mm), time in the trial in months, child age and breastfeeding status (18 mo+ × not breastfeeding is the base case), logarithm of per capita income, gender of the child, mother's (or caregiver's) age, mother's (or caregiver's) education level, and month of the year (to reflect seasonality). The use of 18 mo as an age reference is based on our data that the transition from breastfeeding as the primary source of child nutrition through a period of increased intake of other food sources occurs up to month 18 (Fig. 2). × represents the interaction between two variables.

‡Average servings reported over past visits were used for the HAZ regression, and current reported servings (last 3 d) were used for the WHZ regression. *SI Appendix*, Supplementary Text S3 and Table S5 have a robustness analysis of this specification. For the WHZ regression, ln(Food Group) is the logarithm of servings for the current visit. For the HAZ regression, ln(Food) is the logarithm of average servings for that food category reported in all household visits to date.

§For the WHZ regression, Breastfed and Not Breastfed indicate whether a child is currently being breastfed. For the HAZ regression, they indicate whether a child has ever been breastfed during the trial period to date.

occurred just prior to study enrollment or the duration of breastfeeding in the "breastfed" children during the study). These effects are qualitatively the same across different specifications, including breastfeeding status (*SI Appendix*, Supplementary Text S3 and Table S5).

Direct, Indirect, and Total Impacts of Household NDV Vaccination of Chickens on Child Growth. The increased child consumption of ASF in treatment households, both absolute and relative to grains (Table 2 and *SI Appendix*, Table S4), represents the primary driver of the effects of NDV vaccination on growth outcomes mediated through changes in food consumption, denoted as indirect effects. In addition, the WHZ and HAZ regressions show that there are also methodologically defined direct effects of treatment that are captured by our data but are not accrued to the nutritional data we collected. These may be treatment group impacts on nutrition not included or accurately measured in our assessments as well as unidentified behavioral or household management changes linked to being in the treatment arm of the study. Eq. **3** and associated discussion (*Materials and Methods*) describe how the parameter estimates (Tables 2 and 3) are used to calculate the indirect food consumption effects and the total effects of being in a treatment household on child growth.

The estimated direct, calculated indirect, and calculated total treatment effects measured at 18 mo after initiation of treatment are provided in Table 4 under the headings "Average treatment effect: HAZ" and "Average treatment effect: WHZ." For HAZ, the estimated direct monthly NDV vaccine treatment effect is 0.0071 ($P = 0.380$), the indirect effect is 0.0045 ($P = 0.084$), and the total monthly effect is 0.0116 ($P = 0.170$). While the estimated direct effect is larger in magnitude than the indirect effect, the indirect effect leading to an increase in HAZ through food intake is statistically more compelling. Over the trial period of 18 mo, these estimates translate to a direct effect of 0.1269, an indirect effect of 0.0817, and a total effect of 0.2087 (*P* values are the same as corresponding monthly effects). For WHZ, the monthly estimated increase in WHZ through the direct effect is 0.0060 ($P = 0.253$; 0.1074 over 18 mo), the indirect effect is −0.0005 (−0.0095 over 18 mo; $P = 0.286$), and the total estimated effect is 0.0054 (0.0979 over 18 mo; $P = 0.302$).

These results suggest a statistically clear positive effect of the NDV vaccination of household chickens on HAZ through changes in food consumption and especially, ASF consumption. The total effect of the NDV treatment on WHZ is positive but statistically weaker. Figs. 3 and 4 provide additional perspective on these outcomes. Fig. 3 illustrates total treatment effects on HAZ (Fig. 3, *Upper*) and WHZ (Fig. 3, *Lower*) in the context of the distribution of children in the control group at the end of the trial (trial participation through a minimum of 15 mo). Average total treatment effect for HAZ is about 17% of one SD of HAZ [$100 \times (0.2087/1.223)$] of the control group at the end of the trial. The average total treatment effect for WHZ is about 10.6% of one SD of WHZ [$100 \times (0.0979/0.9221)$]. Fig. 4 illustrates the estimated time path of average treatment effects over the course of the trial. The indirect component of the treatment effect for HAZ is the positive maroon line (Fig. 4, *Left*), with slope 0.0045 ($P = 0.084$) representing the cumulative indirect effect of observed food consumption differences between treatment and control groups over the course of the trial. The maroon shaded triangle is the associated 90% CI for the cumulative indirect effect. Estimated direct effects are larger but statistically weaker, suggesting there are other impacts of the NDV treatment process that are not captured by our data on food consumption and these regressions. In contrast, the indirect effect of NDV treatment on WHZ through food consumption (Fig. 4, *Right*) is negative, although small and not statistically different from zero (monthly effect = slope = −0.0005; $P = 0.286$). This weak indirect effect is more than compensated for by the positive direct effects. The opposing indirect treatment effects, strongly positive for HAZ and weakly negative for WHZ, suggest an apparent trade-off in growth characteristics due to the substitution away from grains toward other food groups, especially ASF, as shown in *SI Appendix*, Table S4. The estimated direct and total treatment effects are represented by the blue dashed lines and the green dashed lines, respectively, in each panel (Fig. 4).

Effects of Trial Participation. *Time in trial* (months) captures any general effect on food consumption that might result from participating in the trial that is not otherwise captured as a treatment effect, such as the effect of chicken parasite control in both arms of the trial or the effects of other time-varying factors not otherwise controlled for in the regressions, whether they are induced by the trial itself or not (e.g., changes in weather or community economic conditions). The direct and indirect effects of *Time in trial* are calculated using parameter estimates from Tables 2 and 3 (based on Eq. **5** in *Materials and Methods*) and are summarized in Table 4. The monthly direct effect on HAZ of being in the trial is 0.0227 ($P = 0.008$), the indirect monthly effect through food consumption changes during the trial is 0.0045 ($P = 0.043$), and the total monthly effect is 0.0272 ($P = 0.001$). The total effect on HAZ over the full trial period for children 18 mo or older is 0.498 ($P = 0.001$). Time in trial effects for WHZ are mixed (Table 4). Monthly direct and total effects are positive but not statistically significant. Indirect monthly effects of time in trial on WHZ are −0.0011 ($P = 0.045$). The total effect over the trial period on WHZ for children 18 mo and older is 0.0951 ($P = 0.219$).

Impact of Nutritional Interventions for Acute Malnutrition. If nutrition counseling after a finding of MAM or counseling and food supplementation after a finding of SAM had positive effects over time on the intake of a food category, then the associated parameters [*Time since first MAM* and *Time since first SAM* represented in Eqs. **1** and **2** as $(M_m \times t_m)$] (*Materials and Methods*) should be positive (after controlling for baseline MAM and SAM incidence). Table 2 shows that the signs of the parameters on *Time since MAM* and *Time since SAM* are all positive in the food intake equations, although significance is weak. The two strongest effects are due to *Time since SAM*; ASF increases by about 5.3% per month (0.053; $P = 0.129$), and grains increase by about 10% per month ($P = 0.014$). These results suggest that counseling effectively promotes ASF consumption within the household as the supplemental feeding provided upon an SAM diagnosis during the trial did not have a specified ASF component. As with the NDV vaccination treatment effect, the nutritional interventions have both indirect and direct effects on HAZ and WHZ, shown in Table 4 (bottom two blocks of results; direct effects are also shown in the HAZ and WHZ regression results in Table 3). The estimated effects of MAM and SAM treatments on HAZ are all negative. This is somewhat unintuitive, especially given the effect of SAM on ASF consumption in the food regressions. Including *MAM @ one or more visits* is intended to control for the fact that HAZ will tend to be lower in children diagnosed with MAM. It could be that this summary statistic may not be

Table 4. Estimated direct, indirect, and total effects of treatment (NDV vaccination), time in trial, and MAM and SAM diagnoses and interventions

		Estimate	$P > \|z\|$	90% CI	
Average treatment effect: HAZ†					
Direct	Average monthly	0.0071	0.380	−0.0062	0.0203
Indirect	Average monthly	0.0045*	0.084	0.0002	0.0089
Total	Average monthly	0.0116	0.170	−0.0023	0.0255
Direct	Full trial	0.1269	0.380	−0.1111	0.365
Indirect	Full trial	0.0817*	0.084	0.0039	0.1596
Total	Full trial	0.2087	0.170	−0.0416	0.459
Average treatment effect: WHZ†					
Direct	Average monthly	0.0060	0.253	−0.0026	0.0145
Indirect	Average monthly	−0.0005	0.286	−0.0013	0.0003
Total	Average monthly	0.0054	0.302	−0.0032	0.0141
Direct	Full trial	0.1074	0.253	−0.0470	0.2619
Indirect	Full trial	−0.0095	0.286	−0.0243	0.0052
Total	Full trial	0.0979	0.302	−0.0582	0.254
Time in trial: HAZ‡					
Direct	Average monthly	0.0227***	0.008	0.0086	0.0368
Indirect	Average monthly	0.0045**	0.043	0.0008	0.0082
Total	Average monthly	0.0272***	0.001	0.0135	0.0409
Direct	Full trial	0.4088**	0.008	0.1546	0.6629
Indirect	Full trial	0.0810**	0.043	0.0151	0.1470
Total	Full trial	0.4898***	0.001	0.2432	0.7364
Time in trial: WHZ‡					
Direct	Average monthly	0.0063	0.138	−0.0007	0.0134
Indirect	Average monthly	−0.0011**	0.045	−0.0019	−0.0002
Total	Average monthly	0.0053	0.219	−0.0018	0.0124
Direct	Full trial	0.1143	0.138	−0.0126	0.2411
Indirect	Full trial	−0.0191**	0.045	−0.0348	−0.0034
Total	Full trial	0.0951	0.219	−0.0322	0.2225
Time since MAM and SAM diagnosis intervention effect: HAZ‡					
Direct	MAM	−0.0095	0.607	−0.0398	0.0208
Indirect	MAM	0.0003	0.954	−0.0076	0.0081
Total	MAM	−0.0092	0.617	−0.0395	0.0211
Direct	SAM	−0.0133	0.562	−0.0510	0.0244
Indirect	SAM	−0.0124	0.429	−0.0382	0.0134
Total	SAM	−0.0257	0.312	−0.0675	0.0161
Time since MAM and SAM diagnosis intervention effect: WHZ‡					
Direct	MAM	0.0202	0.130	−0.0018	0.0421
Indirect	MAM	0.0001	0.870	−0.0011	0.0013
Total	MAM	0.0203	0.129	−0.0017	0.0423
Direct	SAM	0.0494*	0.042	0.0094	0.0893
Indirect	SAM	−0.0017	0.708	−0.0094	0.0059
Total	SAM	0.0476*	0.056	0.0067	0.0886

Average treatment effect estimates for HAZ and WHZ are based on Eq. **3** (*Materials and Methods*) and the applicable parameter estimates from regressions presented in Tables 2 and 3. Time in trial effects were calculated based on Eq. **5** and parameter estimates from Tables 2 and 3. MAM and SAM diagnosis and intervention effects (applied to both the primary treatment group and the control group) are calculated based on Eq. **4** (*Materials and Methods*) and parameter estimates from Tables 2 and 3. All estimates in this table and associated *P* values and CIs were generated using Stata 17 nlcom routine. *Statistically significant at $P < 0.10$; **statistically significant at $P < 0.05$; ***statistically significant at $P < 0.01$.

†Treatment households only.

‡All households.

fully controlling for this baseline characteristic sufficiently. In contrast, however, the effects of time since MAM and SAM have consistently positive effects on WHZ, except for the indirect effect of SAM. Despite this, the total estimated monthly increase in WHZ in response to an intervention for SAM is about 4.8% ($P = 0.056$). MAM interventions were information based, and SAM interventions were implemented on a quarterly timescale (between visits).

Effect of household income. Households with higher per capita income show greater ASF consumption (Table 2), and conditional on food intake, HAZ is higher in higher-income households (0.028; $P = 0.045$) (Table 3). The total direct and indirect effect of household income on HAZ is 0.032 ($P = 0.018$). The direct and total effects of higher income on WHZ were not statistically significant ($P = 0.373$ and $P = 0.270$, respectively).

Effect of child gender. Girls had weakly higher HAZ and WHZ as compared with boys (Table 3). The higher HAZ scores as measured directly with the *Female Child* indicator variable are in addition to the implied HAZ benefits from weakly higher ASF consumption (Table 2) and in ASF consumption relative to grains (*SI Appendix*, Table S4) shown for girls. The total

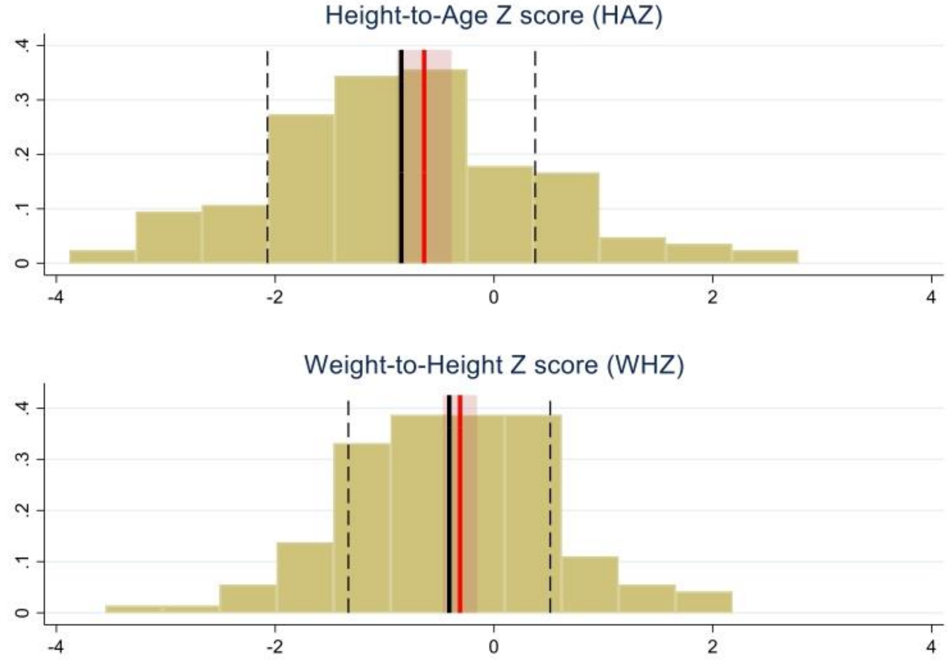

Fig. 3. Overall effect of being in a treatment group household on child growth relative to the control group. The histograms represent the distributions of HAZ and WHZ scores for control group age over 18 mo of age for those who have been in the trial at least 15 mo. Thick solid black vertical lines are control group means, and the dashed black lines are one SD from the distribution mean (SDs are 1.22 for HAZ and 0.922 for WHZ). The red lines represent the mean Z score plus the total effect of treatment (direct plus indirect effects; 0.209 for HAZ and 0.098 for WHZ). The lightly shaded maroon areas are 90% CIs for the total treatment effect; P values for the total effects are $P = 0.170$ for HAZ and $P = 0.302$ for WHZ. Estimates for average total effects and CIs are taken from Table 4.

direct and indirect estimated difference in HAZ of girls relative to boys is 0.15, indicating that growth benefits accrued at least equally to girls as well as boys.

Discussion

Despite measurable progress over the past two decades, childhood growth faltering, especially stunting, remains unacceptably high in many countries and communities in Africa (1, 6). Rural communities highly dependent on household food production and with limited off-farm income or liquid assets to bridge seasonal food availability are especially vulnerable (15, 16). The study community in Siaya County, Kenya reflects this vulnerability. We have focused on chickens as a widely held autochthonous resource, managed at the household level by women, that can provide foods high in protein and rich in micronutrients that are critically important in preventing stunting (17–19). We initiated the vaccine intervention recognizing that the pathway from veterinary vaccination to increased chicken productivity through household decision-making to improved nutrition and child growth is complex and impacted by multiple known and unknown factors. Nonetheless, our data show statistically significant impacts on the intermediate measure of increased ASF consumption, estimated at a 24% increase over the course of the trial, that translated into improved child growth parameters. This supports concerted

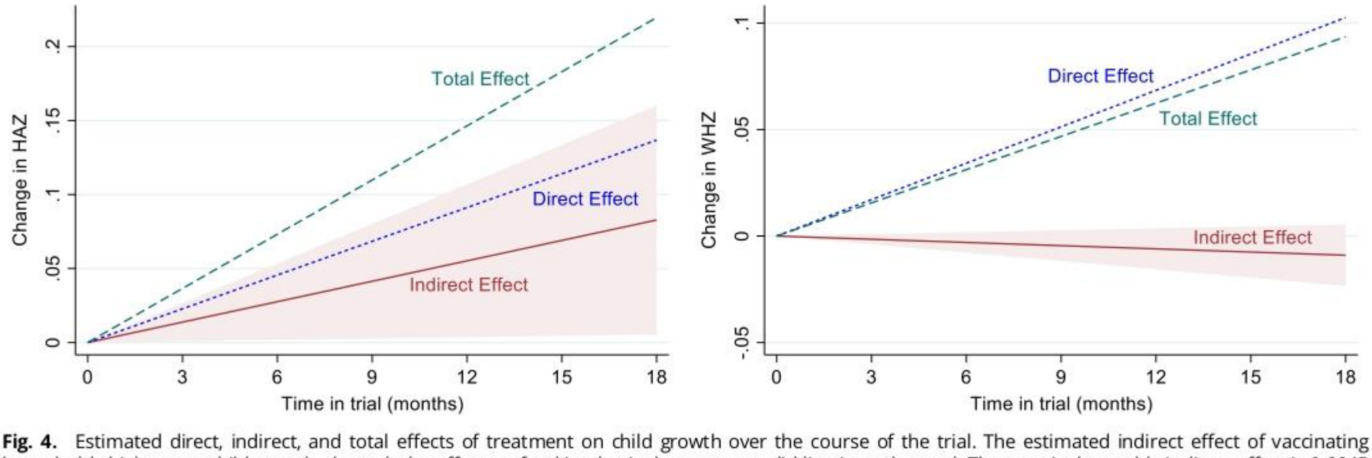

Fig. 4. Estimated direct, indirect, and total effects of treatment on child growth over the course of the trial. The estimated indirect effect of vaccinating household chickens on child growth, through the effect on food intake, is the maroon solid line in each panel. The marginal monthly indirect effect is 0.0045 ($P = 0.084$) for HAZ and -0.0005 ($P = 0.286$) for WHZ (Table 4). These estimates are the slopes of the indirect effect lines in each panel. The maroon shaded triangles are 90% CIs for the indirect effects. Direct effects (blue dashed lines) and total effects (green dashed lines) are shown without CIs to limit visual complexity. The monthly total treatment effects are 0.0116 ($P = 0.17$) for HAZ and 0.0054 ($P = 0.30$) for WHZ, which are the slopes of the green dashed lines for HAZ and WHZ, respectively (Table 4). Fig. 3 provides a complementary perspective on the total treatment effect. Table 4 estimates are based on regression results presented in Tables 2 and 3.

NDV vaccination of household chickens, for which willingness to pay studies indicate strong household interest (20), as part of a multipronged approach to enhance childhood nutrition and reduce stunting in rural communities where chickens are a common household resource.

The gains measured in treatment households relative to control households very likely underestimate the impact of NDV vaccination on ASF consumption and childhood growth due to several inherencies of the trial design. First is that, for ethical reasons, the control households also received an intervention: medication for parasites in their chickens (as did the treatment households in addition to NDV vaccination). This medication plus any unmeasured veterinary advice provided at the time of treatment is reflected in the increase in flock size in the control households over baseline (13) and may be a driver of the significant effect of "time in trial" effects on ASF intake reported here. The second is that vaccine was only delivered at quarterly intervals—sufficient to maintain immunity in previously vaccinated chickens but misses all chicks hatched in the interim, the age group most susceptible to dying from NDV (11, 21). This challenge is addressed in the final discussion paragraph. Third, all households received data on their children's growth and ad hoc dietary and poultry management guidance that may have influenced their decisions on both flock management and children's diets. Notably, girls appear to be at least equal beneficiaries of the substitution toward ASF consumption. Although not linked to a treatment effect, overall the coincident increase in both HAZ and WHZ in girls relative to boys is discrepant from prior studies in other regions of Kenya, where girls had significantly lower HAZ and WHZ (22). We speculate that this reflects the primary role of women in management of household poultry linked to dietary consumption choices (23–26).

Translating the increase in flock size gained through NDV vaccination to a change in a young child's diet is mediated through a household decision by, in rural Kenya, usually maternal or a female relative (6, 7, 17, 24, 27). The most significant increase in flock size in treatment households as compared with control households was due to an increase in laying hens (13). Based on prior studies of indigenous chicken productivity in this region, an increase in one hen per flock would result in average production of six to seven eggs per month, with a potential increase of 6 g of protein per egg (28, 29). The impact of increased egg production on child growth is supported by a study demonstrating that each instance of child consumption of an egg during a prior 3 d period was significantly linked to an increase in child height (7). In the present study, the positive and negative signs of the treatment coefficient in the ASF and grains regressions on HAZ and WHZ, respectively, are consistent with the substitution away from high-carbohydrate, low-protein grains toward high-protein ASF, leading a higher-protein diet but fewer total calories. This implies that although the treatment provided households with more in-home, accessible protein, there is still a trade-off in resource-constrained households: a small drop in WHZ for a larger increase in HAZ. This type of apparent substitution is not inevitable but is a common behavioral response in resource-constrained decision-making. In contrast to vegetables and fruits, ASF are much less influenced by season (30) and thus, may enhance HAZ to a greater degree due to HAZ reflecting growth over time.

Unidentified effects on child growth of being in a NDV treatment household, here denominated as direct treatment effects in the WHZ and HAZ regressions, are captured by the *Treatment Group* × *Time in trial* interaction term. As child growth measures, collection of data on the type and quantity of foods consumed by the child, and antiparasite medications given to the flocks were common to both treatment and control groups and we rigorously controlled for possible bias in household participation over time (*SI Appendix*, Fig. S1, Supplementary Text S1, and Tables S1–S3), we posit that the direct effects appear to derive from either households observing vaccination or the increased time that the animal health technicians were on the premises due to the additional time requirement for vaccination. The latter provided more time for household members to interact with the animal health technicians and potentially receive additional advice regarding poultry and livestock husbandry, crop management, or other issues affecting food production and availability. The direct observation of NDV vaccination may also have had an effect. Campbell et al. (31) identified that knowing a neighbor who vaccinated his or her chickens had the most significant impact on the decision of a given household to vaccinate. Whether households in the treatment group that routinely observed the vaccination process invested more of their own resources into poultry management was not captured in our study.

In addition to the effect of being in a treatment household on child growth mediated through food consumption, household income has consistent positive impacts on ASF consumption, ASF and vegetable consumption relative to grains, and HAZ. This result is consistent with prior findings that ASF and vegetables tend to be more income responsive than other food groups, while grains are less responsive to income or decline as a share of food expenditures (32, 33). Importantly, even modest income increases can help bridge the seasonal fluctuations in on-farm food availability and avoid periods of undernutrition in young children at the time they are most vulnerable to childhood stunting (15, 16, 34).

Prior studies have established a link between the health of poultry and livestock in rural smallholder farms and both decreased human disease and enhanced childhood growth (6, 9). While previous studies have documented the impacts of livestock ownership on child nutrition (6, 35, 36), we provide evidence on the impact of livestock health interventions. The positive impact of NDV vaccination on flock size and its translation into increased ASF consumption and improved child growth provide a compelling rationale for the minimal investments required for widespread vaccination in rural households. The high percentage of chicken ownership in our study site in Siaya County in western Kenya is representative of rural households across Africa and other rural, low-income regions within Asia and South and Central America. A willingness to pay study indicated that households in Tanzania with similar characteristics as in western Kenya were willing to pay roughly twice the market price for NDV vaccines, which are readily available in east Africa (20). However, the small number of chickens per household disincentivizes market-based delivery mechanisms, and the opportunity cost to individual household members, usually women, to travel and purchase vaccines is a disincentive (20, 21). This additional burden on women is consistent with studies examining the impact of early childhood interventions on mothers (37). Subsidizing animal health technicians to deliver vaccines to households is proposed to overcome these disincentives and provide a pathway to enhance household poultry productivity with impacts on household well-being and reduced childhood growth faltering.

Materials and Methods

Ethical Approval. The research was approved by the Ethical and Animal Care and Use Committees (Scientific Steering Committee protocol no. 3159) of the

Kenya Medical Research Institute and by the University of Nairobi Faculty of Veterinary Medicine Animal Use and Bioethics Committee to conduct research on enhancing childhood nutrition and growth. Informed consent covered the study overview, objectives of the study, potential benefits to the household participants, potential risks to the participants and mitigation steps, contact information for both medical and veterinary personnel involved in the study, and contact information for the ethical review committees. Participation was voluntary, and the decision was at the household level; there was no community-wide presentation or interdependency among households that may pressure a given household to participate. Both oral and written consents (in Luo, the language of the community) were obtained from the household head, and written consent has been retained.

Trial Design and Participant Selection. The longitudinal, randomized, controlled trial was conducted in the Rarieda subcounty of Siaya County in western Kenya within a health and demographic surveillance system (HDSS) site run by the Kenya Medical Research Institute and the US Centers for Disease Control and Prevention (38). HDSS data from December 2016 (6 mo prior to the study initiation) indicated 667 potentially eligible households meeting the criteria of 1) chicken ownership, 2) a child 3 y or younger, and 3) location within a 5.5-km radius of St. Elizabeth Lwak Mission Hospital, which could provide nutritional support for children identified as suffering from acute malnutrition.

Vaccination. Animal health technicians delivered the intervention (vaccination of chickens against NDV) quarterly, independent of the food consumption and anthropometric data collection. All chickens in treatment households were vaccinated with two drops of NDV AVIVAX I-2 thermostable vaccine ($10^{9.7}$ egg infectious doses per milliliter) intranasally or intraocularly (depending on the chicken's age at recruitment) and then, every 3 mo thereafter (13). AVIVAX I-2 is a freeze-dried live attenuated Newcastle disease vaccine prepared from the La Sota strain manufactured by the Kenya Veterinary Vaccines Production Institute. In all households, treatment and control, all chickens were also treated with endo- and ectoparasiticides, piperazine citrate, and carbaryl every 3 mo by the animal health technicians.

Data Collection. Assessments were conducted at the time of enrollment as a baseline and then, approximately quarterly for up to 18 mo to collect anthropometric data on children, the type and quantity of foods consumed, and household socioeconomic data. In the event the mother (or principal caregiver/respondent) or child was not present at the time of the scheduled visit, the interviewers returned twice more within the week. If the third attempt was unsuccessful, the household visit was scheduled for the next quarter. Child growth was measured using a Shorrboard for length ($<$2 y) and height ($\geq$2 y) and caregiver/child standing scale for weight. MUAC was assessed using three standardized colored tapes: red ($<$115 mm) indicating SAM, which triggered referral to a program that provided vitamin A and fortified maize flour and nutritional counseling; yellow (115 to 124 mm) indicating MAM, which triggered nutritional counseling; and green ($>$125 mm), which was considered healthy growth and the caregiver was encouraged to continue with nutritious feeding. For dietary assessment, caregivers were requested to recall the type of food, quantity, and the number of times the child was fed each type of the food in the 3 d prior to the interview. The interviewers asked about each specific food and provided a standardized set of containers to assist in estimating the quantity of each food item. Socioeconomic data included the mother's (or principal caregiver's) age and level of education; the number of family members; and household income consisting of both on-farm and off-farm earnings, where Kenya shillings were converted to US dollars using the year 2016 average as an exchange rate reference (1 US dollar = 101.50 shillings) and discounted at an annual rate of 3.5%. All data were collected by community health interviewers in the local language (Luo), entered onto an electronic data capture tool, downloaded, and stored in a Microsoft Access database. All datasets underwent validation and consistency checks to identify and resolve errors before they were merged using unique household identifiers on each of the participating households.

Blinding. Households were not notified of which group they belonged to but were informed (oral and written) that their chickens would receive treatment to prevent disease. The enumerators who conducted the household interviews and collected both food consumption data and the anthropometric measurements were blind to the control and treatment group allocation. The animal health technicians and the enumerators were never on the premises at the same time to prevent enumerators from identifying the group. Household allocation data were unmasked only at the end of the study by the investigators.

Data Analysis. Child height, age, and sex were referenced to the World Health Organization standards to create continuous measures for HAZ score and WHZ score using Epi Info (39) upon importation of anthropometric variables. Stunting and wasting were defined as greater than two SDs below the World Health Organization reference mean for HAZ and WHZ, respectively. Analyses on all the relational factors over the study period were done using STATA, version 17 (40).

Statistical regression analysis was performed to 1) model the determinants of food consumption by children over the course of the study, including treatment effects, and 2) model the determinants of the biometric outcomes and WHZ and HAZ scores, including food consumption and treatment effects. The response variables in the food consumption regressions are the natural logarithms of the number of food servings for each of four food groups: ASF, fruits, vegetables, and grains. The food intake regression equations can be written as

$$\ln(S_f) = \alpha_0^f + \alpha_t^f t + \alpha_{Tt}^f\,(T \times t) + \sum_m \alpha_m^f M_m^e + \alpha_{mt}^f (M_m \times t_m) + \sum_{k=1}^{K} \alpha_k^f X_k + \varepsilon_f, \qquad [1]$$

where $\ln(S_f)$ is the logarithmic transformation of the four food group servings, with $f \in (A,\ V,\ F,\ G)$ representing ASF, vegetables, fruit, and grains, respectively. Greek letters are parameters to be estimated, t is time since first household visit [*Time in Trial* (months)], and T is an indicator variable equaling one if a household is in the *Treatment Group* and zero otherwise. M represents two malnutrition indicators: MAM and SAM; the subscript m identifies MAM and SAM indicator variables, the superscript e indicates whether a child was ever diagnosed with MAM or SAM during the full trial, the superscript t_m measures time since first record of MAM or SAM, respectively. X represents other control variables (including interaction terms) in each equation, and ε_f is a random error term.

We hypothesize that a treatment effect of NDV vaccination of household chickens on a child's diet would accumulate over the course of the trial. To capture this effect, we created an interaction variable $(T \times t)$ in Eq. **1** by multiplying T and t. Because T equals zero for control households and one for treatment households, $(T \times t)$ equals the time in trial for treatment households and is zero for control households. The control group is, therefore, the base case, and α_t^f represents the direction and rate of change in consumption of food group f over the course of the trial in the control group. The parameter α_{Tt}^f associated with $(T \times t)$ represents the difference in the rate of change in food consumption in the treatment group relative to consumption in the control group. If $\alpha_{Tt}^f > 0$, consumption of food category f is increasing relative to consumption in the control group. The treatment indicator is included directly in each food and Z score equation to control for conditional baseline differences in treatment and control groups in relation to each dependent variable. The time in trial variable t captures changes in the dependent variable over the course of the trial that are not attributable to the treatment itself.

The category of variables $M_m \in (MAM, SAM)$ in Eq. **1** relates to two forms of malnutrition: MAM and SAM. The related category $M_m^e \in (MAM[ever],\ SAM[ever])$ indicates whether a child was ever diagnosed with MAM or SAM during the trial and is used to control for unobserved baseline conditions that contribute to MAM and SAM risk. The base case is no acute malnutrition. The interaction term $(M_m \times t_m)$ takes the value of zero for each individual until a first finding of either MAM or SAM, after which it counts months (in increments of days) since this first finding until the end of the trial to assess whether these interventions had measurable effects on outcomes over time.

Food consumption history in our data reflects the transition from breastfeeding as the primary source of child nutrition through a period of increased intake of other food sources up to month 18 (Fig. 2). This age-dependent transition was captured in the food consumption regressions (represented generally by X_k in Eq. **1**) by utilizing an indicator variable *>18 mo* taking a value of one if a child was over 18 mo old at the time of a household visit and zero otherwise and interacting this variable with child age, age squared (to capture the diminishing increase in servings through 18 mo), and a variable indicating whether a child was currently being breastfed (*Breastfed* = 1, *Not Breastfed* = 0). Further regressors in Eq. **1** include the natural logarithm of per capita household income, an indicator for female child (vs. male), the age and education level of the mother or principal caregiver, and a sine/cosine pair (trigonometric functions of month) to capture seasonality in food intake.

HAZ and WHZ regressions were estimated simultaneously with food intake equations represented by Eq. **1**. These regressions can be represented as

$$Z = \beta_0^z + \beta_t^z t + \beta_{Tt}^z (T \times t) + \sum_{\mathrm{m}} \beta_m^{e,z} M_m^e + \beta_{mt}^z (M_m \times t_m) + \sum_{f=1}^{4} \beta_f^z \ln(S_f) + \sum_{k=1}^{K} \beta_k^z X_k + \varepsilon_z, \quad [2]$$

where $Z \in (WHZ,\ HAZ)$ are weight-to-height and height-to-age measures, respectively, and the rest of the content shown in Eq. **2** is as described for Eq. **1**. However, there are differences between regressions [**1**] and [**2**]. First, Eq. **2** includes the four categories of food intake, $\ln(S_f)$, as explanatory variables to capture food intake effect on biometric outcomes. Second, because height to age tends to reflect the cumulative effects of nutrition during the entire growth path of a child, while weight to height tends to more reflect recent nutrition, the explanatory variables in these regressions differ between the two regressions. Third, we include current breastfeeding status in the WHZ regression, but in the HAZ regression, we use an indicator of whether a child was ever breastfed (*Never Breastfed* vs. *Breastfed Ever)* during the trial prior to a given visit. We provide robustness analysis for specifying these differences in HAZ and WHZ regressors in *SI Appendix*, Supplementary Text S3 and Table S5.

Our data contain up to six records per child, one for each household visit. We apply a random effects model to account for unobserved similarities in children/households between visits. The error component of the regressions can be represented as $\varepsilon_{y,it} = u_i + v_{it}$, where $y \in (z, f)$ and the i and t indices identify child and visit number, respectively (subscripts are omitted from Eqs. **1** and **2** to minimize notational clutter). Robust SEs are clustered by individual child. The food intake and *Z*-score regressions (six equations) were estimated simultaneously in Stata 17 using the gsem routine with random effects for each individual child (40). To account for systematic sample selectivity bias that might accompany this attrition, we use inverse probability weights (41–43) based on predicted probabilities generated from a Probit regression shown and discussed in *SI Appendix*, Supplementary Text S1 and Table S3.

Of particular interest are the effects of treatment on food intake, the effects of food intake on biometric scores, and the effects of treatment on *Z* scores conditional on food intake. Eqs. **1** and **2** allow for estimation of what we refer to as direct effects of treatment on food intake and biometric scores as well as indirect effects of treatment on *Z* scores through measured food consumption effects. The indirect effects are defined as the effects of treatment on *Z* scores through food consumption as measured in these regressions. The direct effect is defined as the measured effect of the treatment variables $(T \times t)$ and $(M_m \times t_m)$ included directly in the *Z* regressions and represents effects of treatment on nutritional outcomes not otherwise represented by our food consumption data and therefore, not captured in the food intake–related parameter estimates in regressions [**1**] and [**2**]. These effects are methodologically "direct" in the sense that they are captured directly in Eq. **2** parameters, but they may reflect complex and varied pathways from treatment to nutritional outcomes for the children in the study. The full effect of treatment on $Z \in (WHZ,\ HAZ)$ can be described as

$$\text{Total Treatment Effect on } Z = \text{Direct Effect on } Z + \text{Indirect effect on } Z \text{ through food intake } (f).$$

The sum of direct and indirect treatment effects is calculated mathematically based on Eqs. **1** and **2** as

$$\frac{\mathrm{d}Z}{\mathrm{d}T} = \left(\frac{\partial Z}{\partial Tt} + \sum_{f=1}^{4} \frac{\partial Z}{\partial \ln(S_f)} \frac{\partial ln(S_f)}{\partial Tt} \right) \frac{\partial Tt}{\partial T} = \left(\beta_{Tt}^Z + \sum_{f=1}^{4} \alpha_{Tt}^f \beta_f^z \right) t, \quad [3]$$

where T is a treatment indicator variable equaling one if a child is in a treatment household and zero otherwise. The direct effects of treatment on Z are β_{Tt}^Z, and the indirect effects of treatment on Z through measured impacts of treatment on food consumption are the sum of the effects of treatment on food intake (α_{Tt}^f) and the effects of food intake on Z (β_f^z). These effects are conditional on the duration of $t = (\textit{Time in Trial}\ (\text{months}))$, up to a maximum of $t = 18$.

Household and child participation may affect both the control and treatment groups over the course of the trial. If a child was found to be moderately or acutely malnourished, they were referred to a therapeutic feeding program regardless of whether they were in the treatment or control group. A mathematically analogous calculation for estimating the direct effects of MAM and SAM intervention after diagnosis is calculated as

$$\frac{dZ}{dt_m} = \frac{\partial Z}{\partial M_m t_m} \frac{\partial M_m t_m}{\partial t_m} + \sum_{f=1}^{4} \frac{\partial Z}{\partial \ln(S_f)} \frac{\partial \ln(S_f)}{d \partial t_m} \frac{\partial M_m t_m}{\partial t_m} = \beta_{mt}^z + \sum_{f=1}^{4} \beta_f^z \alpha_{mt}^f, \quad [4]$$

where $(M_m \times t_m)$ in Eqs. **1** and **2** is abbreviated as $M_m t_m$; $M_m \in (MAM,\ SAM)$; and $\frac{\partial Z}{\partial M_m t_m} \frac{\partial M_m t_m}{\partial t_m}$ and $\frac{\partial \ln(S_f)}{\partial M_m t_m} \frac{\partial M_m t_m}{\partial t_m}$ are estimated by one parameter each in each equation in Tables 2 and 4, respectively, associated with *Time since [first] MAM* and *Time since [first] SAM*.

There are other known and unknown reasons that food intake and *Z* scores might be affected by trial participation independent of being in a treatment or control household. The full change in biometric scores *Z* that occurs over time during the course of study participation is

$$\frac{dZ}{dt} = \frac{\partial Z}{\partial t} + \sum_{f=1}^{4} \frac{\partial Z}{\partial \ln(S_f)} \frac{\partial \ln(S_f)}{\partial t} = (\beta_t^z + \beta_{Tt}^z T) + \sum_{f=1}^{4} \beta_f^z (\alpha_t^f + \alpha_{Tt}^f T).$$

For the control group, the treatment indicator $T = 0$, so the "time in control group," which nets out the treatment effect, simplifies to

$$\left. \frac{dZ}{dt} \right|_{T=0} = \beta_t^z + \sum_{f=1}^{4} \alpha_t^f \beta_f^z. \quad [5]$$

Eq. **5** is the baseline for potential effects of trial participation on biometric outcomes. Because the control group status is the base case in the regression, it reflects effects of known and unknown factors affecting both groups. We designate this as the time in trial effect. Total effects of other regressors in both food and *Z*-score regressions are calculated from regression parameters in an analogous fashion. These indirect and total effects are calculated using the Stata nlcom routine (40).

Data Availability. All study data are included in the article, the *SI Appendix*, and on the Open Science Framework (OSF) at https://osf.io/m4vs2/?view_only=1428f4bb179b4d7bbde8a00c81d95b5d (or see ref. 44).

Author affiliations: [a]College of Agriculture and Veterinary Sciences, University of Nairobi, Nairobi 00100, Kenya; [b]Center for Global Health Research, Kenya Medical Research Institute, Kisumu 40100, Kenya; [c]Washington State University Global Health–Kenya, Nairobi 72938-00200, Kenya; [d]Paul G. Allen School for Global Health, Washington State University, Pullman, WA 99164; [e]School of Economic Sciences, Washington State University, Pullman, WA 99164; [f]International Livestock Research Institute, Nairobi 00100, Kenya; and [g]University of Nairobi Institute of Tropical Infectious Diseases, Nairobi 72938-00200, Kenya

1. Local Burden of Disease Child Growth Failure Collaborators, Mapping child growth failure across low- and middle-income countries. *Nature* **577**, 231–234 (2020).
2. M. de Onis *et al.*, The World Health Organization's global target for reducing childhood stunting by 2025: Rationale and proposed actions. *Matern. Child Nutr.* **9** (suppl. 2), 6–26 (2013).
3. World Health Organization, Global nutrition targets 2025 (2014). https://www.who.int/publications/i/item/WHO-NMH-NHD-14.2. Accessed 8 August 2021.
4. G. Grosso, A. Mateo, N. Rangelov, T. Buzeti, C. Birt, Nutrition in the context of the Sustainable Development Goals. *Eur. J. Public Health* **30** (suppl. 1), i19–i23 (2020).
5. E. Bloss, F. Wainaina, R. C. Bailey, Prevalence and predictors of underweight, stunting, and wasting among children aged 5 and under in western Kenya. *J. Trop. Pediatr.* **50**, 260–270 (2004).
6. E. Mosites *et al.*, Relations between household livestock ownership, livestock disease, and young child growth. *J. Nutr.* **146**, 1118–1124 (2016).

7. E. Mosites *et al.*, Child height gain is associated with consumption of animal-source foods in livestock-owning households in Western Kenya. *Public Health Nutr.* **20**, 336–345 (2017).
8. KIPPRA, Siaya County Integrated Development Plan 2018-2022 (2018). https://repository.kippra.or.ke/handle/123456789/1218. Accessed 8 August 2021.
9. S. M. Thumbi *et al.*, Linking human health and livestock health: A "one-health" platform for integrated analysis of human health, livestock health, and economic welfare in livestock dependent communities. *PLoS One* **10**, e0120761 (2015).
10. E. Otiang *et al.*, Mortality as the primary constraint to enhancing nutritional and financial gains from poultry: A multi-year longitudinal study of smallholder farmers in western Kenya. *PLoS One* **15**, e0233691 (2020).
11. R. Alders *et al.*, "Controlling Newcastle Disease in Village Chickens: A Training Manual" in *ACIAR Monograph No. 86* (Australian Centre for International Agricultural Research, Canberra, ACT, Australia, 2002).
12. L. W. Njagi, *et al.*, Prevalence of Newcastle disease virus in village indigenous chickens in varied agro-ecological zones in Kenya. *Livest. Res. Rural Dev.* **22**, 95 (2010).
13. E. Otiang *et al.*, Impact of routine Newcastle disease vaccination on chicken flock size in smallholder farms in western Kenya. *PLoS One* **16**, e0248596 (2021).
14. J. L. Hulett *et al.*, Animal source foods have a positive impact on the primary school test scores of Kenyan schoolchildren in a cluster-randomised, controlled feeding intervention trial. *Br. J. Nutr.* **111**, 875–886 (2014).
15. Livestock for Health, "Seasonality of malnutrition: Community knowledge on patterns and causes of undernutrition in children and women in Laisamis, Marsabit County, Kenya" (Rep., FOA, Food and Agricultural Organization of the United Nations, UNICEF, United Nations Children's Fund, Washington State University, 2021).
16. A. Catley, R. Lotira, C. Hopkins, Hidden peaks: Women's knowledge on seasonality and route causes of child malnutrition in Karamoja, Uganda and their programming preferences (2018). https://karamojaresilience.org/wp-content/uploads/2021/05/201811_krsu_hidden_peaks_nutrition_online.pdf. Accessed 1 March 2022.
17. L. A. F. Akinola, A. Essien, Relevance of rural poultry production in developing countries with special reference to Africa. *Worlds Poult. Sci. J.* **67**, 697–705 (2011).
18. L. H. Allen, D. K. Dror, Effects of animal source foods, with emphasis on milk, in the diet of children in low-income countries. *Nestle Nutr Works SE* **67**, 113–130 (2011).
19. H. Asare, A. Rosi, M. Faber, C. M. Smuts, C. Ricci, Animal-source foods as a suitable complementary food for improved physical growth in 6 to 24-month-old children in low- and middle-income countries: A systematic review and meta-analysis of randomised controlled trials. *Br. J. Nutr.* **3**, 1–11 (2022).
20. Z. A. Campbell, L. Otieno, G. M. Shirima, T. L. Marsh, G. H. Palmer, Drivers of vaccination preferences to protect a low-value livestock resource: Willingness to pay for Newcastle disease vaccines by smallholder households. *Vaccine* **37**, 11–18 (2019).
21. A. E. Absalón, D. V. Cortés-Espinosa, E. Lucio, P. J. Miller, C. L. Afonso, Epidemiology, control, and prevention of Newcastle disease in endemic regions: Latin America. *Trop. Anim. Health Prod.* **51**, 1033–1048 (2019).
22. M. Ndiku, K. Jaceldo-Siegl, P. Singh, J. Sabaté, Gender inequality in food intake and nutritional status of children under 5 years old in rural Eastern Kenya. *Eur. J. Clin. Nutr.* **65**, 26–31 (2011).
23. E. H. Gueye, Women and family poultry production in rural Africa. *Dev. Pract.* **10**, 98–102 (2000).
24. E. F. Guèye, Gender aspects in family poultry management systems in developing countries. *Worlds Poult. Sci. J.* **61**, 39–46 (2005).
25. S. Nordhagen, R. Klemm, Implementing small-scale poultry-for-nutrition projects: Successes and lessons learned. *Matern. Child Nutr.* **14** (suppl. 3), e2676 (2018).
26. K. R. Wilson, Who reaps the benefits? Exploring access and social exclusion among village chicken producers in Kenya. *Gend. Place Cult.* **28**, 1196–1200 (2021).
27. M. T. Ruel, H. Alderman; Maternal and Child Nutrition Study Group, Nutrition-sensitive interventions and programmes: How can they help to accelerate progress in improving maternal and child nutrition? *Lancet* **382**, 536–551 (2013).
28. M. Chepkemoi *et al.*, "Nutritional diversity of meat and eggs of five poultry species in Kenya" in *2015 JKUAT Scientific Conference* (Jomo Kenyatta University of Agriculture and Technology, Nairobi, Kenya, 2016), pp. 124–131.
29. P. O. Olwande *et al.*, Assessing the productivity of indigenous chickens in an extensive management system in southern Nyanza, Kenya. *Trop. Anim. Health Prod.* **42**, 283–288 (2010).
30. P. Kristjanson *et al.*, R., Are food insecure smallholder households making changes in their farming practices? Evidence from East Africa. *Food Secur.* **4**, 381–397 (2012).
31. Z. A. Campbell, T. L. Marsh, E. A. Mpolya, S. M. Thumbi, G. H. Palmer, Newcastle disease vaccine adoption by smallholder households in Tanzania: Identifying determinants and barriers. *PLoS One* **13**, e0206058 (2018).
32. L. Colen *et al.*, Income elasticities for food, calories and nutrients across Africa: A meta-analysis. *Food Policy* **77**, 116–132 (2018).
33. S. Desiere, Y. Hung, W. Verbeke, M. D'Haese, Assessing current and future meat and fish consumption in sub-Sahara Africa: Learnings from FAO Food Balance Sheets and LSMS household survey data. *Glob. Food Sec.* **16**, 116–126 (2018).
34. J. L. Leroy, M. Ruel, J. P. Habicht, E. A. Frongillo, Linear growth deficit continues to accumulate beyond the first 1000 days in low- and middle-income countries: Global evidence from 51 national surveys. *J. Nutr.* **144**, 1460–1466 (2014).
35. R. Rawlins, S. Pimkina, C. B. Barrett, S. Pedersen, B. Wydick, Got milk? The impact of Heifer International's livestock donation programs in Rwanda on nutritional outcomes. *Food Policy* **44**, 202–213 (2014).
36. D. Headey, K. Hirvonen, J. Hoddinott, Animal sourced foods and child stunting. *Am. J. Agric. Econ.* **100**, 1302–1319 (2018).
37. D. K. Evans, P. Jakiela, H. A. Knauer, The impact of early childhood interventions on mothers. *Science* **372**, 794–796 (2021).
38. D. R. Feikin *et al.*, Evaluation of the optimal recall period for disease symptoms in home-based morbidity surveillance in rural and urban Kenya. *Int. J. Epidemiol.* **39**, 450–458 (2010).
39. Centers for Disease Control and Prevention, Epi Info (2019). https://www.cdc.gov/epiinfo/index.html. Accessed 5 December 2021.
40. StataCorp, *Stata Statistical Software: Release 17* (StataCorp LLC, 2021).
41. J. M. Wooldridge, Inverse probability weighted M-estimators for sample selection, attrition, and stratification. *Port. Econ. J.* **1**, 117–139 (2002).
42. M. Huber, Identification of average treatment effects in social experiments under alternative forms of attrition. *J. Educ. Behav. Stat.* **37**, 443–474 (2012).
43. J. Weuve *et al.*, Accounting for bias due to selective attrition: The example of smoking and cognitive decline. *Epidemiology* **23**, 119–128 (2012).
44. E. Otiang, Vaccination of household chickens results in a shift in young children's diet and improves child growth. Open Science Framework. https://osf.io/m4vs2/?view_only=1428f4bb179b4d7bbde8a00c81d95b5d. Deposited 8 August 2021.