

Bridging Implementation Science and Demography to Understand the Dynamics of Child
Survival in Tanzania during the Millennium Development Goal Era

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Abstract

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Although on the decline, the global burden of preventable child mortality remains concentrated in sub-Saharan Africa. In recent decades, governments in the region have sought to ameliorate this disparity by introducing numerous evidence-based interventions known to improve child survival. In Tanzania, these strategies were effective, as evidenced in 2015 by the country's achievement of the child survival Millennium Development Goal (MDG). Contemporaneously, however, fertility levels in Tanzania stalled despite anticipation that a transition would ensue in the wake of widespread child mortality reduction. Understanding the determinants and mechanisms through which child survival interventions in Tanzania achieved impact can guide strategy to address global inequalities in child mortality risk. Furthermore, knowledge about how mortality and fertility processes are linked in the context of rural Tanzania in the MDG era (2000-15) can help programs influence the course of population change in this region. In this dissertation, we bridge theory and methods from demography and implementation science to address those objectives. In Chapters 2 and 3, we use data from a cluster-randomized trial of the impact of a community-health worker (CHW) program that was conducted from 2011-15 in rural settings of Tanzania that comprised sentinel areas of the Ifakara and Rufiji Demographic Surveillance Systems (HDSS).

The first paper uses data from a 2011 facility survey that quantified the implementation of maternal, newborn and child health (MNCH)-related inputs at the health facility level. We combined weighted-additive methods, principal components analysis and Bayesian mixed effects modelling to reduce these data into three scales of the implementation strength exerted by groups of facilities on communities from which their clients came. We linked these scales to longitudinal data on the survival of children that were born in these communities in 2011 and followed up through 2015. We fit survival time regression models which reported that increases in the strength of MNCH implementation gauged by two scales were associated with child mortality risks that were appreciably lower. We conclude that strong implementation of MNCH services can enhance child survival, and that routine data from facilities can be used to detect small area inequalities in the effectiveness of MNCH coverage and to measure the impacts of health systems strengthening.

In Chapter 3, we report on a mixed methods analysis that explains the outcomes of the CHW intervention. Program effects on MNCH service utilization, childhood morbidity and sick childcare seeking were evaluated using difference-in-difference regression analysis with outcomes measured through pre- and post-intervention household surveys in intervention and comparison trial arms. A qualitative process evaluation was conducted from 2012-2014. The CHW program reduced incidence of childhood illness and improved access to timely and appropriate sick childcare; however, there was no effect on MNCH service utilization. The positive outcomes were achieved through mechanisms that triggered high levels of acceptability of CHW among community-members, and motivation and confidence of the CHW. Implementation factors that generated these effects were related to the engagement of communities in program startup; the training, remuneration and configuration of supervision and support to the CHW from the health system and community. Null results were attributed to the fragile health systems context and health

systems' limited capacity for strategic change. We conclude that strategies that strengthen and align communities' and health systems core capacities, as well as their ability to learn, adapt and integrate evidence-based interventions, are needed to maximize the MNCH impact of CHW.

Our third paper, in Chapter 4, we employ generalized hazard regression analysis to examine the fertility response to child mortality using longitudinal data compiled by the Ifakara and Rufiji HDSS from 2000-2015. The analysis adjusts for individual and contextual covariates, including annual cluster-level child mortality rates. Results show that child mortality accelerates parity progression. Time to conception is most reduced if a child dies during its subsequent birth interval, representing the "replacement" effect of child loss on fertility. Deaths occurring during prior birth intervals were, to a lesser magnitude, associated with accelerated time to conception during future intervals, which is consistent with hypothesized "insurance" effects of anticipating future child loss. Investigation of interactions suggests that contrary to evidence from similar studies and expectations raised by classical demographic theory, insurance effects on fertility tend to be greater, and replacement effects lower, in communities in which child mortality is relatively rare. We conclude that future research should interrogate this finding and generate evidence on ways that programs can optimize women's opportunities and abilities to adapt their fertility intentions, and achieve them, in the context of demographic change in transitional societies, like Tanzania.

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Glossary of Acronyms

AFT	Accelerated failure time
ANC	Antenatal care
BIS	Basic emergency obstetric and newborn care implementation strength
CHMT	Council Health Management Team
CHW	Community health worker
CONSORT	Consolidated Standards of Reporting Trials
CPM	Causal pathway model
DDT	Demographic Transition Theory
DHS	Demographic and Health Survey
EBI	Evidence based intervention
EPI	Extended Program on Immunization
FGD	Focus group discussion
HDSS	Health and Demographic Surveillance System
HR	Hazards ratio
HRR	Hazards ratios ratio (Ratio of hazard ratios)
ICF	Informed Consent Form
IDI	In-depth Interview
IHI	Ifakara Health Institute
IMCI	Integrated Management of Childhood Illness
IRB	Institutional Review Board
ITN	Insecticide treated nets
LMIC	Low and middle income countries
MDG	Millennium Development Goal
MICE	Multiple Imputation with Chained Equations
MMAM	Mpango wa Maendeleo wa Afya ya Msingi (Primary Health Care Services Development Plan)
MNCH	Maternal, newborn and child health

MOSHW	Ministry of Health and Social Welfare
NIMR	National Institutes of Medical Research (of Tanzania)
PC	Principal Component
PCA	Principal Components Analysis
PHC	Primary Health Care
PNC	Postnatal care
SARA	Service Availability and Readiness Assessment
sSA	sub-Saharan Africa
UNICEF	United Nations Children's Fund
WAJA	Wawezashaji wa Afya ya Jamii (Community Health Enablers)

We shall not cease from exploration, and the end of all our exploring will be to arrive where we started and know the place for the first time.

T. S. Eliot, from “Little Gidding,” *Four Quartets*

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Chapter 1:

Introduction

1. Background

The burden of childhood mortality is inequitably distributed throughout the world and is particularly high in sub-Saharan Africa (sSA).¹ The majority of these deaths can be prevented by the delivery of evidence-based primary health care (PHC) interventions², and although governments and development partners in the region have invested heavily in strengthening PHC systems, according to recent estimates, children in sSA are still between six and fifteen times more likely to die compared to children in other countries.³ The ramifications of PHC implementation challenges are far reaching. Not only do they drive high levels of preventable morbidity and mortality⁴; moreover, in doing so, they constrain the development trajectories of societies faced with increasing population pressures.⁵ Understanding the causes and consequences of child survival in the context of evolving PHC systems in sSA is crucial to formulating policies and programs that accelerate and broaden the equity of improvements and position societies to address the longer-term implications of changes in child mortality risk.

1.1 Tanzania during the Millennium Development Goal Era.

The experience of the United Republic of Tanzania provides a unique opportunity to explore the dynamics of PHC program implementation, child mortality and demographic transition.^{6,7} Between 2000 and 2015, the era of the Millennium Development Goals (MDG), the Government of Tanzania (GoT) invested considerably in minimizing preventable child deaths. These efforts included national policy reforms articulating commitment to improving maternal, newborn and child survival; vast increases in public expenditure on health; decentralization and sector wide basket funding to strengthen district's abilities to coordinate PHC; enhancing information systems

and planning tools to help districts address the burden of disease cost-effectively; and scaling up construction of PHC dispensaries and health centers throughout the country.⁸⁻¹⁰ Doing so allowed the introduction and scale up of new evidence-based interventions, such as the Integrated Management of Childhood Illness (IMCI), which facilitated adoption of new treatment policies that replaced less-effective first-line treatments with optimized approaches for addressing major causes of childhood death, importantly malaria, diarrhea and pneumonia, at the PHC level.¹¹⁻¹³ The IMCI program also assisted promotion of the use of preventive interventions that save children's lives, such as insecticide treated nets to avoid malaria, immunizations, child growth monitoring, exclusive breastfeeding and vitamin A and iron supplementation.¹⁴ In addition, GoT health sector reforms called for the establishment of a national cadre of community health workers (CHW) to extend the reach of these services to villages.^{8,15} To guide this initiative, the GoT commissioned studies to understand ways to introduce and deploy CHW to achieve that goal and evaluate the impact of CHW deployment on child survival.¹⁶

The child survival outcomes of Tanzania's investments were impressive. By 2015, the country had achieved the MDG 4 of reducing under-five year-old mortality to two-thirds of the 1990 level.¹⁷ Although this decline started in the earlier decade, after 2000 the trend accelerated and by 2015 the mortality rate of under-fives had declined by an annual rate of 8.5%.¹⁸

2. Motivation for new evidence generation

In the advent of precipitous child mortality reduction in Tanzania, numerous studies have set out to determine how this was achieved. Collectively, the findings of this research have attributed childhood mortality reduction to the interplay between policy and management reforms and the scale up of child survival EBI.⁷ These studies' dependency on retrospective and ecological study designs and cross-sectional data has tended to preclude more rigorous evaluations of the GoT's

PHC improvement strategies, and the relative role of specific interventions on child survival.^{7,14} Furthermore, there were few examinations of small area inequities in the effectiveness of PHC service coverage and longitudinal investigations into whether such variation is associated with child mortality risk.^{19,20} Since the 1970s, CHW have been central to PHC policy and practice in Tanzania; however, most evaluations of CHW programs in the country have lacked statistical rigor and components that explain the mechanisms through which they affect child survival and the determinants that shape whether those mechanisms succeed.^{21,22} Generating evidence at different levels of PHC systems, community- and facility-level, on the determinants and processes that enhance child survival is needed to sustain MDG era progress in Tanzania and expand it in areas of the country where mortality reduction is less pronounced.

There is a paucity of research in Tanzania, as well as throughout sSA, that evaluates the demographic impact of child mortality on reproductive health and fertility. According to the Demographic Transition Theory, and voluminous empirical evidence from around the world, there is a strong correlation between child mortality and fertility.²³⁻²⁶ Indeed, numerous studies have indicated that reduction in under-five deaths is a prerequisite for fertility decline. Yet the relative immobility of mean birth intervals and fertility rates in Tanzania from 2000-15, which fluctuated between 33 and 37 months and from 5.6 to 5.2 births, respectively, indicate that Tanzania may be an exception.^{27,28} To date, there are not any methodologically rigorous, longitudinal examinations of the relationship between child loss and fertility behavior in Tanzania, even though understanding this in the context of strengthening PHC programs is key.⁶ If reproductive behavior does not respond to changes in child mortality, then a likely consequence of strengthening PHC and scaling up child survival interventions is high fertility. If, on the other hand, it does, then the

success of family planning policies and programs may depend on the level of mortality and the effectiveness of PHC systems.²⁹

In this dissertation I draw upon data collected in three rural districts of Tanzania between 2000 and 2015 and report on analysis that fills these knowledge gaps. By bridging implementation and demographic research methods, this dissertation examines three critical issues: (1) Whether variation in effective coverage of different PHC services affects childhood mortality risk, (2) The effect of CHW deployment on child health and the generative process through which those effects are achieved, and (3) The impact of childhood mortality on fertility in the context of improving child survival. In addressing these topics, I will provide new and holistic evidence on the causes and consequences of child mortality that will benefit health policy and programs in Tanzania and similar settings.

3. Aims

To fill these knowledge gaps, this dissertation will pursue three aims:

Aim 1. In first paper, I link cross-sectional data from service availability and readiness assessments conducted in PHC facilities in three districts of Tanzania with longitudinal records of childbirth and survival in surveyed facilities' underlying population. With this, I develop scales of PHC service 'implementation strength' that gauge small area variation in effective coverage of different dimensions of facility-based PHC and examine the "dose response" relationship between differences in individual-level exposure to PHC implementation strength and child mortality risk. This will address the hypothesis that PHC implementation strength is inversely associated with child mortality and identify the service specific drivers of this relationship.

Aim 2. The second paper draws upon data from the baseline and endline household surveys of a cluster-randomized controlled trial of the impact a CHW intervention on maternal and child health and qualitative data from an embedded process evaluation. The analysis triangulates findings from a quantitative evaluation of the effect of CHW deployment on maternal and child health behaviors, care seeking and childhood morbidity with a qualitative analysis that explains the mechanisms of CHW impact and the barriers and facilitators that enabled, constrained, and moderated the levels of effect. This will test the proposition that CHW deployment accelerates child health gains and elucidate the processes and interplay of determinants that condition this relationship.

Aim 3. Using longitudinal demographic surveillance data on births and deaths that were obtained from households in the three districts of Tanzania, I will operationalize salient theoretical perspectives on the effect of child loss on reproductive behavior and rigorously examine the fertility response to under-five year-old mortality between 2000 and 2015 and whether this relationship is moderated by socio-economic factors and the child mortality context in which fertility behaviors unfold. Doing so will advance knowledge on the drivers of demographic transition in a unique setting where high fertility has persisted despite improvements in health and survival of children, give insight on the mechanisms through which child loss influences reproductive behavior, and illuminate implications relevant to population policies and programs.

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Chapter 2:

Measuring the strength of maternal, newborn and child health care implementation and its association with childhood mortality risk in three rural districts of Tanzania.

Abstract

This paper explores the association between the strength of maternal, newborn and child health (MNCH) provision and child survival in three rural districts of Tanzania between 2011 and 2015. We used data from a 2011 facility survey that quantified the implementation of MNCH-related inputs at the health facility level and a population-level household survey that measured the utilization of MNCH interventions to develop summary measures of the effective coverage of facility-based MNCH services. We reduced these domain specific effective coverage scores into three scales of implementation strength using principal components analysis and integrated them into scales of the collective implementation strength exerted by groups of facilities on communities from which their clients came using Bayesian mixed effects models. We linked these scales to longitudinal data on the survival of children that were born in the catchment areas of facilities during the period of the facility survey and followed up until December 31, 2015. We fit survival time models to estimate the relationship between MNCH implementation strength established in the study area during children's early infancy and their risk of dying before the end of the cohort. We found that children that lived in communities that received stronger levels of MNCH implementation were at a significantly lower risk of dying during childhood. Specifically, increases in the strength of implementation gauged by our first scale, which represented general facility readiness and the provision of antenatal, postnatal, and early childhood preventive services, were, on average, associated with risks of child mortality that were 0.61 times lower. Increases in implementation strength gauged by our second scale, which represented the provision of sick

childcare services, were, on average, associated with child mortality risks that were 0.54 times lower. We detected no significant child mortality response to implementation strength variation measured by our third scale, which represented intrapartum care provision. The findings reinforce the notion that strong implementation of antenatal, postnatal, early childhood preventive services and sick child care can accelerate child mortality reduction and suggest that routine data on service availability and readiness can be used to measure health systems strengthening and its impacts.

1. Introduction

In the past two decades, the spread of evidence-based child survival interventions has precipitated large-scale reductions in child mortality globally; however, considerable geographic disparities exist.^{1,2} Preventable loss of life during childhood remains concentrated in sub-Saharan Africa (sSA), where children are between six and fifteen times more likely to die before reaching age five compared to children in more developed regions.^{3,4} To address this inequality health sectors in sSA have departed disease-specific programming strategies and adopted holistic frameworks for bundling low cost, evidence-based interventions (EBI) along the maternal, newborn and child health (MNCH) ‘continuum of care’, and integrated these bundles into primary health care (PHC) delivery systems.⁵⁻⁸ This introduced challenges to the task of assessing the impact of child survival programs. Whereas evaluations had traditionally focused on the effectiveness of specific interventions, the advent of new programming approaches implied the need to measure the ‘implementation strength’ with which PHC systems deliver EBI packages and evaluate whether improvement in implementation strength is associated with child survival.⁹⁻¹²

The term ‘implementation strength’ has been used interchangeably with other terms, such as ‘implementation intensity’ and ‘effective coverage’ and is defined here as a quantitative measure of the ‘dose’, or the amount of input or activity, delivered to implement a program. Implementation strength has a rich history in global health.^{13,14} Efforts to scale up emergency obstetric care led to a ‘signal function framework’, which acknowledged that a set of performance indicators was needed to evaluate maternal survival programs since the occurrence of obstetric emergencies was rare within program settings.¹⁵ For similar reasons, researchers use signal functions to gauge the effects of expanding access to abortion care services.^{16,17} The advent of ‘health systems strengthening’ focused attention on the need to measure processes and outcomes of improving the

capacity of health systems to deliver PHC.^{18,19} The WHO's 'health systems building blocks' framework was central to this and provided a structure for monitoring and evaluating health systems performance.²⁰⁻²² This has included large-scale health facility and household surveys in low- and middle-income countries, notably Service Provision Assessments, Demographic Health Surveys (DHS), Service Availability and Readiness Assessments (SARA), and Multiple Indicator Cluster Surveys.²³⁻²⁵ In addition, researchers have adapted common evaluation frameworks and applied them across health systems to measure implementation strength.^{26,27} Approaches, such as the Balanced Score Card, provide a dashboard of performance indicators that are used to detect variation and change in PHC implementation strength.²⁸⁻³² Similar approaches have been adapted to measure the same with respect to domains of care, such as emergency obstetric and newborn care^{33,34}, integrated management of childhood illness¹² (IMCI), and family planning.³⁵

1.1 Implementation strength: Measurement and association with child health gains

Efforts to apply the concept of implementation strength and evaluate its child health impacts have faced methodological challenges. The first relates to derivation of the exposure variable. A common approach has been to presuppose the intervention components that are most crucial to the causal pathway and their relative importance based on intimate knowledge of the program, literature review and credence to global measurement approaches.^{36,37} Researchers then design methods and tools for capturing data on those components and perform simple or weighted additive analysis to generate summary indices that represent implementation strength. Studies that have used this approach to measure the individual-level child health response to implementation strength have reported that there is a positive association between 'dose strength' of EBI implementation and desirous MNCH behaviors.^{38,39} Although their exposure measurement strategy is easy to conduct, theoretically justifiable and can produce results amenable to

comparison between geographies, it assumes that implementation strength is unidimensional, and can create non-normal, skewed distributions. Thus, the additive summary score is not always conceptually meaningful and may not accurately portray overall implementation strength.^{40,41} Furthermore, this approach does not consider patterns between the wider, underlying set of variables of program inputs and activities between units of analysis (e.g., facility, district). This is problematic since these relationships contribute to the real variation between analytic units in terms of the underlying construct that represents the exposure of interest, i.e., the implementation strength that the health system exerted during the time and place of the evaluation at hand.⁴² Neglecting this issue risks misallocating weight to given input and activity variables and omitting potentially crucial ones from analysis.⁴³ This, in turn, may undermine the quality of associational analyses which assume that differences in measures of implementation explain variation in health outcomes.

Second, several analyses of the relationship between implementation strength and child health have aggregated data on outcomes to the level of district, country or other clusters that represent the level at which implementation actions are taken. Such studies have generated conflicting results with respect to the association between dose strength of program implementation and child health related care-seeking and child mortality.^{39,44} While this may be due to differences in the strategy and content of programs, it's important to recognize that these studies are beset with ecological biases.⁴⁵ For example, one study in Uganda, which compared approaches for linking individual-level service utilization indicators from household surveys with data on the provision of maternal and newborn services at the facility- and ecological-level (district) to estimate levels of effective coverage, found large discrepancies between estimates between the two approaches, concluding that the ecological approach appreciably overestimates effective coverage.⁴⁶ Third,

individual and ecological studies of implementation strength and child health have almost exclusively depended on cross-sectional data.^{38,39,44} Although this often reflects the best use of data available to address the research question, it precludes ascertainment of temporal order between exposure to program intensity and the occurrence of the outcome. This temporal bias raises questions about ‘reverse causality’ and prevents a ‘dose response’ interpretation of the relationship between implementation strength and child health gains.

1.2 Purpose and objectives

The purpose of this paper is to demonstrate an analytic approach that overcomes these challenges. We first develop summary indices of implementation strength using data from a SARA that was conducted in three rural districts of Tanzania between May and August of 2011. SARA are comprised of measures of general and domain specific attributes of PHC facilities’ structural quality and are frequently implemented in low- and middle-income countries by special projects or to assess health sector performance at national scale.⁴⁷ Our analysis blends weighted-additive methods and multivariate statistical procedures and applies them on a voluminous range of indicators on MNCH provision. In doing so, we derive a minimal set of independent scales that collectively represent variation in the intensity with which local health systems in the three districts provided MNCH EBI. The analysis proceeds by linking scales of implementation strength with longitudinal data that contain the survival trajectories of approximately 9,000 children, starting with their birth from March to November 2011 in the catchment communities served by SARA facilities, until the end of the cohort in December 2015. With this, we assess whether differences in the dose of implementation strength exerted by PHC facilities on communities in which children experienced infancy are associated with these children’s risk of dying during childhood.

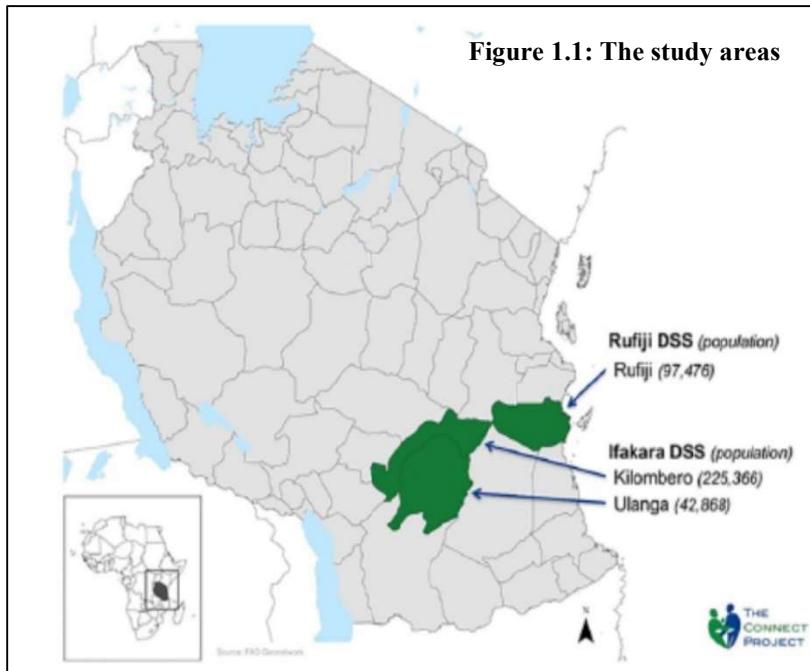
In conducting this analysis, we address two objectives. The first is to validate the methodology we use to measure implementation strength using routine facility data obtained through a popular data collection methodology. We hypothesize that detection of an association between implementation strength and child mortality, given that the SARA measured the provision of child survival EBI that have been proven repeatedly and in various contexts, indicates that our measurement approach is worthy of consideration for uptake by other studies that use SARA or similar data to evaluate health system performance. Our second objective is to identify the components of the Tanzanian integrated PHC program that drove child mortality reduction and give insight on inputs and activities to emphasize to maximize program impact on child survival.

2. Methods

2.1 The Tanzanian health system

Since its founding, Tanzania has demonstrated commitment to achieving universal access to PHC. In the 1970s, the country launched a national expansion of PHC facilities, called dispensaries and rolled out a national village health worker program.⁴⁸ During this period, the Ministry of Health promulgated a policy that guaranteed mothers and children free access to basic services such as immunization, nutrition, antenatal and postnatal care, growth monitoring, and treatment for minor illness.⁴⁹ In 2007, the Ministry of Health initiated the Primary Health Care Services Improvement Program, which accelerated the expansion of dispensaries and higher-level PHC facilities, called health centers, across the country.⁵⁰ Since then, strategies to guide implementation of this policy have emphasized making the above essential service package, as well as skilled obstetric care, IMCI, and family planning available at the health center and dispensary level.⁵¹⁻⁵⁵ The implementation of these strategies co-occurred with reduction in child mortality in Tanzania, which between 2000 and 2015 declined from 130 to 58 per 1,000 live births.⁵⁶

2.2 Study environment and data sources



Data for this study were obtained from the *Connect Project*. *Connect* was a cluster-randomized controlled trial of the impact of community health workers on child survival that was conducted by the Ifakara Health Institute in Tanzania from 2011- 2015.^{57,58}

Connect was situated in the

sentinel areas of the Ifakara and Rufiji Health and Demographic Surveillance Systems (HDSS) in three rural districts.^{59,60} The Ifakara HDSS is in Morogoro, a landlocked region in southwestern Tanzania, and is approximately 500 km from Dar es Salaam. The Rufiji HDSS is in Rufiji district on the Indian Ocean coast approximately 150 km south of Dar es Salaam. The communities within these sites are largely agrarian and depend on subsistence agriculture, fishing, and petty trading.^{61,62} See Figure 1.1.

Between April and September 2011 *Connect* conducted a household survey in 101 communities to obtain baseline measures of key MNCH behavior and service utilization indicators. At this time, *Connect* also carried out a SARA in the 109 health facilities in the three districts to understand the context of MNCH service provision. The SARA obtained information from facilities' staff on the communities in their catchment areas. Data collectors used geographic positioning system tracking

devices and mapped facility-to-community travel distances in a geographic information system database.^{61,62}

Connect leveraged the longitudinal HDSS platforms for its impact evaluation. Until December 2015, staff of both HDSS visited households in the sentinel areas every four months to collect information on household members, their relationships, ages, and sexes; births, deaths, and in- and out-migration of household members. Every 1-2 years, censuses were undertaken to enumerate old and new households. The censuses obtained data on maternal education attainment and household income and assets.^{63,64} For our analysis, we linked household survey, SARA and HDSS data by community.

2.3 *Creating 'effective coverage scales' for specific domains of MNCH*

We followed a multi-step process to develop scales of MNCH implementation strength that was exerted by PHC facilities in the *Connect* study areas on the communities that they served. For this, we used data from four modules of the SARA (excluding the fifth module which obtained data on implementation costs) and the household survey. We restricted our sample to the PHC facilities (not hospitals) to which residents from the 101 study communities would go for care.

The four SARA modules compiled categorical indicators on multiple domains of care: (1) general facility readiness (staffing levels, management practices and infrastructure), (2) family planning, (3) antenatal care, (4) intrapartum care, (5) postnatal care, (6) preventive services for children (e.g., immunizations, insecticide treated nets, counseling, assessment, classification components of IMCI) and (7) sick childcare (trained staff and supplies to care for respiratory illness, diarrheal disease, malaria, malnutrition). For indicators with more than one response category, we created dummy variables so that all indicators used in the analysis were binary. For all indicators in each domain, we calculated Cronbach's alpha coefficients to establish internal consistency and found

that all sets of indicators reported coefficients of 0.8 or higher.⁶⁵ For each domain, we calculated ‘effective coverage indices’ using a weighted average approach. For each facility, we created the general facility readiness effective coverage index by summing the values for each of the indicators on staffing levels, management practices and infrastructure availability, respectively, and dividing each total by the number of indicators related to each of these sub-categories. We summed these averages and divided this sum by three, the number of categories in this domain.

We followed a similar procedure for the six service specific domains. Within each domain for each facility, we summed the values indicators that fell within three common sub-categories: (i) staff training on domain-specific skills, (ii) the range and frequency with which domain specific services were available at the facility, and (iii) current availability and recent stock outs of domain-specific supplies, medicines, and equipment. We divided each of these sums by the total number of indicators related to each sub-category, then summed those averages and divided this sum by three. To incorporate coverage into this score, we identified the communities that were in each facility’s catchment area , and used data from the household survey to calculate community-level averages of met need for family planning, and, with respect to respondents’ most recent birth, receipt of ≥ 4 antenatal care visits, facility-based delivery, postnatal care, immunizations and, for respondents with children that had recently had diarrheal, respiratory or febrile illness, receipt of needed medications. We aggregated these averages to estimate the ‘catchment specific coverage’ for each domain. We then multiplied the six measures of ‘catchment specific coverage’ by their corresponding domain-specific scores for each facility. With this, for each facility, we obtained seven domain specific effective coverage indices.

2.4 Combining domain specific effective coverage indices to reduce data into independent, parsimonious scales of implementation strength

To obtain a smaller set of scales of implementation strength, we used principal components analysis (PCA). We chose PCA because of its ability to reduce the highly correlated effective coverage indices into fewer orthogonal principal components (PC), or scales, that maximize the variation in the data and represent facilities' relative position in terms of implementation strength.⁶⁶ We determined the number of PC to retain in our analysis via parallel analysis. Per convention, we retained PC with an eigenvalue of greater than or equal to 1.⁴² To interpret each PC, we examined the factor loadings and cosine² values that were reported for each of the indicators used to formulate them. The higher loadings and cosine² values indicated greater and higher-quality representation of each domain specific effective coverage index to each PC.^{67,68}

Next, we created scales that represented the levels of implementation strength to which each study community was exposed. For this, we grouped facilities together if SARA respondents reported that members of the same communities went to them for care or if the facility was within five kilometers of the same communities. We combined the PC values of facilities in the same group using three-level Bayesian mixed effects models with communities nested within catchment areas, and catchment areas nested within districts, and fixed effects to denote communities distance from the nearest facility and population size.⁶⁹ Functionally, these models used the PC values of each facility for each retained scale as the prior distribution to produce a posterior distribution of values that represented the overall levels of implementation strength that facility groups exerted collectively.⁷⁰ The benefit of this approach is that it borrows information from facilities within each group to estimate mean implementation strength scores that are shrunk to a central value, which results in more stable estimates with smaller standard errors.⁷¹

2.5 *Estimating the association between three dimensions of implementation strength on newborn and child mortality*

We merged the combined scale values that had been assigned to communities to a longitudinal dataset from the HDSS that included the survival trajectories and individual- and household level covariates for children that were born between March and November 2011. We then estimated the relationship between the implementation strength scores exerted by facility groups and the risk of child mortality, modelling the implementation strength scales in their continuous form. To address potential confounding, we incorporated fixed effects for covariates at the child level (child sex, birth order, previous and subsequent birth interval durations), mother-level (age, marital status, years of schooling), household socio-economic status (SES) ranking (1-5), and contextual variables (distance from community to nearest health facility, and HDSS zone in which communities were nested).¹ To select our modelling strategy, we conducted the Schoenfeld test of residuals to determine whether the assumption of proportional hazards was met.⁷² Because multiple covariates marginally failed to satisfy this requirement, we elected to use Weibull parametric hazard regression models, which capture the underlying hazard of child mortality that is known to be high during the newborn period and decline as children age. To account for clustering of births within communities, we incorporated in our models a random effect for community.

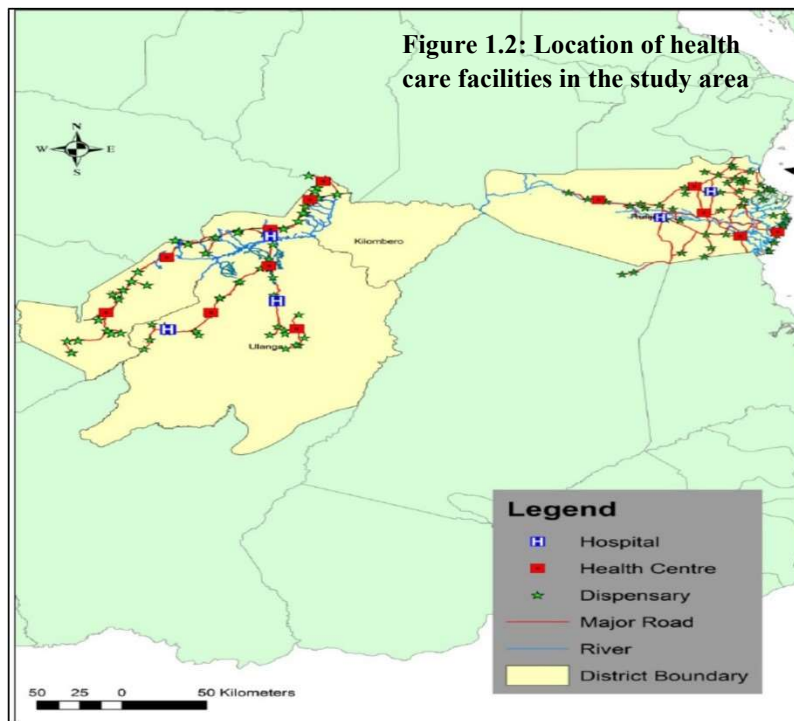
In our longitudinal dataset, there was missing data for two covariates, household SES and mothers' years of schooling for roughly one-fourth of the sample. After examination, we determined that the omission of values reflected a 'missingness at random' pattern. To address this challenge, we compared three approaches: First, we omitted the two covariates from our models and conducted a complete case analysis; Second, we imputed missing values with the community-level median

¹ The grouping of communities into 5 zones was done by the Ifakara and Rufiji HDSS for management purposes to aid in management HDSS field operations.

value for the two covariates; Third, we used multiple imputation with chain equations (MICE).⁷³ The results that these approaches produced were virtually identical (Supplemental File 1.1). Therefore, we report the results of the third approach. We conducted the entire analysis in R Studio 4.0.2.

3. Results

Based on information from SARA respondents and travel distances, we found that the 101 study communities sought PHC from 56 primary health care facilities. See Figure 1.2 and Table 1.1.



In total, 234 indicators from the SARA were used to create domain specific effective coverage indices (47 indicators on general facility readiness, 23 family planning services, 22 antenatal care, 54 delivery services, 23 postnatal care, 31 on preventive childhood services, 34 on sick child care). Table 1

presents descriptive data on the domain specific effective coverage indices calculated for the facilities in our sample. Of the 56 facilities, 6 were health centers, and 50 were dispensaries. 35 of the facilities were in the sentinel areas of the Ifakara HDSS (12 in Ifakara Rural, 5 Ifakara Urban and 18 Ifakara Expansion) and 21 in the Rufiji HDSS (17 in Rufiji Rural, 4 Rufiji Urban). Supplemental File 1.2 contains a table that presents the median and range of scores of the effective coverage indices by HDSS zone.

Table 1.1: Background characteristics of primary health care facilities (n = 56)

	n (%)
Number of primary health care facilities retained	56 (100)
Facility type	
Health Center	6 (11)
Dispensary	50 (89)
Managing authority of health facility	
Government/public	36 (65)
Faith-based	14(25)
Military	3 (5)
Private	3 (5)
Geographic zone	
Ifakara Rural	12(21)
Ifakara Urban	5(9)
Ifakara Expansion ²	18 (32)
Rufiji Rural	17 (30)
Rufiji Urban	4 (7)

Supplemental File 1.3 shows the Scree plot that was produced by the PCA and guided our parallel analysis. The first three PC reported eigenvalues of approximately 1 or greater³ and together accounted for 77% of the variance among the seven domain specific effective coverage indices, whereas the remaining four scales hold appreciably less explanatory potential. Thus, we chose to retain the first three scales only.

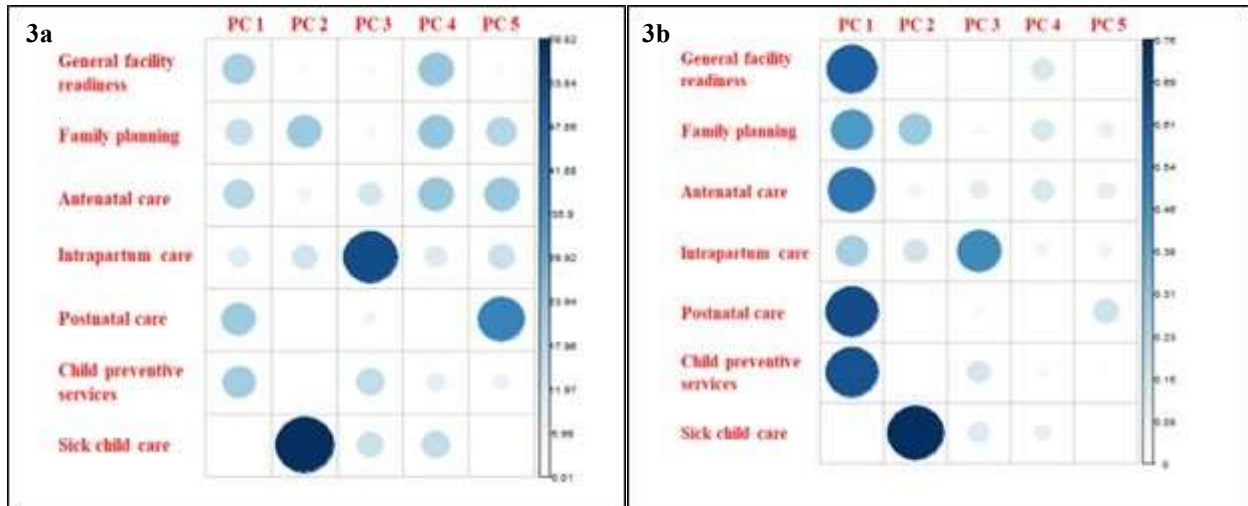
Figures 1.3a and 1.3b reflect the factor loadings and cosine² values for each PC, which reflect the relative contribution of each underlying effective coverage index to the retained scales. We found that the first PC, which accounted for 46% of variation in the effective coverage indices, was an implementation strength scale that reflected facilities' relative position in terms of the availability and readiness to provide preventive health services, mostly child-related, with high loadings and cosine² values for general facility readiness, antenatal care, postnatal care, and preventive

² Expansion refers to the geographic area comprising of communities in which the Ifakara HDSS was expanded in 2010.

³ We retained the third PC even though its eigenvalue was 0.94 because it dominantly represented the availability of intrapartum care, which is recognized as relevant to survival of children, particularly newborns. Intrapartum care was not represented in the other PC whose eigenvalues were greater.

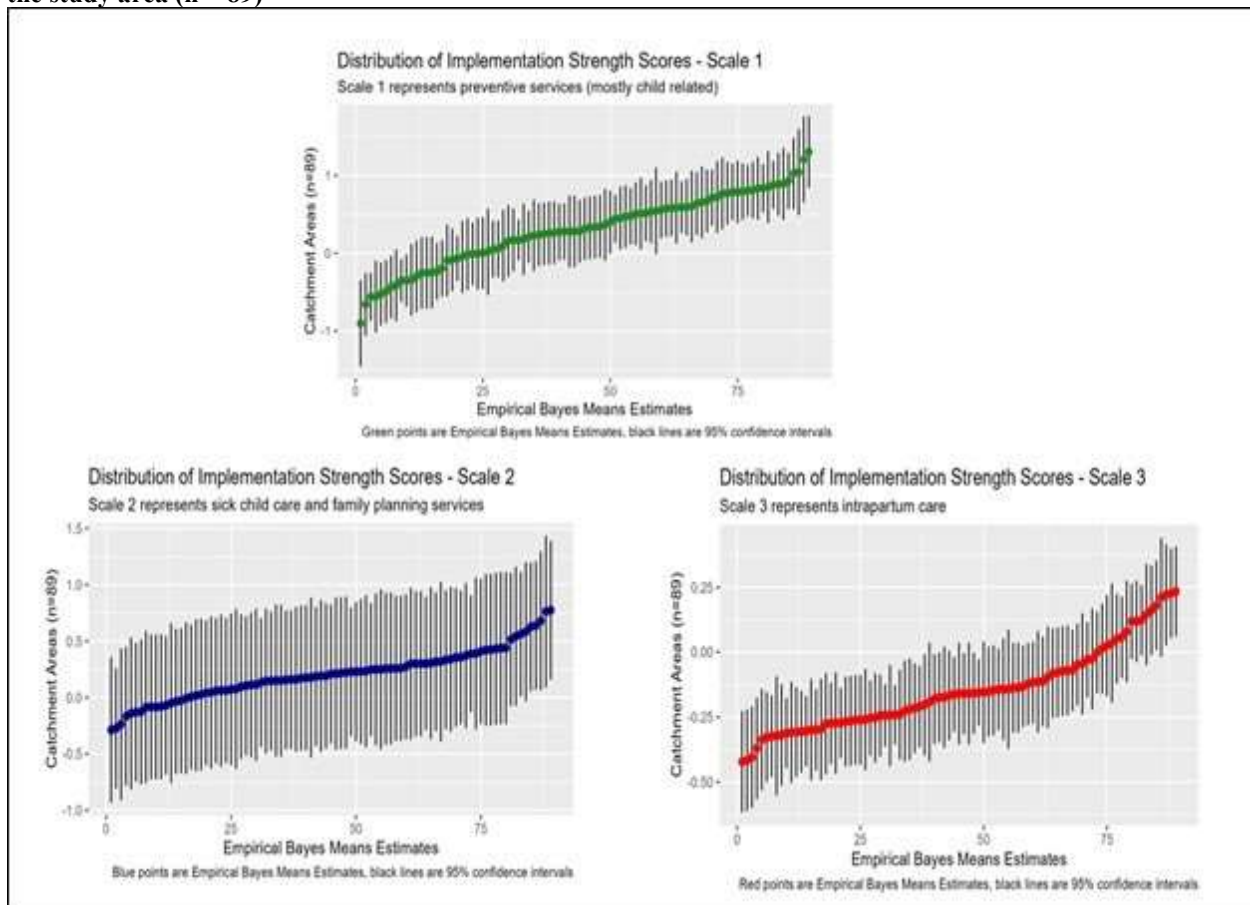
childhood services. The second PC, which accounted for 18% of the variation, represented a scale of facilities' readiness to provide sick childcare and, to a lesser extent, family planning services; and the third PC, which accounted for 13% of the variation, represented a scale of facilities' readiness to provide intrapartum care.

Figure 1.3a and 1.3b: Correlation plot of factor loadings (3a) and Cosine² values (3b) produced by PCA of MNCH effective coverage indices.



We used information provided by SARA respondents and geographic data to determine which of the 56 facilities in our sample served the 101 communities in the study area respectively. In doing so, we identified 89 different combinations of facilities. On average, villages were within the catchment areas of three primary care facilities (range: 1, 7). For each community, we computed the collective implementation strength that was exerted on them by the group of facilities in whose catchments they were located using a Bayesian mixed effects model and estimating the empirical Bayes mean implementation strength scores for the three scales that we retained. Figures 1.4a-c illustrates the distribution of implementation strength scores assigned to the 89 different facility groups that defined the catchment areas in which the 101 study communities were nested.

Figures 1.4a-c: Distribution of implementation scores across primary health care facility catchment areas in the study area (n = 89)



Supplemental Files 1.4a-c illustrates the geographic variation in implementation strength, comparing the distribution of implementation strength scores between groups of communities that were in five administrative zones within the study area, 3 in the Ifakara HDSS (Ifakara rural, urban and expansion areas) and 2 in Rufiji (rural and urban). We observe the greatest variation in median implementation strength scores among zones for the first scale, which represents the availability and readiness to provide preventive care services for newborns and children as well as antenatal care, and the least variation between zones for the second scale, which represents access to sick childcare and family planning services.

Table 1.2 describes the characteristics of 8,999 children that were born from March-November 2011, the period spanning approximately 3-months before and after the start and end of the SARA,

in the villages whose residents report to the above-described 56 facilities for care. Among these children, 526 (5.8%) died before December 31, 2015, when the follow up of the cohort ended.

Table 1.2: Characteristics of children born in catchment areas of the 56 primary health care facilities in the study area between March and November 2011 (n = 8,999)

Characteristic	n (%)	Under-five year-old deaths (n, %)
Sex		
Male	4541 (50.1)	293 (55.7)
Female	4458 (50.0)	233 (44.3)
Birth order		
1	6193 (68.8)	365 (69.4)
2	1420 (15.7)	91 (17.3)
3	757 (8.4)	39 (7.4)
≥4	629 (7.0)	31 (5.9)
Previous birth interval duration		
First born child	6284 (69.8)	387 (73.4)
≤18 months	150 (1.7)	12 (2.3)
19-24 month	292 (3.2)	17 (3.3)
25-36 months	721 (8.0)	35 (6.7)
≥36 months	1551 (17.2)	75 (14.3)
Subsequent birth interval duration		
Last born child	7048 (78.3)	315 (59.9)
≤18 months	164 (1.8)	67 (12.7)
19-24 month	255 (2.8)	36 (6.8)
25-36 months	778 (8.8)	61 (11.6)
≥36 months	754 (8.4)	47 (8.9)
Mother's age at birth		
<20	1430 (15.9)	84 (16.0)
20-24	2173 (24.1)	121 (23.0)
25-29	2193 (24.4)	110 (20.9)
30-34	1679 (18.7)	113 (21.5)
35-39	1044 (11.6)	71 (13.5)
≥40	480 (5.3)	27 (5.1)
Mother marital status at birth		
Married/in union	5900 (65.6)	313 (59.5)
Single	3099 (34.4)	213 (40.5)
Mother number years of schooling		
None	1824 (20.3)	134 (25.5)
1-6 years (primary)	1064 (11.8)	66 (12.5)
≥ 6 (some secondary or more)	3283 (36.5)	197 (37.5)
Unknown ⁴	2798 (31.3)	129 (24.5)

⁴ 2798 values were missing.

Household SES at birth (quintile ranking)		
First (lowest)	1313 (14.6)	80 (15.2)
Second	1202 (13.4)	59 (11.2)
Third	1368 (15.2)	80 (15.2)
Fourth	1284 (14.3)	62 (11.8)
Fifth	1199 (13.3)	74 (14.1)
Unknown ⁵	2633 (29.2)	171 (32.3)
Distance to nearest health facility		
<5 kilometers	4839 (53.8)	271 (51.5)
5-10 kilometers	2719 (30.2)	164 (32.2)
>10 kilometers	1441 (16.0)	91 (17.3)
HDSS zone		
Ifakara Rural	3490 (38.8)	185 (35.2)
Ifakara Urban	939 (10.4)	71 (13.5)
Ifakara Expansion	2346 (26.1)	160 (30.4)
Rufiji Rural	1185 (13.2)	62 (11.8)
Rufiji Urban	1039 (11.5)	48 (9.1)

Table 1.3 presents the results of our analysis of the relationship between the three scales which represent the implementation strength of MNCH to which children in the study area were exposed during early infancy in 2011 and the risk of their dying before December 31, 2015. The first model is an unadjusted analysis of the effect of the three dimensions of implementation strength on child mortality, the second the same analysis however adjusting for individual mother- and child-level covariates, and the third the same analysis however adjusting for individual mother- and child-level and contextual covariates. As discussed above, to address missingness of data on SES and mothers' years of education, we fit Models 2 and 3 using MICE. From our analysis, we observe an association between the strength of antenatal, postnatal, and preventive early childhood health care services accessible to the population and the risk of dying during childhood. After adjustment for potentially confounding individual- and contextual-level factors, the third model reports that for each unit increase in the implementation strength of these interventions, the risk of dying during childhood is 0.61 times lower (HR: 0.61; 95% CI: 0.39, 0.96).

⁵ 2633 values were missing.

Table 1.3: Associations between child mortality risk and implementation strength scores reported from Model 1 (adjusted, scores 1-3 only), Model 2 (scores and mother- and child-level covariates), and Model 3 (scores and mother- and child-level covariates and contextual-level covariates) (n = 8,999)

	Model 1		Model 2		Model 3	
	HR	95% CI	HR	95% CI	HR	95% CI
Implementation strength scores						
Score 1 (ANC, PNC, and preventive child care)	0.76	0.60, 1.00	0.70*	0.54, 0.90	0.61*	0.39, 0.96
Score 2 (sick child care)	0.74	0.43, 1.25	0.60*	0.35, 0.99	0.54*	0.30, 0.99
Score 3 (intrapartum care)	0.86	0.43, 1.73	0.62	0.31, 1.23	0.48	0.19, 1.24
Child sex						
Female	-	-	-	-	-	-
Male	-	-	1.25*	1.05, 1.48	1.25*	1.05, 1.49
Birth order						
No. children (cont.)	-	-	0.99	0.88, 1.13	1.00	0.88, 1.13
Previous birth interval						
Months (cont.)	-	-	1.00	0.99, 1.00	1.00	0.99, 1.01
Subsequent birth interval						
Months (cont.)	-	-	1.01***	1.00, 1.02	1.01***	1.00, 1.02
Mother age at birth						
Years (cont.)	-	-	1.01	0.99, 1.02	1.01	1.00, 1.02
Mother marital status at birth						
Married/in union	-	-	-	-	-	-
Single	-	-	1.44**	1.20, 1.72	1.43**	1.19, 1.72
Mother number years of schooling						
Year of schooling (cont.)	-	-	0.97 [†]	0.94, 1.00	0.97	0.95, 1.01
Household SES at birth (quintile ranking)						
Fifth	-	-	-	-	-	-
Fourth	-	-	0.78 [†]	0.60, 1.02	0.79 [†]	0.60, 1.03
Third	-	-	0.96	0.74, 1.26	0.99	0.75, 1.29
Second	-	-	0.92	0.70, 1.22	0.95	0.71, 1.26
First	-	-	0.91	0.68, 1.21	0.93	0.70, 1.25
Distance to nearest health facility						
Kilometers (cont.)	-	-	-	-	1.14 [†]	0.98, 1.32
HDSS zone						
Ifakara Expansion	-	-	-	-	-	-
Ifakara Rural	-	-	-	-	0.85	0.58, 1.22
Ifakara Urban	-	-	-	-	1.25	0.81, 1.95
Rufiji Rural	-	-	-	-	1.12	0.70, 1.81
Rufiji Urban	-	-	-	-	0.95	0.54, 1.68

[†] = p-values < 0.1, * = p-values < 0.05, 0.** = p-values < 0.01, *** = p-values < 0.001

The third model suggests a more protective effect of implementation strength on child mortality with regards to the second scale, which represents the availability and readiness of nearby facilities

to provide sick child care. After adjustment for multi-level covariates, we noted that among children in our cohort, increases exposure to higher levels of sick child care implementation intensity were associated with, on average, with 0.54 times lower risk of dying during the first 4-5 years of life (HR: 0.54, 95% CI: 0.30, 0.99). There was no statistically significant association detected between variation in the third scale of implementation strength, which represents the strength of intrapartum care services available in study facilities, and child mortality risk. In addition to evaluating the child mortality response, we adapted Models 1-3 so that they could indicate whether variation in implementation strength, as represented by the three scales, were associated with children's risk of dying in the first month and first year of life, respectively. These analyses reported hazards ratios of magnitudes that were similar to those reported by Model 3, but these results were not statistically significant (Supplemental File 1.5).

4. Discussion

Our analysis reports that children exposed to higher levels of implementation strength of preventive and curative childcare, including antenatal care, postnatal care, and family planning services available to mothers, from the PHC facilities that served their community at the time of birth and early infancy were less likely to die during the subsequent 4-5 years of life than those exposed to lower levels of implementation strength. Whereas the same analyses of the relative risk of newborn and infant mortality associated with implementation strength did not reveal a significant relationship, we believe that this owes the limitations in the power accorded by the sample size and number of mortality events in the first month and year of life in our cohort. Furthermore, we observe no association between child mortality and the third implementation strength scale, which represents the strength of intrapartum care services. This finding contradicts similar studies which measured the health response to obstetric care quality. For example, Tiruneh

et al. in Ethiopia used an additive approach to develop a gradient of basic emergency obstetric and newborn care implementation strength (BIS) and conducted an analysis which reported a positive association between higher levels of BIS and facility-based delivery and met need for emergency obstetric and newborn care.³⁴ Although, our finding may be surprising, it is important to recall that the third scale explained a relatively small proportion of the overall variance among the effective coverage indices that we reduced into our independent gradients of implementation strength. If, in our analysis of SARA data, we had included hospitals and higher-level health centers, which are relatively better equipped to handle labor and delivery, it is likely that the sample would have contained more variation with respect to this domain of care. In turn, this might have led to a set of scales that demonstrated a more potent effect of intrapartum care implementation strength.

Our findings contribute to the debate about the role of primary health care programs and services in the precipitous child mortality decline that Tanzania experienced from 2000-2015. Importantly, they corroborate the findings issued by Masanja et al. (2008) in their analysis of the trends and drivers of child mortality using four consecutive DHS surveys between 1990 and 2008. This study identified the synergistic effect of increases in public sector health expenditure, implementation of decentralization policies and expansion in the coverage of high impact child health interventions, such as insecticide treated nets (ITN), extended program immunizations (EPI), and IMCI, as contextual factors important to the reduction child mortality observed in Tanzania during this period.⁷⁴ Afnan-Holmes and coauthors, make similar observations when reviewing Tanzania's achievement of Millennium Development Goal 4, also attributing success to increases coverage of EPI, IMCI and ITN.⁷⁵ Our analysis underscores the importance of investments in expanding these interventions. Furthermore, our interpretation of the first set of factor loadings produced by the PCA suggests that ensuring effective coverage of routine antenatal and postnatal care as well as

general facility readiness, in terms of regular staffing, general infrastructure and routine management inputs and processes, may be as important to enhancing child survival prospects in the population as preventive and promotive health interventions that target children under five. This information is relevant to health system managers and planners in Tanzania as they prioritize ways to invest limited resources in ensuring effective coverage of PHC services and scaling up.

This analysis illuminated an appreciable child mortality response to slight variation in the strength of PHC performance within small geographic areas. In our examination we distinguished the relative contributions of different domains of MNCH to this variability and provide insight on how improvements vis-à-vis these domains can impact children's prospects of surviving childhood. Yet, we did not explore the factors that explain why, within relatively small areas, some facilities perform better than others. Previous studies in Tanzania have sought to understand how differences between nearby facilities condition patient care seeking. For example, one study reported that care seeking behaviors were shaped by differences between facilities in terms of the quality of consultations and availability prescriptions, knowledge-level of staff, and availability of physicians and essential supplies and equipment.⁷⁶ Another study sought to understand the drivers of variation between nearby facilities in rural areas in terms of provider competence and practice quality, and reported that this was a function of facility ownership (private for profit, non-governmental organization, public sector), population density of facilities' catchment area, and health workers training, tenure and experience.⁷⁷ Other studies in Tanzania on this topic have focused on specific domains of MNCH. Kahabuka et al. (2011) attributed patient care seeking for preventive and sick child care services to differences between nearby facilities in terms of the availability of diagnostic equipment, essential medicines and skilled staff.⁷⁸ Kanté et al. (2016) reported that differences between facilities' performance of emergency obstetric care signal

functions conditioned women's care seeking for intrapartum care.⁷⁹ Future research, in Tanzania and similar settings, should continue to examine the determinants and processes that generate within-small area variation in service readiness, availability and quality since this information, as our findings suggest, could help address problems that underlie preventable child deaths.

Effective coverage measures that link population level data on access to health care and health outcomes with facility data on health care quality are increasingly reported.⁸⁰⁻⁸² However, there is little guidance on appropriate methods for linking, e.g. which data to link, for which units and with what temporal alignment.⁴⁶ Furthermore, decisions to link data sets are usually not foreseen during study design, and, therefore, researchers must be opportunistic in the linkage methods they employ. In our analysis, we linked individual-level data on child survival to summary scores of MNCH coverage effectiveness of the facilities in the environs of children's household and communities and found that the contextual effect of implementation strength in these areas was associated in lower risks of child mortality. However, this might differ from an analysis of the same relationship that linked individual outcomes with performance scores of exact facilities where children obtained care. Future research is needed on effective coverage estimation and its health effects that compares methods that operationalize coverage in geographic terms versus in terms of where individuals utilized services. Identification of biases associated with either linkage approach will help health systems researchers determine the data and data systems requirements for measuring health systems strengthening and its impacts.

To our knowledge this is the only study that has benefitted from the ability to link data on 'dose delivered' of implementation strength within local health systems with longitudinal data on the survival of children nested in the underlying population. Although other studies, for example in Ethiopia and Malawi, addressed similar questions related to implementation strength of MNCH

services, the larger geographic scope of their analysis and lack of prospective data on the outcome compelled authors to draw upon repeat cross-sectional data from the DHS or special project surveys, and perform ecological analyses, which are subject to biases.^{38,44} Whereas these findings are valuable insofar as they evaluate the impact of the large-scale rollout of national child survival programs, our analysis fills an important gap in that it reports the effects of variation in dose delivered of implementation strength by routine delivery systems within a relatively small area on individual level mortality risk that unfolds over time after exposure. Future research should seek opportunities to leverage existing longitudinal data collection platforms and integrate them into investigations of the population-level health response to changes in health systems strength.

Our analysis is not without limitations. Though we opted to use PCA as a data reduction approach, we recognize that this approach has drawbacks. For example, there are examples that have demonstrated that the use of PCA results in the misclassification of subjects vis-à-vis the gradient of the underlying construct when the PCs used explained less than 30% of total variance.⁸³ Although our finding that higher levels of readiness and availability of sick child care was associated with lower child mortality risk seems intuitive, it is not immune to this type of critique. Second, despite our review of the risks of weighted-additive methods, we used that approach to derive effective coverage indices of different domains of MNCH that we later subjected to PCA. This came after reviewing alternative data reduction strategies, including use of PCA only to reduce the 234 indicators in the SARA to implementation strength scales. In the end, we felt that the weighted-additive approach, enabled us to represent the natural partition in our data between domain-specific measures of MNCH availability and readiness and best identify which components of MNCH care are most relevant to child mortality reduction. Third, there were significant levels of missingness for two covariates in the HDSS, which supplied our data on child

survival. However, after comparing three approaches for addressing this problem (complete case analysis, median imputation, and MICE) we found that comparable results were obtained under all strategies, which indicated that missing data did not seriously affect the overall association between implementation strength and child survival. Fourth, the period for which we subset the longitudinal data from the HDSS (March to November 2011), given the cross-sectional nature of the SARA data (May to September 2011) calls to question whether children in the cohort were truly exposed to the levels of implementation strength that we ascertained from the analysis of SARA data. Fifth, our analysis links children born during the period surrounding the SARA with data on the facilities in whose catchment their communities were located; however, our analysis could not ascertain whether these children ever sought health care at other health facilities during their first 4-5 years of life. Finally, although the analysis was able to link within district variation in levels of implementation strength exerted by primary health care facilities with longitudinal data on child survival, these data are observational and, thus, fall short of permitting inference that is truly causal.

5. Conclusion

We developed gradients that quantify the strength with which MNCH services were delivered to the populations within in the local health systems in three districts of Tanzania in 2011, and, by linking these scales with longitudinal survival trajectories of children born in the underlying population, evaluated whether variation in implementation strength at the time of childbirth and early infancy were associated with mortality risk during childhood. The results suggest that the intensity with which preventive care services, including general facility readiness, antenatal and postnatal care, and preventive care for children, were made available to the population, as well as that of curative care and family planning services, was associated with lower levels of mortality

risk. Since these scales reflect the performance of interventions that are part of the Tanzania's essential MNCH services package through routine delivery system channels, local health authorities can use these metrics, which were derived by using data readily available from facilities, to better understand the health impact of their implementation on the populations they serve. Additional research of this nature should be undertaken using data from hospitals to obtain data with greater variation on the performance of obstetric and gynecological services to better understand how health systems meet the needs of women during the crucial intra- and postpartum periods. Health system managers and decision-makers can use this information to inform planning, resource allocation and implementation adjustments and maximize the impact of health system strengthening on population health.

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Chapter 3:

The implementation and effectiveness of multi-tasked, paid community health workers on maternal and child health: A cluster-randomized pragmatic trial and embedded qualitative process evaluation in Tanzania

Abstract

Community health worker (CHW) programs have proliferated throughout health systems of low- and middle-income countries based on evidence that access to essential, low-cost interventions from these cadres can help end preventable mortality, particularly among children. However, there is limited evidence on CHW program implementation processes, determinants, and effectiveness from randomized studies delivered through routine health systems. In this paper, we report on the results of a cluster-randomized pragmatic implementation trial and embedded qualitative process evaluation of a CHW program in Tanzania that was implemented from 2011-2015. Program effects on maternal, child and neonatal health (MNCH) service utilization, childhood morbidity and sick childcare seeking were evaluated using difference-in-difference regression analysis with outcomes measured through pre- and post-intervention household surveys in intervention and comparison trial arms. Qualitative health systems research was conducted in intervention settings from 2012-2014 as case studies of the implementation experience. 75 in-depth interviews and focus group discussions were completed during three rounds of data collection among respondents, including CHW, community members, facility-based health workers and members of local health management teams. The CHW program reduced incidence of childhood diarrheal, respiratory, and febrile illness and improved access to timely and appropriate sick childcare; however, there was no effect on MNCH service utilization behaviors. The positive outcomes were achieved through implementation mechanisms that triggered high levels of acceptability of CHW among community-members, and motivation and confidence of the CHW. Implementation factors that

generated these effects related to the engagement of communities in program startup; the training, remuneration and configuration of supervisory support to the CHW from the local health system and community. The lack of program effects on MNCH service utilization were attributed to the fragile health systems context, which was replete with lapses in the availability of needed care at facilities and timely resupplying of working materials to CHW, and the cost and complexity of the intervention vis-à-vis local health systems absorptive capacity for strategic change. Strategies that strengthen and align communities' and health systems core capacities, as well as their ability to learn, adapt and integrate evidence-based interventions, are needed to maximize the impact of CHW on MNCH.

1. Introduction

The evidence that community-based primary health care (PHC) interventions can improve maternal, neonatal and child health (MNCH) has increased over the past few decades.¹ This owes, in large part, to studies that focus on specific tasks of community health workers (CHW), such as activities related to safe motherhood²⁻⁴; child health⁵⁻⁸; family planning^{9,10}; infectious and non-communicable diseases¹¹⁻¹⁵; neglected tropical diseases¹⁶ and mental health.^{17,18} In recognition of their effectiveness, there have been numerous publications collating and comparing CHW experiences across countries¹⁹⁻²², reporting the costs and cost-effectiveness of CHW models^{23,24}, synthesizing evidence on the design and effects of CHW programs²⁵⁻²⁸ and developing conceptual understandings of how to scale up and integrate them into health systems.²⁹⁻³⁵ With this, opinions converged in favor of deploying CHW, with increasing numbers of proponents citing evidence that access to essential, low-cost interventions from these cadres can help end preventable mortality, particularly among children in low- and middle-income countries (LMIC).³⁶

Accordingly, in recent decades there has been a rapid expansion of CHW deployment in many countries. Despite this, respected observers have argued that the use of CHW remains an underdeveloped component of health systems in LMIC.¹ Indeed studies on the implementation of CHW programs have noted that support for CHW, their performance and integration into communities and health systems is uneven across and within countries.^{37,38} Frequently, evidence-based recommendations are not effectively applied as CHW programs are designed and implemented^{30,35,39}, and many CHW programs are fraught with challenges including poor planning; unclear or fragmented roles; inadequate training; weak supervision; lapses in logistical processes; tenuous accountability linkages; ineffective incentive structures; poor selection processes and dissatisfaction of communities.⁴⁰⁻⁴³

The impact of CHW can be maximized if implementers and policymakers understand the reasons for these problems well and adapt implementation strategies for deploying CHW accordingly. However, most research has focused on the effects of CHW on service utilization and population health with less attention to implementation process⁴⁴, and the contextual factors that influence success.^{45,46} Studies of CHW program implementation are often detached from rigorous evaluations of programs' impact.⁴⁷ To date there is limited evidence on CHW effectiveness from randomized studies delivered through routine health systems^{48,49}, and, furthermore, there is a lack of pragmatic evidence on CHW programs that achieved mixed results even though analyses of barriers to implementation success are needed to help programs.²⁵ In this paper, we share results from a cluster-randomized pragmatic implementation trial of a CHW program in Tanzania that was conducted from 2011-2015. This trial, called *Connect*, evaluated the impact of a CHW program on child survival, the primary outcome; and on MNCH behaviors, childhood morbidity and care seeking for sick children, the secondary outcomes. Since previous publications on *Connect* have reported that WAJA implementation had no statistically significant effect on childhood mortality,⁵⁰ we report here the impact of the intervention on secondary outcomes and findings from an embedded qualitative process evaluation conducted from 2012-2014.

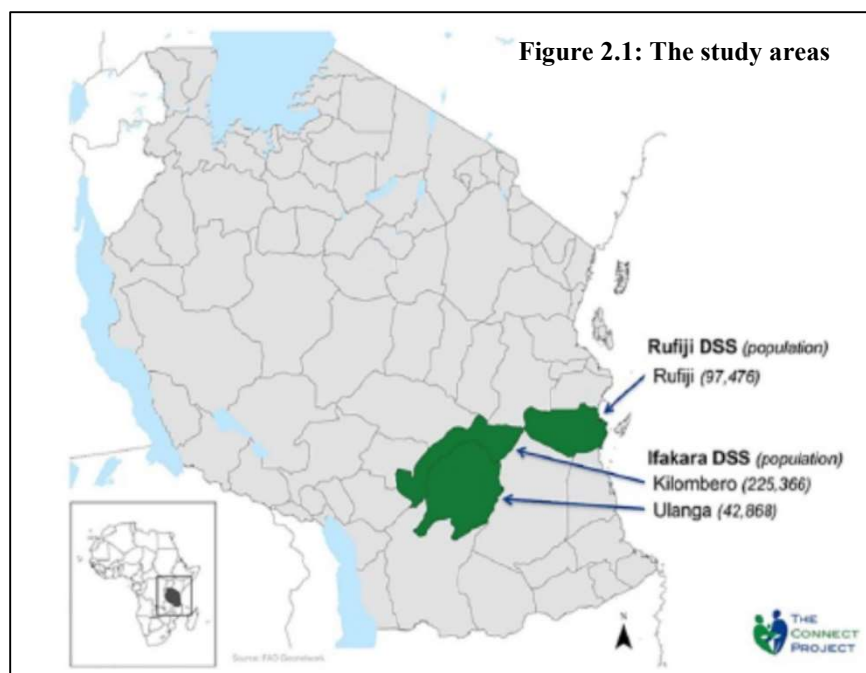
2. Methods

2.1 Study setting

Connect was situated in the sentinel areas of the Ifakara and Rufiji Health and Demographic Surveillance Systems (HDSS) managed by the Ifakara Health Institute (IHI). The Ifakara HDSS is in Morogoro, a landlocked region in southwestern Tanzania, and traverses two districts, Kilombero and Ulanga, that are approximately 500 km by road from Dar es Salaam, Tanzania's largest city. The Rufiji HDSS is in Rufiji district on the Indian Ocean coast approximately 150 km south of

Dar es Salaam by road. The population under surveillance in 2015 was approximately 380,000 (280,073 in Ifakara and 99,206 in Rufiji).^{51,52} See Figure 2.1.

2.2 Intervention design



The use of CHW has precedent in Tanzania.⁵³ Since their introduction in the 1970s, the deployment of volunteer CHW proved fraught with implementation and maintenance problems and failed to provide evidence that unpaid workers could

provide effective and sustainable means to extend PHC to communities.⁵⁴

In 2007, the Government of Tanzania promulgated the Primary Health Care Services Development Plan, known in Swahili as *Mpango wa Maendeleo wa Afya ya Msingi* (MMAM), which called for the revitalization community PHC by way of establishing a national cadre of paid, multi-tasked CHW.⁵⁵ In 2010, UNICEF and the Ministry of Health and Social Welfare of Tanzania (MOHSW) carried out a situation analysis of existing CHW programs in the country to inform recommendations on strategy for operationalizing the MMAM vision. Their report recommended that the national CHW, which they named *Wawezashaji wa Afya ya Jamii* (“community health enablers” or “WAJA”), be selected by their communities, formally trained and enrolled in national health sector scheme of service, accorded a salary and government recognition and tasked with

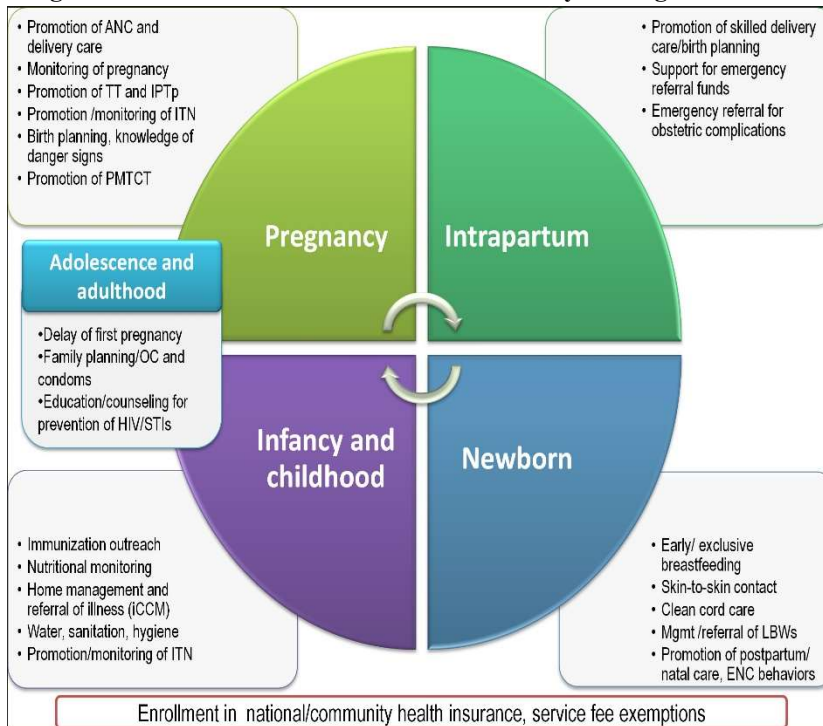
performing an integrated package of MNCH services.⁵⁶ Yet, at this time, there was no experience of operationalizing such a program, nor systematic evidence on whether the proposed CHW model would be acceptable, feasible and cost-effective or have an incremental impact on MNCH. In 2010, the IHI, MOHSW, the Tanzania Training Center for International Health and Mailman School for Public Health at Columbia University launched *Connect* to address that knowledge gap.

CHW recruitment and selection. The design of the *Connect* intervention is described in depth elsewhere.^{57,58} In 2010, IHI staff and Council Health Management Teams (CHMT) in Kilombero, Ulanga and Rufiji oriented community leaders to the intervention and recruited community members to stand for election to become WAJA. Community members could become a WAJA if they had received form four level education attainment (US grade 10) and passing grades in science, as per requirements for government employment, and longstanding residency in their current home village. Candidates whose eligibility was confirmed by local authorities were given 2-3 weeks to make their case to the community as candidates, and after that villages held elections to select their WAJA.

CHW training and service package. In October 2010, candidates chosen by their communities went to Ifakara to undertake training of 9-months, the minimum duration required for government employment. The curricula received national accreditation and comprised of two semesters of didactic and practical, clinic-based training in human biology; basic clinical skills; health promotion and disease prevention in the community; sexual and reproductive health; integrated management of childhood illness (IMCI); management of basic pharmaceuticals; stakeholder mapping and networking and community mobilization; and a community-based practicum. The WAJA service package was developed as a strategy to expand access to education on MNCH, mobilize villages to collectively promote community health, enhance referrals and utilization of

antenatal care (ANC), facility-delivery by skilled birth attendants, postnatal care (PNC), and make IMCI available in communities. WAJA also distributed oral contraceptives and condoms and performed basic curative care for gut infections caused by worms, fever, and non-complicated cases respiratory infection, diarrhea, and malaria. See Figure 2.2.⁵⁹

Figure 2.2: The *Connect* WAJA Service Delivery Package



CHW deployment and implementation management.

WAJA received employment contracts from local government authorities upon their graduation from training.

This accorded them a salary of approximately \$112 (United States dollar) per month.

Connect developed a two-

tiered system for supervising WAJA to promote the clinical quality of care, and to ensure that WAJA were accountable to communities. During training the CHMT appointed nurses or clinical officers to be ‘facility supervisors’ of WAJA that were deployed to communities in their facilities’ catchment areas. Village Health Committees from intervention communities appointed a ‘village supervisor’. Both supervisors participated in practical sessions of the WAJA training and participated in workshops with IHI staff on the WAJA role and work package, supervision and mentoring and community engagement.

Implementation arrangements of *Connect* balanced the overriding goal of understanding the effects of WAJA deployment through the routine health system with the reality that to deliver the program,

the health systems required support from development partners.⁶⁰ To provide this support, the IHI issued a financial subaward to Tanzania Training Center for International Health to develop and implement the training program. The IHI also issued subawards to the respective CHMTs to cover expenses associated with WAJA recruitment and selection; field practicum; the orientation of WAJA supervisors and their performance of timely supervision; health information system staffs' compilation of routine service delivery data recorded by WAJA; procurement and distribution of WAJA equipment, supplies and medicines; and payment of WAJA salaries. In addition, the IHI seconded Implementation Coordinators to the CHMT to help manage the deployment of WAJA and the launch of their activities in communities and establish processes for routinizing the provision of management and health system supports to the new cadre.

The provision of this assistance lasted for the first two years of the trial (2010-12) after which, the IHI withdrew direct support for implementation. At this point, CHMT were to obtain resources for maintaining the WAJA in their posts through the routine comprehensive council health planning process. From 2013 onward, the IHI led the evaluation of the WAJA intervention only, setting aside resources to ensure availability of remuneration, supervision, and essential supplies to WAJA if CHMTs failed to secure resources through the health system. *Connect* never intervened to influence the readiness or ability of facilities to provide referral level care. WAJA, 142 in total, were recruited, selected, and trained in two cohorts, the first deployed to 25 intervention villages in August 2011 and the second to the remaining 25 intervention villages in August of 2012.

2.3 *Outcome evaluation*

2.3.1 *Study design*

The detailed protocol for the *Connect* trial (registration number ISRCTN96819844) has been published elsewhere, including CONSORT checklist for pragmatic trials (Supplemental File

2.1).⁵⁷ *Connect* was a cluster-randomized pragmatic trial in the 101 villages within the areas of the Ifakara and Rufiji HDSS (63 in Ifakara, 38 in Rufiji). Stratified randomization was used to allocate 50 villages to the intervention arm and 51 to the comparison arm. The unit of randomization was the village. In 2010, a public drawing was organized to randomly assign villages to the two arms. Villages were block-randomized within the strata defined by village population size. Stratification was segmented by four categories to achieve a 1:1 match of communities in each arm that had <1000 population, 1000-2999, 3000-4999, and ≥ 5000 . Local government and village leaders attended the drawing, selecting representatives to draw pieces of paper with the name of each village written on them from containers numbered for each stratum. The villages chosen by the representatives were to be randomized to the intervention and comparison arms, and which representative represented which arm and the villages that they picked were concealed until after the drawing. Within the intervention arm, villages received between one and four WAJA depending on their strata. Villages allocated to the comparison received the ‘standard of care’ which comprised of routine activities coordinated by village governments to promote community health and households’ recourse to facility-based care for preventive or curative care. Because of the nature of the intervention, it was not possible to mask participants to their treatment status.

2.3.2 *Data sources*

The HDSS provided the platform to ascertain *Connect’s* primary outcome, childhood mortality. As this has been published elsewhere, we report here the effect of WAJA on secondary outcomes, MNCH behaviors and incidence of childhood disease. To obtain data for the evaluation of secondary outcomes, the IHI conducted household surveys in villages in the 101 villages before deployment of WAJA in April-August 2011 and after four years of implementation, April-August 2015.

HDSS censuses conducted in 2010 and 2014 provided the sampling frame for the pre- and post-intervention surveys. Calculations of data from a survey conducted in the same districts the year before indicated that to detect a minimum of 12% difference in the prevalence of secondary outcomes, the surveys needed to enroll per village a minimum of eight women that had had a live birth in the previous two years, and that to detect a minimum of 5% difference between arms in the incidence of childhood diarrheal or febrile/respiratory illness, the surveys would have enroll per community primary caregivers of at least 15 under-five year-old children (assuming $\alpha=0.05$, $\beta=0.80$, $k=0.25$ and two-tailed test).

Connect researchers employed ‘probability proportional to size’ techniques and used census data to randomly select households for recruiting survey participants.⁶¹ Participants were eligible if they were a female between the ages of 18-49 or the primary caregiver of an under-five year-old child and resident in the household that was randomly selected. In the end, both baseline and endline household surveys met the minimum sampling requirements, enrolling 3,267 and 3,027 women aged 18-49, respectively, including 882 and 778 mothers that had delivered a live birth in the previous two years. The participants reported on 2,104 under-five year-old children at baseline and 1,565 at endline. Over time, no adverse events were reported in either trial arm. See Supplemental File 2.2a and 2.2b, the participant flow diagrams.

2.4.3 *Data collection and management*

The data collection team comprised of Tanzanian research assistants with degrees in public health, sociology, or other relevant disciplines. They underwent a one-week training, which was followed by two days of pre-testing during which all data collectors administered the full survey at least twice. *Connect* staff divided the data collection team into groups and assigned each group to clusters of communities. Data collectors recruited all women of reproductive age that were resident

in each of the selected households no matter their childbearing history. Those that agreed to participate were read aloud an informed consent form (ICF). Individuals that agreed to participate either signed the ICF or provided an inked thumbprint to confirm their agreement to participate. Data collection was paper based. *Connect* embedded staff members in each data collection group to review completed surveys to assure their quality and pass them on for data entry into an Epi-Info database where they were cleaned and prepared for analysis.

2.4.4 Outcome measures

This study reports on the effect of WAJA deployment on secondary outcomes listed in Table 2.1. All outcomes were self-reported by household survey participants. Outcomes one to five in Table 2.1 refer to service utilization and health behaviors practiced by mothers with respect to their most recently born child if the child was born within two years prior to the survey. Outcomes six and seven refer to the incidence of childhood morbidities in the two weeks prior to the survey among all children under five years of age whose mothers or other primary care givers participated in the survey. Outcomes eight to 10 refer to the timeliness and appropriateness of care received by all under-five children if their mother or caregiver reported that they had become ill with diarrheal, febrile, or respiratory sickness during the two weeks before the survey.

Table 2.1: Secondary outcomes measured through *Connect* household surveys (2011 and 2015)

1	First trimester antenatal care (ANC) initiation
2	4+ ANC sessions
3	Facility delivery
4	Exclusive breastfeeding
5	Postnatal care (newborn)
6	U5 diarrhea incidence
7	U5 cough/fever/difficulty breathing incidence
8	Oral rehydration therapy (ORT) for children with diarrhea
9	Malaria test for febrile children
10	Appropriate care ⁶ for children with febrile or respiratory symptoms

⁶ Artemisinin Combined Therapy (ACT) for malaria or antibiotics for respiratory illness.

2.4.5 Data analysis

We estimated the effects of WAJA deployment using an intent-to-treat approach and logistic difference-in-difference (DiD) regression analysis with fixed effects for time and trial arm and random-intercepts to account for clustering of observations within villages. See Equation 1:

$$\text{logit}(y_{ijk}) = \beta_0 + \beta_1 \text{time}_k + \beta_2 \text{int}_{jk} + \beta_3 (\text{time}_k \times \text{int}_k) + u_{ij} \quad (1)$$

where y_{ijk} is the log of the odds that the k th individual in the j th cluster in the i th treatment arm experienced the outcome (Table 1), $time$ is an indicator of data source of observation k whereby $time$ equals zero for baseline and one for endline, int is the intervention group dummy for individual k in cluster j , and $time \times int$ is the DiD indicator of the time by intervention interaction. β_3 is the DiD estimator for the effect of WAJA deployment on outcomes above and beyond changes associated with the passage of time.

Given the balance of the sample before the intervention (Supplemental File 2.3), we did not control for individual-level characteristics of participants. In the model, u_{ij} is the random effect corresponding to the j th cluster in the i th trial arm and is normally distributed. Because the combination of binomial variation within clusters and normal variation between clusters, quadrature methods were used to maximize the likelihood and obtain parameter estimates, cluster robust standard errors and confidence intervals and conduct the significance test for this model. The DiD approach relies on the ‘parallel trends’ assumption. While we could not formally test this assumption, we used data from the HDSS to verify that child mortality trends in the two arms were similar during the 10 years prior to the trial.

2.5 Process evaluation

2.5.1 Qualitative study design

In July and August 2012, 2013, and 2014, respectively, we carried out a qualitative process evaluation. The goal of this was to understand the changes that arose in the local health system and communities as a result of introducing WAJA, whether those changes led to proximal outcomes associated with desired health and behavioral changes, and how contextual factors shaped that process. We situated the qualitative process evaluation within the same ‘nodes’ in each year, which we defined as intervention villages, their aligned PHC facilities which received WAJA referrals and provided WAJA supervision, and their respective CHMT.

We purposively sampled four nodes in Ifakara and two in Rufiji. We sampled two urban nodes in Ifakara and Rufiji, respectively, and four rural nodes. Participants from the community-level were parents of under-five year-old children, WAJA, village supervisors of WAJA, Village Executive Officers and Village Chairpersons. Participants from the health system included WAJA health facility supervisors, District Medical Officers, District Reproductive and Child Health Coordinators and District *Connect* focal persons. We analyzed transcriptions of 75 focus group discussions (FGD) and in-depth interviews (IDI). See Table 2.2.

Table 2.2: Qualitative data collection methods and participants

Informant Type	Year and data collection method					
	2012		2013		2014	
	IDI	FGD	IDI	FGD	IDI	FGD
WAJA	5	2	4	4	8	0
Other community stakeholder	3	4	5	0	6	0
Health facility staff	4	2	4	2	5	0
District-level health management staff	5	0	6	0	6	0

2.5.2 Qualitative data collection and management

Qualitative data collectors comprised of a team of experienced Tanzanian researchers with degrees in public health, sociology or another relevant discipline who received a 3-day training. After this, they pre-tested the instruments in an intervention setting that were not situated in the process

evaluation nodes. These steps were conducted in all three years of the process evaluation. Each IDI and FGD was conducted in Swahili and facilitated by two data collectors, one that led the interview and the other that took notes. Prior to the onset of the interviews, the data collectors administered an informed consent process in which they read aloud an ICF. Participants that agreed to participate either signed the ICF or provided an inked thumbprint. All interviews and discussions were recorded on a digital device. Data collection pairs transcribed all interviews and discussions, in Swahili, within one day of completing them. Swahili language transcripts were reviewed by a qualitative specialist from the *Connect* team to assure their quality. Transcripts and ICF were maintained as password encrypted electronic files or in locked cabinets at the IHI. Transcripts were then cleaned and translated into English.

2.5.3 *Qualitative data analysis*

To analyze the data, we first reviewed the transcripts, memoing extensively on patterns in the data, their meanings and ways in which these could be studied in a more structured analytical process.^{62,63} Based on this, we constructed causal pathway models (CPM) to develop a theory of how WAJA worked to produce the evaluation outcomes and how contextual conditions influenced that process.⁶⁴ In doing so, we identified the relationships between the following constructs in the data: elements of the *Connect* implementation strategy, proximal outcomes, the mechanisms the strategy triggered to affect proximal outcomes, and the determinants that either helped or hindered implementation. We established these constructs as analytic themes and created codes aligned to each theme.

We uploaded the 75 transcripts into *Dedoose* analytic software and coded. For this we adapted steps associated with grounded theory.^{65,66} First, we conducted ‘open coding’ in which we utilized codes from the ‘strategy’, ‘outcome’ and ‘determinant’ themes. Then, we sorted the coded

transcripts by implementation outcomes codes and examined relationships within and across segments of code to examine which factors exerted the most influence over implementation, whether these influences were positive or negative, and how they shaped outcomes. Using segments of text that were assigned ‘open codes’, we, then, pursued ‘axial coding’, utilizing codes from the ‘mechanism’ theme only. In this analysis, we defined ‘mechanisms’ as the events or process through which strategies produce outcomes.⁶⁷ After identifying mechanisms, we observed thematic linkages in the data with an emphasis on understanding how determinants affected the activation of mechanisms and whether they generated expected outcomes. We used coded segments of text to map findings against CPM configurations that we had developed earlier to refine them and reach conclusions about the mechanisms and determinants of WAJA implementation.⁶⁸ Finally, we triangulated our qualitative and quantitative findings, and linked conclusions on the generative process of implementation outcomes with evidence on WAJA effectiveness. Based on this, we formulated hypotheses on the relationship between implementation dynamics and MNCH outcomes that were measured during the trial.

2.6 *Ethical considerations*

Approval for the *Connect* trial was granted by the ethical review boards of the IHI (IHI/IRB/No. 16-2010), the National Institute for Medical Research of Tanzania (NIMR/HQ/R.8a/Vol.IX/1203), and the Institutional Review Board of Columbia University Medical Center (Protocol AAF3452).

3. Results

3.1 *Outcome evaluation*

3.1.1 *Effect of WAJA intervention on MNCH outcomes*

The characteristics of women and children across intervention and comparison arms before the trial were generally similar (Supplemental File 2.3). Table 2.3 shows the effect of WAJA on MNCH outcomes. During the analysis, we ran two analyses to evaluate WAJA impact on ANC and PNC utilization. The first was a per protocol analysis that only considered facility-based visits as events. Because WAJAs' service package with regards to ANC and PNC is equivalent to care available at frontline PHC facilities which tended to lack laboratory capabilities and clinicians, we conducted an additional analysis in which we considered WAJA performance of safe motherhood promotion and postnatal visits to pregnant women as events that count toward indicator 2 and 5 in Table 1. The intervention had no effect on the mother-level outcomes that had been established *a priori*. There is some indication that the intervention might have inhibited utilization of ANC and PNC at clinics, possibly since WAJA performed the information, education, and counseling components of these services in households, though all findings are statistically insignificant. Our analysis that considered WAJA performance of safe motherhood and essential newborn care counseling in households toward the outcome, suggests that, even though the intervention failed to increase referrals from communities to clinics, WAJA implementation improved population-level coverage of information, education, and communication related components of ANC ($\beta_3 = 1.91$; 95% CI=1.14-2.86, $p = 0.035$), and PNC ($\beta_3 = 1.98$; 95% CI=1.08-3.31, $p = 0.010$) information, education, and counseling.

Our findings indicate that the WAJA intervention improved child health. Above and beyond changes in the incidence of childhood illness that occurred with the passage of time, the reduction in the odds of diarrhea incidence was greater among under-five year-olds in intervention villages than in comparison villages ($\beta_3 = 0.51$, 95% CI: 0.32-0.81, $p = 0.004$). We found a similar outcome regarding the incidence of childhood febrile and respiratory illness ($\beta_3 = 0.59$, 95% CI: 0.38-0.94,

Table 2.3: Effect of WAJA deployment and implementation on MNCH outcomes.

Outcome		Baseline (2011)			Endline (2015)			Difference in Difference (Impact)		
		Intervention (%, (n))	Comparison (%, (n))	Diff. %	Intervention (%, (n))	Comparison (%, (n))	Diff. %	β_3 (DiD estimator)	95% CI	<i>p</i> value
<i>Mother-level outcomes^z</i>										
1	First trimester ANC initiation	16 (72)	17 (72)	1	17 (67)	24 (92)	7**	0.65	0.38, 1.13	0.13
2	4+ ANC sessions (facility only)	41 (188)	44 (189)	3	43 (169)	46 (177)	3	0.81	0.39, 2.32	0.35
	4+ ANC sessions (Facility and WAJA) [^]	NA	NA	NA	53 (212)	46 (177)	7**	1.91	1.14, 2.86	0.04
3	Facility delivery	71 (322)	74 (320)	3	83 (326)	86 (331)	3	0.81	0.46, 1.44	0.48
4	Exclusive breastfeeding	41 (186)	39 (168)	2	45 (177)	39 (150)	6	1.33	0.85, 2.06	0.21
5	≥1 PNC session (Facility only)	36 (166)	34 (147)	2*	25 (98)	33 (127)	8*	0.83	0.52, 1.33	0.44
	≥1 PNC sessions (Facility and WAJA) [^]	NA	NA	NA	54 (212)	33 (127)	11**	1.98	1.08, 3.31	0.01
<i>Child-level outcomes^o</i>										
6	Diarrhea in past 2 weeks	13 (135)	11 (124)	2	6 (48)	12 (92)	6***	0.51	0.32, 0.81	<0.01
7	Febrile & respiratory symptoms in past 2 weeks	14 (138)	12 (127)	2	5 (40)	11 (85)	6*	0.59	0.38, 0.94	0.03
8	ORT for children with diarrhea [†]	56 (75)	61 (75)	5	54 (26)	41 (38)	13**	1.71	1.06, 3.18	0.07
9	Malaria test for febrile children [†]	49 (171)	55 (200)	6*	69 (84)	60 (93)	9**	1.80	0.99, 3.27	0.05
10	Appropriate care for children w/ febrile or respiratory symptoms [†]	50 (182)	49 (195)	1	58 (110)	49 (112)	9**	1.68	1.00, 2.91	0.05

***denotes statistical significance at 1%, ** 5%, * 10%

^zFor mother level outcomes, n=454 and 394 in intervention arm at baseline and endline; and 428 and 384 in comparison arm at baseline and endline.

^oFor child-level outcomes, n=1,038 and 798 in intervention arm at baseline and endline; and 1,066 and 767 in comparison arm at baseline and endline.

[^]ANC and PNC services components performed by WAJA were information, education, and counseling activities only, no clinical interventions.

[†]Out of 135 and 48 children with diarrhea in intervention arm at baseline and endline; out of 124 and 92 children with diarrhea in comparison arm at baseline and endline.

p=0.028). Findings indicate significant effects of WAJA on access to essential care and treatment for diarrheal, febrile, and respiratory sickness in children. After accounting for changes that occurred with the passage of time, we found that children in intervention villages that had diarrhea in the two weeks prior to data collection were 1.71 times more likely to receive ORT than such children in comparison settings; however, this finding was not significant at a 0.05 level ($\beta_3 = 1.71$, 95% CI: 1.06-3.18, p=0.074). However, the effects of WAJA exposure on children with febrile and respiratory illness were greater. Compared to children with fever in comparison areas, those in intervention communities were 1.80 times more likely to receive a malaria test ($\beta_3=1.80$, 95% CI: 0.99-3.18, p = 0.050), and compared to children with febrile and/or respiratory symptoms in comparison communities, those in intervention communities were 1.68 times more likely to receive either antibiotic or ACT treatment ($\beta_3 = 1.68$, 1.00-2.91, p=0.048).

3.2 Process evaluation

Figures 2.3-2.6 are the refined CPM that illustrate the findings of the qualitative process evaluation. *Connect* deployed an implementation strategy that included nine salient components (Cells 2, Figure 3-6) to address specific modifiable factors (Cells 1) by activating mechanisms (Cells 3), which *Connect* hypothesized would generate proximal outcomes (Cells 4) on the pathway to the MNCH outcomes (Cells 5). Whether the implementation strategy components could succeed was determined by preconditions in the environment, the presence or absence of which were essential for or prevented implementation success (Cells 8 and 9). Implementation effectiveness was also moderated by contextual factors (Cells 6 and 7), which amplified or diminished the force with which mechanisms incurred the intended effect.

The qualitative analysis reveals how the *Connect* causal pathway played out at four levels.⁶⁹ The implementation strategy engaged community members in recruiting, selecting, and deploying

WAJA to generate the perception that the intervention originated locally and had been adapted to meet local needs, and, with this, promote communities' acceptance of WAJA and adoption of desired behavior changes (Figure 2.3). *Connect* fostered collaboration between health system and community stakeholders, designed a WAJA work package and eligibility requirements that were responsive to stakeholders' needs and consistent with health system processes. The objective of this was to align communities' and health systems' receptivity to the program, ensure the intervention was compatible with the meanings that stakeholders attached to it, and make it feasible for them to adapt to changes instigated by WAJA implementation (Figure 2.4). *Connect* also built WAJAs' capacities through training; contracted and remunerated the cadre; and created systems to supervise WAJA and meet their logistical requirements. *Connect* believed this would motivate and engender confidence in the cadre, establish PHC delivery readiness in communities, and, thereby, help achieve better MNCH outcomes (Figure 2.5). Finally, by providing financial and technical support to CHMT, *Connect* sought to enable the health systems' adoption of the WAJA program as a core and sustainable component of the larger PHC system (Figure 2.6). These causal pathways came to fruition with mixed results.

3.2.1 Beneficiary level

At the beneficiary level consistent patterns emerged across examples of community members that developed positive connections with WAJA. Community members generally referred to their relationship with WAJA in terms of a kinship bond, calling them '*WAJA, watoto wetu*' (WAJA, our children), '*vijana vyetu*' (our youths), '*wanangu*' (my child). In addition, the participatory recruitment and selection instilled in villagers' confidence in the cadre. According to a mother: "Because of our faith in [WAJA], we selected them. So, they cannot do anything to betray us." (Mother, Lukolongo, Kilombero, 2012).

However, their connection to communities occasionally caused difficulty.

One day I was educating my old friends about family planning, and they asked me 'how many are we in our family', because they know we are many. They told me 'How come you are many and now you are telling us about family planning?' That also is a problem. (WAJA, Nyambunda, Rufiji, 2013).

Others believed that it was inappropriate for youth to get involved in reproductive health issues. A

Village Supervisor reported:

Many pregnant women are not free to show their condition. If you give them education, they says 'this [WAJA]with no family, how does he know to tell me about [being pregnant]?' (Village Supervisor, Lumemo, Kilombero, 2012).

The qualitative data, however, illustrates that most resistance to the intervention dissipated as communities acquired experience with WAJA. This was facilitated by involvement of village leaders in introducing WAJA to communities and establishing clarity on their roles. Community members recall leaders “calling a village meeting where [leaders] say to citizens ‘those WAJA who studied are back with this responsibility’ and sending them to the streets where we recognize them” (Mother, Minepa, Ulanga, 2012). In other instances, WAJA reported difficulty in gaining communities’ trust: “At the beginning, the biggest thing was the lack of being known [because] there was no meeting. In our village there are conflicts and there have not been village meetings in months.” (WAJA, Lukolongo, Kilombero, 2012).

Over time, word spread in communities that helped establish widespread acceptability of the program. According to one early skeptic,

“We first saw these [WAJA] and said how is it that they have become our doctors? Then we hear from more [people] that [WAJA’s] medicines were good, and they give good lessons. Before when our child got sick there was rushing [to facilities], but now we do not rush to facilities anymore.” (Father, Kisawasawa, Kilombero, 2014).

Community members believed that WAJA understood them especially and tailored services to meet their needs. According to a mother:

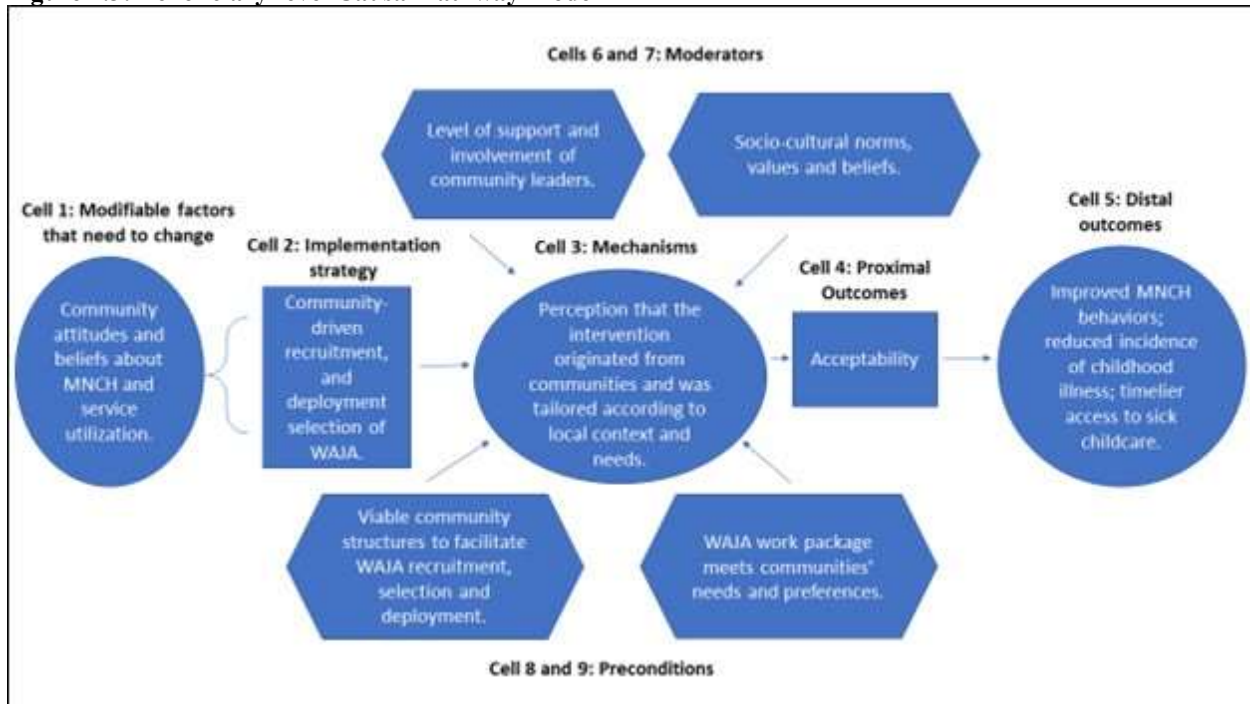
The method [WAJA] use is good language. If your child needs a treatment, they sit with [the caregiver] and they advise her. By telling her to go to the hospital based on the good language he used, she understands and will go rather than short answer language we don't understand.” (Mother, Mgomba North, Rufiji, 2013).

The acceptability of the WAJA created conditions in which beneficiaries could learn and adopt desired MNCH practices. Nevertheless, participants, especially men, lamented that WAJA could not perform more services.

WAJA don't have an idea on how to treat [men]. So, I would like to see WAJA being trained more so that they can help us rather than going to the health facilities (Father, Mgomba North, Rufiji, 2014).

As Figure 3 shows, components of the implementation strategy that targeted factors at the beneficiary level achieved success. Preconditions for effectiveness were in place at the outset of implementation, and *Connect* activities withstood adverse moderating factors or helped to mitigate their negative influences. Overall, the analysis suggests that change mechanisms were activated and generated the intended responses. See Figure 2.3.

Figure 2.3: Beneficiary-level Causal Pathway Model



3.2.2 Stakeholder level (local health system and community)

At the support system stakeholder level, *Connect* sought to establish an ‘intervention-values-systems fit’ in which members of communities and the local health system had a shared receptivity to the intervention and opportunities and abilities to adapt to the changes it introduced. This was based on the belief that if there was alignment between characteristics of the intervention, the meanings and values attached to the WAJA program by those affected by it in communities and local health system, and their absorptive capacities for strategic change, then effectiveness of the program would be greater.^{70,71} To establish a shared receptivity to the intervention, *Connect* facilitated collaboration between district employees and community members during intervention startup. According to a member of the Kilombero CHMT that served as focal person to *Connect*:

The village has a process to make sure that applicants belong to that village and know how to serve [the village]. Then, still the district team checks the [authenticity of] education certificates and interviews [applicants]. Then if [WAJA] are chosen by the village and complete studies the district hires them. We have boarded the same bus to start this program (Connect Focal Person, Kilombero CHMT, 2012).

In addition, the WAJA service delivery package included both preventive and basic curative care for children as a measure of “first aid” to handle simple illness. As a village supervisor from Rufiji explains, this combination helped address communities’ perceived needs.

Before WAJA your child may be sick but [parents] are not aware because lack of education. Then it becomes emergency, so the child will get more sick while you get the money, but now when the child is sick you can see it right away, and WAJA gives drugs as first aid (Village Supervisor, Mangwi, Rufiji, 2013).

Facility staff also appreciated the blended service delivery package, which they felt helped rationalize recourse to clinics for care. They were also pleased that the training and eligibility requirements were compatible with how the local health system worked.

This project has been received well by the district because WAJA have received nine-month training, which means they can be hired and because of that their contracts were approved. This shows that [WAJA] are employed and will work as normal staff do. (District Medical Officer, Rufiji, 2014).

However, as implementation continued, weaknesses of local health system constrained districts’ absorptive capacity. Rather than intervene to ensure optimal implementation conditions at facilities, *Connect* relied on local health systems to adapt independently to the changes that were instigated by WAJA deployment. This frustrated WAJAs, who recalled situations such as “I educated a pregnant woman before delivery to go [deliver at hospital] but when she reached [the hospital] she observed that there was no service, and she told her husband ‘there is not any service. Its better I deliver at home’” (WAJA, Mgomba North, Rufiji, 2013). Health care workers at facilities were upset by these failures. When WAJA referred sick children, staff at facilities, often, could not help:

A child might have malaria scorching hot, but you can fail to get medicine, or you can get medicine but fail to get syringe. You tell [the caregiver] to buy [the missing item] because we do not have enough working equipment (Health Facility Supervisor, Mngeta, Kilombero, 2013).

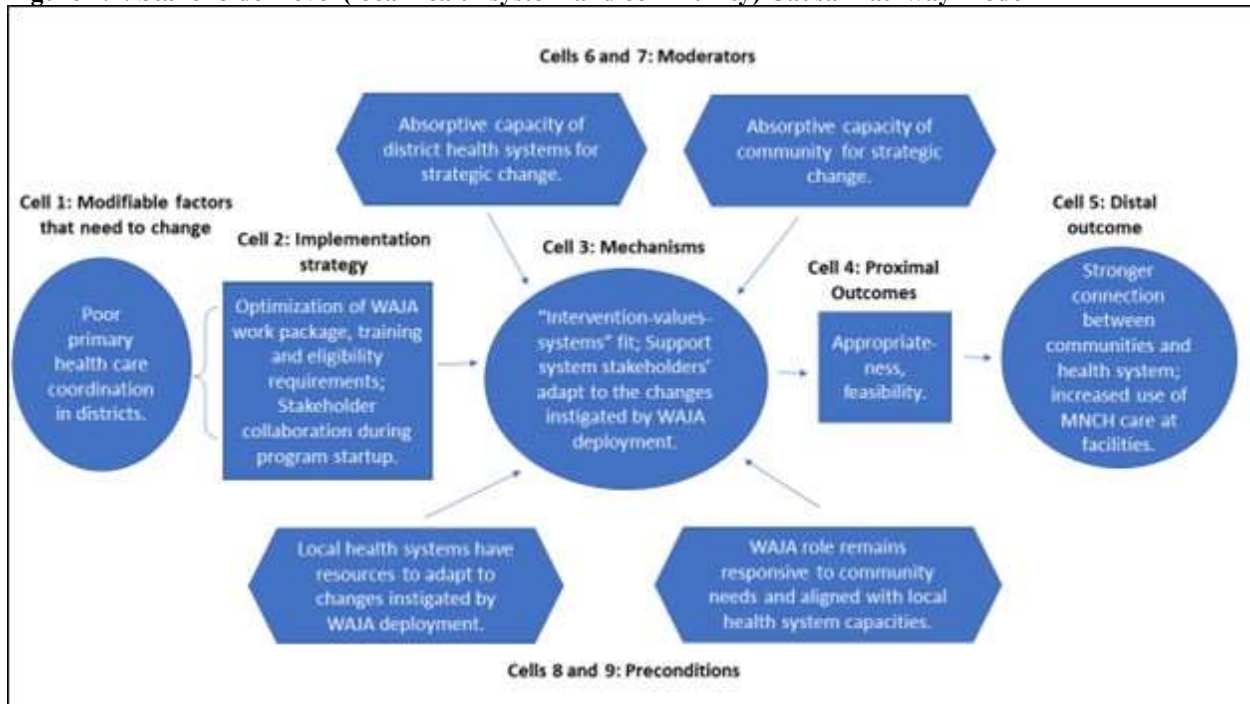
The fragility of logistics systems illuminated a deeper divergence between individuals involved in the intervention. Whereas *Connect* staff and district counterparts deployed WAJA to empower communities and *connect* them to the health system, community members felt WAJA should act as ‘doctors’ that address needs unmet due to the weak health system. According to one WAJA: “The difficulty is that people see me as a doctor. When I refer the patient... They ask, ‘why go to the facility where we get nothing if we chose you to work here for us?’” (WAJA, Lukolongo, Ifakara, 2012). Mothers voiced that perspective: “I would like WAJA to be given more trainings so that it will not be necessary to go to the hospital because when we go there, there are no medications” (Mother, Mgomba North, Rufiji, 2014).

Figure 4 illustrates the stakeholder causal pathway. Preconditions for the implementation effectiveness were not in place and the strategy did not contain elements that strengthened absorptive capacities of support system actors to adapt to the changes that ensued after WAJA deployment. Because of this, the targeted change mechanism, i.e., the ‘intervention-values-systems’ fit, was never activated. In turn, proximal and distal outcomes did not arise as intended. See Figure 2.4.

3.2.3 WAJA level

In addition, *Connect* sought to build capacity, inspire motivation and, in doing so, help establish PHC implementation readiness in communities. These interventions focused on WAJA, primarily, and included training, employment and remuneration, and clinical and community supervision. Throughout the trial, these inputs had their intended effect: beyond the training and deployment of WAJA, being paid and accorded an ‘organizational identity’ motivated WAJAs’ productivity and commitment:

Figure 2.4: Stakeholder-level (local health system and community) Causal Pathway Model



The thing that pushed me is that I had no work [before becoming a WAJA] and I had received little education and I observed that the community was struggling... Now when we pass through the community [community members] recognize us as ones that can help them... This motivates me (WAJA, Kisawasawa, Ifakara, 2012).

However, over time, three contextual factors affected WAJAs' sense of 'organizational citizenship'. Chronic failures in the supply chain in the later years of the project left WAJA feeling betrayed: "It is as if [the intervention] has entered the government system. First, they will replace [bicycle] tires, then they say they will replace medicines. But, when you wait you do not get the promises that they told us" (WAJA, Lumemo, 2014).

Relationships with health facility supervisors were crucial to WAJAs' motivation: "He always comes to ask us about the challenges we face and keeps regularly in touch to see if we need help with a patient. This is what motivates us, too" (WAJA, Minepa, Ifakara, 2013). Yet, there were frequent lapses in supervision, which supervisors attributed to excessive workload.

We have been given transportation, which gives us motivation to do our work effectively with the WAJA, at the village, but we do not get enough support with our

services. Who can provide our services if we are [in the community]? (Health Facility Supervisor, Mlabani, Ifakara, 2012).

WAJAs' success also depended on their relationships with village leaders. WAJA benefited when village authorities helped mobilize households, enforce community health rules, and solve complex problems. One WAJA reports:

Where I come from, it's the village health committee that makes decisions and implements the fines. So, as I pass through the community to inspect households, which are supposed to have a latrine toilet, I find some households have difficulty with this. When I go to this household, they might refuse or chase [me] away, so I report this so that the committee can help the household or charge fines. (WAJA, Lumemo, Ifakara, 2013).

However, WAJA did not always experience productive collaboration with village leaders:

In my village, the government does not cooperate. The public doesn't trust the executive. We had the lack of cupboards for storing the medicine. This was troubling [me] until there came some doctors who strongly rebuked [the Village Executive Officer]. Another thing the meetings.... The meetings should be held every three months, but since last year we have not had one. (WAJA, Mangwi, Rufiji, 2013).

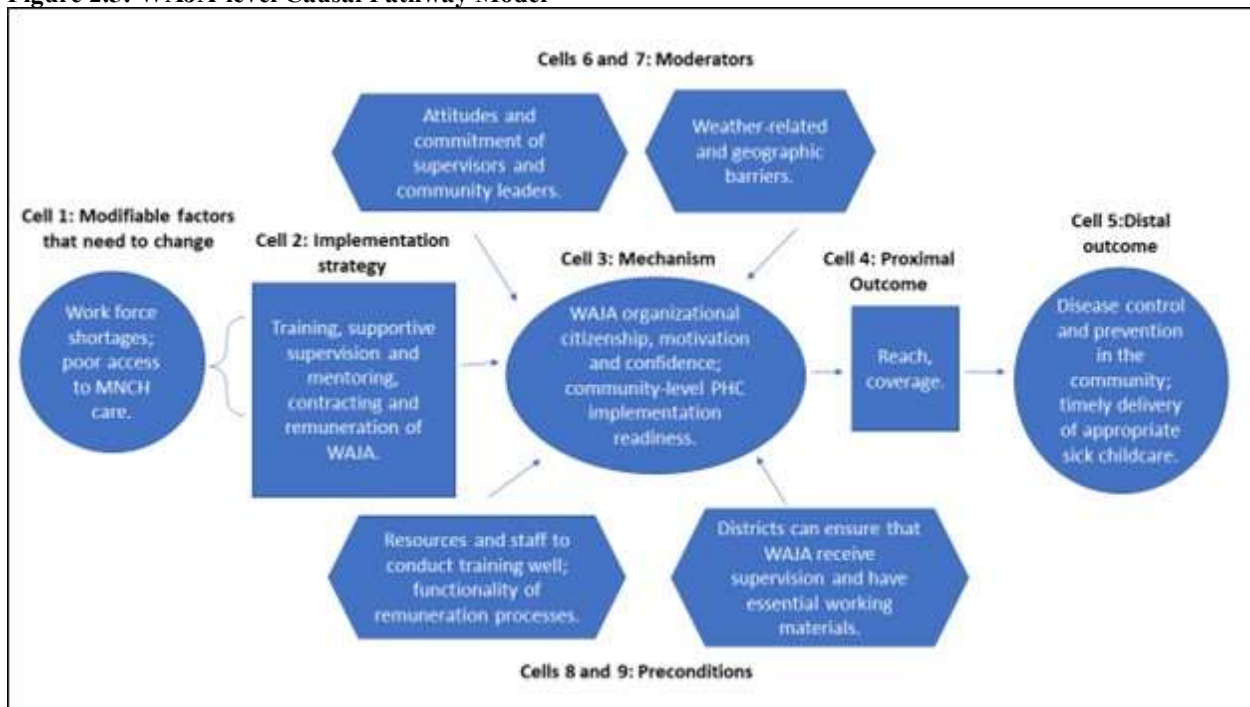
Ubiquitously, village stakeholders complained that while WAJA were remunerated, Village Supervisors and other members of village governments were not. As one Village Supervisor lamented: "We fail to do our job because we don't have allowance, and the WAJA will not listen to you, the supervisor, as you are only a volunteer while that one is getting paid" (Village Supervisor, Nyambunda, Rufiji, 2012).

In addition, geographic and logistical barriers impeded WAJA performance. The wetland terrain of the Rufiji delta was particularly vulnerable to extreme rainfall: "What disrupts our schedule is the weather condition. There are some hamlets which you cannot reach because of rivers that get created by monsoon rains" (WAJA, Nyambunda, Rufiji, 2012). WAJA deployed to expansive, rural communities struggle meeting coverage requirements: "What hinders us is the distances from

some homes and the rest of the community. Sometimes bicycle might be damaged, and the distance is so far, in the bush” (WAJA, Lukolongo, Ifakara, 2012).

Pursuit of the WAJA-level causal pathway was achieved with mixed success. Although the preconditions for implementation effectiveness were in place for some of the program, lapses occurred that were addressed by the *Connect Project* when districts could not. WAJA often struggled to overcome difficult community dynamics and environmental constraints. Nevertheless, triangulation of qualitative and quantitative findings suggests that, despite challenges, WAJA maintained implementation readiness in communities, which helped achieve some of the intended distal MNCH outcomes. See Figure 2.5.

Figure 2.5: WAJA-level Causal Pathway Model



3.2.4 Organizational level

Finally, at the organizational level, *Connect* sought to strengthen systems to adopt and manage the WAJA intervention. This started with the IHI providing financial and embedded technical

assistance to CHMT during the first two years of the trial. Although *Connect* backstopped districts in the provision of essential supports to WAJA from 2013-15, the strategy largely failed at incorporating the intervention in the government system. One stakeholder commented: “The challenge is how we can incorporate [WAJA] in the health system. That challenge is too big for us, as it requires cooperation between us, the Ministry of Health as well Ministry of Regional Government, which now we do not see” (District Medical Officer, Ulanga, 2014).

In addition, district leaders felt that the CHW sub-system was simply too costly and complicated to adopt in two years.

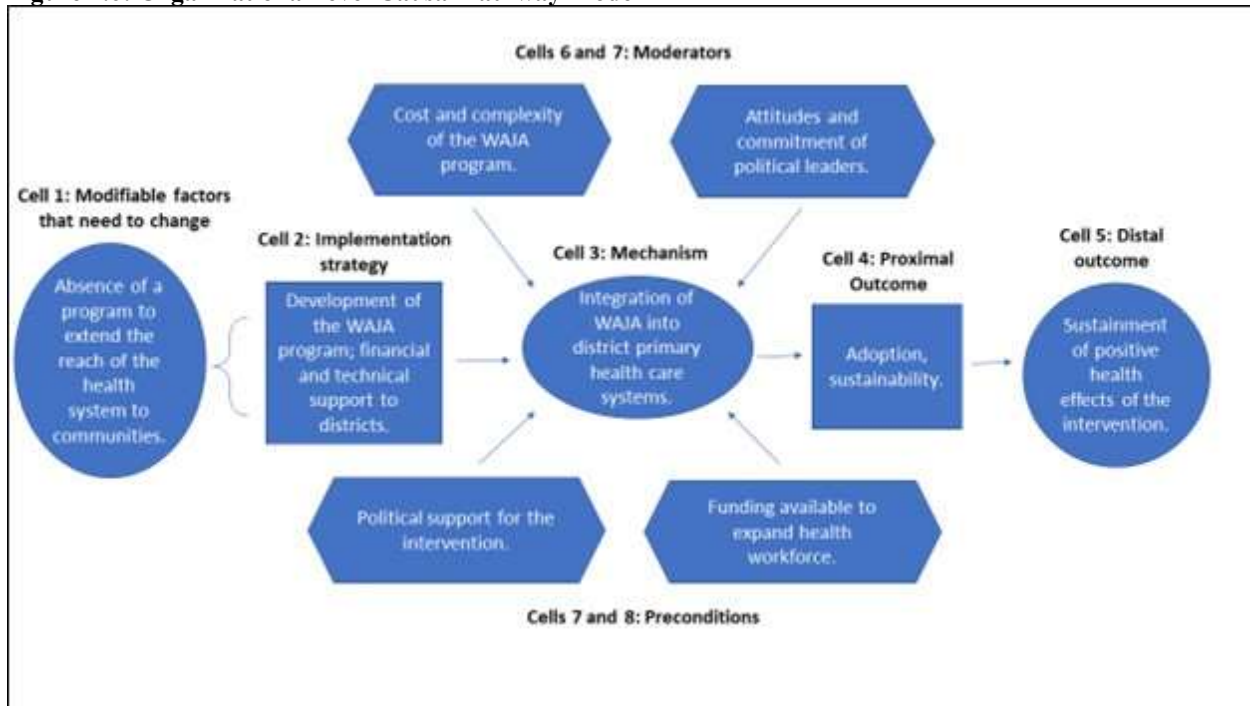
WAJA program has come here as pilot and will not last forever, and we need more resources. Our budget from the basket fund should be spent on services at facilities. ‘OS’ (Other Sources), which we received from the central government, this can go to [the WAJA intervention] ... But if you spend on office affairs, fuel, the allowances, salaries, and everything in the administration activities, it is not enough. (District Medical Officer, Rufiji, 2014).

It follows that, during 2013-2015, WAJA depended on support from the IHI to sustain implementation.

As a district I can't say that we can accommodate WAJA so that they can be sustainable. The WAJA are still doing their duties as usual and I still visit them to deliver supplies and do follow up, but for most things now we wait for it to get done [at the IHI] with Connect (Connect Focal Person, Kilombero, 2014).

As these stakeholders discussed, the combination of weak health systems and an inadequate district support strategy contributed to breakdown in the organizational level causal pathway. Preconditions for strategic success were never in place and *Connect’s* financial and technical support to districts was insufficient vis-à-vis the costs and complexity of integrating the WAJA into local health systems. See Figure 2.6.

Figure 2.6: Organizational-level Causal Pathway Model



4. Discussion

This study evaluated the effect of WAJA deployment on MNCH service utilization, childhood morbidity and access to sick childcare, and explained how these findings were generated. The quantitative analysis demonstrates that the intervention failed to connect communities to the formal health system; however, WAJA deployment was associated with childhood morbidity reduction as well as increases in timely access to appropriate of sick childcare and coverage of information, education and communication components of ANC and PNC. The qualitative analysis elucidates how the implementation strategy did and did not affect intended outcomes and the determinants of that process. Altogether, this study provides an opportunity to hypothesize about the relationship between CHW interventions, the mechanisms through which they change proximate outcomes, how contextual conditions shape these processes, and how this affects MNCH.

Intervention source, adaptability, and visibility of results. By engaging communities in cocreation of the intervention, adapting training procedures, and using community members to provide MNCH care, *Connect* elicited the perception that the WAJA intervention had been internally developed and that lifesaving interventions had been adapted and configured in the social environment to meet communities' needs. Early-stage resistance subsided as skeptics became familiar with the WAJA and observed their impact. These findings add to literature on acceptability and diffusion of innovation, which emphasizes similar factors.^{71,72} This analysis suggests that these mechanisms helped *Connect* achieve critical proximal outcomes, such as legitimacy, household members satisfaction, and community acceptance, which, we conjecture, facilitated caregivers' uptake of health behaviors that, in turn, led to positive health effects, importantly the reduced risk of childhood illness. Additional more focused research is needed to better understand the mechanisms through which community-based programming triggers acceptability, and to demonstrate whether this is associated with behavior change.

However, our analysis found that the process used to determine the WAJA service delivery package did not sufficiently engage communities and include some components that were valuable to them, and was, thus, was a notable barrier to acceptability. Future research ought to investigate the feasibility of strategies that integrate citizen accountability structures into PHC policy and implementation processes.^{73,74} Furthermore, the qualitative analysis found that acceptability was greater in communities with strong leadership that proactively supported WAJA.^{34,75–77} Future research should focus on ways to strengthen these structures as a step toward incorporating communities into wider health systems.

Intervention-values-systems fit. The qualitative findings bring into focus how weaknesses of the health system stymied creation of such a 'intervention-values-systems' fit.^{70,78} Whereas *Connect*

managers viewed WAJAs as ‘connectors’ deployed to link communities to the health system and motivate them to take prevention and promotion of health into their own hands; community members saw WAJA as ambulatory doctors in place to compensate for a health system that failed them. This divergence of perspectives and misalignment of the intervention design, communities’ perceived needs, the readiness of the health system to routinely provide MNCH care, and its limited absorptive capacity produced a climate of implementation replete with referral noncompliance, ill-prepared WAJA and health care workers, and frustrated communities. This finding has implications for CHW programs that are situated in weak health systems. To succeed in these environments, CHW implementation strategies should incorporate systems strengthening components that address root causes of communities’ suboptimal use of facility-based care. We hypothesize that this will help establish an implementation climate of greater compatibility between the roles of CHW, communities’ perceptions and willingness to use the CHW intervention, and the capacity of delivery systems to meet expectations. In doing so, future CHW programs may succeed in areas where *Connect* did not, for example strengthening linkages between communities and the health system and increasing use of facility-based MNCH services.

Previous research has focused on compatibility between intervention features and capacities of delivery systems to learn and adapt^{79,80}, existing workflows and systems in the adopting organization^{81,82}, and implementer characteristics.^{83,84} In addition, studies have reported on the adaptability of interventions vis-a-vis value systems conditioned by religion, traditional social and communication networks and diverse cultures.⁸⁵⁻⁸⁸ This analysis illuminates how characteristics of systems condition the ways in which stakeholders perceive, value and use interventions; and how this affects the introduction of CHW. Future research should explore these relationships, and how these shape the prospects of introducing evidence-based interventions in health systems.

Readiness for implementation. *Connect's* pragmatic objective to build capacity of the system to adopt and implement the WAJA intervention largely failed, a finding that is consistent with examples from many CHW initiatives which encountered organizational and sustainability challenges.⁸⁹ In particular, CHW programs that emphasize IMCI have struggled to maintain capabilities to implement essential components of integrated care systems, such as supervision, remuneration or incentivization of cadres and supply chain logistics.⁹⁰⁻⁹³ Future research should focus on how to strengthen public sector health system leadership and coordination capacities as a precursor to extending the reach of PHC programs to the community-level.^{58,94}

Motivation and organizational commitment. Beyond building WAJA knowledge and skills, *Connect* succeeded at engendering the commitment of the cadre by giving them contracts and remuneration for performance, extending formal supervision processes to the community, and tapping into existing community structures to avail WAJA with necessary support. The qualitative analysis illustrated how this triggered motivation, confidence, and enhanced organizational citizenship behaviors. These mechanisms not only made WAJAs' work more feasible and enabled their reach in communities. Moreover, they withstood adverse contextual influences, such as lapses in health system functionality, inconsistent support from community leaders and geographic and weather-related barriers and enabled the achievement positive health effects. Based on this, we conjecture that programs that blend efforts to professionalize CHW, facilitate opportunities for supervision and support, and ensure the functionality of logistics systems elicit levels of hard work, motivation, and confidence from CHW that, in turn, leads to desirous performance outcomes.

In LMICs, there is dearth of research on the dynamic interplay between individuals and their organizations and how this affects implementation. Our finding that the professionalization of WAJA enhanced their willingness to work hard echoes earlier studies which highlight the

relevance of personal growth, professional development, and both working and social relationships to CHW motivation.⁹⁵ It is noteworthy that WAJA motivation was inhibited when incentivization schemes came into tension with social relationships and hierarchies. Bhattacharrya et al. make a distinction between factors that motivate CHW and factors that motivate others to support and sustain CHW.⁹⁶ Indeed, ‘complementary incentives’ are an important consideration when establishing incentivization schemes not just for CHW but for sustaining the wider ‘community health system’.⁹⁷

Integration of communities into the health system. The qualitative analysis illuminated contextual factors that impeded organizational adoption of the intervention, an objective of *Connect*. Notably, the costs and complexity of doing so was high relative to extant capacities of systems to learn and adapt. Earlier studies have underscored the need to anticipate these demands and address them proactively to ensure that delivery systems are poised to depart from existing practices, mindful of the intricacy and number of steps required to do so and a realistic sense of the increase in organizational target units that must be reached by implementation.^{71,98–100} Previous research has also demonstrated the importance of external policies, incentives, or regulations, led by governments or other central entities, that instruct, motivate and channel direct and indirect support to implementers for the uptake and spread of interventions in health systems.^{37,43}

The study suffered from some limitations. Recall bias may have interfered with measurement in the surveys, which relied on participants’ recollection of care received, in some instances, as far back as two-years prior to data collection. The analysis conducted multiple statistical tests and, therefore, faces risks associated with multiplicity of analyses and outcomes. Qualitative findings might have been affected by ‘social desirability’ bias in that respondents might have adulterated their responses to please the team that led the intervention. Child survival in the study districts

during the time of and surrounding *Connect* had been improving at a pace more rapid than was average for much of Tanzania. Thus, it is not clear if the same results would have been posted had WAJA been deployed in other districts. Finally, our discussion features hypotheses of the linkages between intervention components, mechanisms of change, implementation determinants, proximal outcomes, and health effects. Although these propositions are substantiated by our analysis, additional research is needed to understand these relationships more deeply and test them.

5. Conclusion

The evaluation of *Connect*, a pragmatic trial with embedded implementation research, shows the outcomes and processes of introducing CHW in the realities of a health system struggling to maintain effective coverage of facility based PHC. The attribution of null effects to systemic weaknesses points to the need for strategies that strengthen and align community and health systems' core capacities, as well as their abilities to learn, adapt and integrate best evidence-based interventions. In the case of WAJA in Tanzania, there is evidence that suggests that, by addressing this gap, it is possible to accelerate child mortality reduction and improve MNCH. If policymakers, implementation teams and communities, and researchers work together with a common vision of community health systems strengthening, they can help achieve universal health care in Tanzania and similar settings.

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Chapter 4:

The impact of childhood Mortality on fertility in rural Tanzania: Evidence from the Ifakara and Rufiji Health and Demographic Surveillance Systems

Abstract

This manuscript examines the relationship between child mortality and subsequent fertility using longitudinal data on births and childhood deaths occurring to 15,291 Tanzanian mothers between 2000 and 2015. Generalized hazard regression analyses are employed to assess the effect of under-five year-old mortality on the hazard of conception, adjusting for child-level, mother-level and contextual covariates, including annual cluster-level period child mortality rates in the catchment of communities in which women resided during the time of the cohort. Results show that childhood mortality accelerates parity progression. Time to conception is most reduced if an index child dies during its subsequent birth interval, representing the combined effect of biological and volitional replacement. Deaths occurring during prior birth intervals were associated with accelerated time to conception during future intervals, which is consistent with hypothesized “insurance” effects of anticipating future child loss, but this is smaller than replacement effects. With regards to the main effect of the child mortality context in which birth histories are nested, the analysis reports that residence in areas of relatively high child mortality were associated with hastened parity progression, which is also consistent with the “insurance” hypothesis. Investigation of high-order interactions found that sex of the index child exerted a marginally significant moderating effect on the main effect association between replacement motivations and parity risk. Furthermore, analysis of interactions suggests a tendency for insurance effects to be greater in low mortality communities, for replacement effects to be stronger in high mortality community contexts, and for wealthier families to exhibit a weaker insurance response, but stronger replacement response, to childhood mortality compared to poorer families.

1. Introduction

Questions about the effect of childhood mortality on women's subsequent fertility have long motivated debate in demography and population policy. The original proponents of the demographic transition theory (DTT) argued that societies with high mortality have to compensate for it with commensurate levels of reproduction, and that the advent of socioeconomic development triggers improvements in child survival, and, as a corollary, unsustainable population growth that necessitates reproductive change.^{1,2} In such a manner fertility became, according to this theory, a part of the 'calculus of conscious choice'.³ There are numerous examples of research that have corroborated this hypothesis of the DTT, positing child mortality reduction as an important, possibly even necessary, precondition for a fertility decline.⁴⁻⁷

Challenges to the classical formulation that mortality decline is fundamental precursor to the fertility transition emerged from analyses of World Fertility Surveys in the 1980s. These contended that reproductive change was only loosely correlated with social and economic development, which tends to occur at different paces.⁸ From this arose the perspective that ideational rather than structural change, that is social interaction and the diffusion of new ideas, generated changes in fertility behaviors that tended to cut across socioeconomic strata in transitional societies.⁹ Findings of the Princeton European Fertility Project also countered the DTT hypothesis, giving historical examples in which the onset of the fertility reduction occurred before the start of a child mortality decline.¹⁰⁻¹³ This led to division among demographers: Whereas some saw the mortality-fertility relationship as essentially debunked, believers in it argued that the surprising findings may be spurious, due to studies that used too high levels of aggregation to detect relationships between individual exposures and behavioral changes.¹⁴

Evidence from sub-Saharan Africa (sSA) has not helped establish consistent evidence on either side of this debate. Whereas longitudinal studies indicate a significant fertility response to child loss in the way of shorter birth intervals, the general finding from analyses of repeat cross-sectional data is that in sSA mortality decline has either no effect or unclear effects on childbearing.¹⁵ Understanding these dynamics is critical in East Africa, particularly, where stalls in fertility decline have occurred contemporaneously with precipitous reduction in child mortality.¹⁶ Efforts to explain stalled fertility patterns point to the combination of lackluster economic development, weak family planning (FP) programs, longer birth intervals, and high levels of unmet need.¹⁷ Johnson-Hanks (2004, 2007) suggests that stagnant fertility declines in sSA are due to uncertainty associated with women's access to education, socio-economic resources, and economic opportunities.^{18,19} Moultrie et al. (2012) argue that women's reproductive behavior is less influenced by childbearing history and family size and is more closely related to household finances, employment contingencies, and relationship dynamics.²⁰ Studies examining the unexpected fertility response to child mortality have directed attention to morbidity effects of childhood diseases that persist in sSA despite overall mortality decline.^{21,22} Others have proposed that the fertility response to increased survival flattens as soon as differences in family size cease to appreciably change costs to the household.^{23,24} Investigating these relationships is important because of their policy implications: If reproductive behavior does not respond to changes in infant and child mortality, then a likely consequence of improved child survival may be higher growth rates. If, on the other hand, it does, then the success of FP programs may depend on the level of mortality.^{25,26} Wolpin (1998) echoes this sentiment, arguing that "fertility and mortality processes are the driving forces governing population changes, so an understanding of the way they are

linked is crucial for the design of policies that attempt to influence the course of population change”.²⁷

Answering these questions is particularly relevant for Tanzania. Even though childhood mortality declined in the country from 165 to 58 deaths per 1,000 live births between 1990 and 2015, helping it to achieve Millennium Development Goal (MDG) 4, this coincided with relatively stagnant fertility trends, which changed less appreciably, from 6.2 to 5.1 births per woman.²⁸⁻³⁰ Few studies in Tanzania have thoroughly examined the parental fertility response to child loss. Those that have are dated and depend on retrospective survey data.^{31,32} One such study, a multi-country analysis of data from Demographic and Health Surveys, examines the child mortality-fertility relationship in the broader context of variation in national economic performance, suggesting that child mortality is among the factors that mediates the more determinative relationship between socioeconomic progress and fertility, a direct vindication of the DTT.³³ Research has shown that the death of a child reduces the probability that parents will use contraception, thereby increasing the likelihood of additional pregnancies.³⁴ Qualitative research conducted on birth spacing and interval duration acknowledges that parents that expect their child will survive into maturity will be willing to risk having fewer children; however, these have emphasized appreciable variation in birth spacing patterns between regions of Tanzania and attribute this mainly to socio-economic conditions, dependency on subsistence agriculture, marriage patterns, the influence of traditional religion, maternal education, and gender relations.³⁵⁻³⁷ To date, there are, to our knowledge, no detailed, longitudinal examinations of the relationship between child mortality and childbearing in the context of persistently high fertility and MDG 4 achievement in Tanzania. The goal of this paper is to fill that gap. In doing so, this paper also addresses broader questions about the fertility

response to child mortality in other high fertility settings in sub-Saharan Africa where child mortality reduction has also occurred.

2. The fertility response to child mortality in sub-Saharan Africa

Empirical investigations of the interrelationship between childhood mortality and reproductive behavior have focused on two specific domains of the fertility response: an insurance or “hoarding” effect that arises from the tendency of mothers or couples to hasten childbearing in anticipation of mortality risks, and a replacement effect in which parents’ actual experience of child loss instigates change in their subsequent reproductive preferences and behavior. Replacement effects can occur due to biological factors related to the truncation of lactational amenorrhea after a breastfeeding infant dies, or volitionally, due to a conscious decision of parents to “replace” the child that they lost.³⁸⁻⁴⁰ Scholars have noted that a range of strategies from both domains can coexist within any given community or be adopted by a given family at different points in the reproductive course.⁴¹

Testing the insurance and replacement hypotheses involves clarifying the relationship between the death of a child and the mechanisms of fertility decision-making. In sub-Saharan Africa, studies based on aggregate data have had one important advantage: the potential for measuring the overall implications of child mortality reduction for fertility and population growth, whereas studies based on individual data alone can only measure accurately replacement effects.⁴² Aggregate studies, in general, lack the ability to finely examine the behavioral adaptation to child loss because they are either cross-sectional based on national aggregates using data from several African countries⁴³⁻⁴⁵ or from a specific country or region within a country.⁴⁶⁻⁴⁸ In addition, individual-level studies often use data from retrospective surveys and are thus beset by potential recall bias.

Aggregate-level studies in sub-Saharan African settings have generally reported a lack of evidence of any consistent relation between fertility changes and preceding child mortality trends, thus failing to support the hypotheses that insurance and/or replacement motivations, respectively, affect reproductive patterns at the societal level.¹⁵ Researchers have speculated that findings of no or unclear effects may stem from the fact that intermediate variables, which act more directly on this relationship, are obscuring the strong association at work at the individual-level.^{44,46,47} To the contrary, among the published research from sub-Saharan Africa that use individual-level data, e.g., World Fertility Surveys and Demographic and Health Surveys, there is the consistent finding that the death of a child leads to a shorter interval between that birth and the next. Although most of these analyses assess the proposition that replacement effects motivate childbearing⁴⁹⁻⁵⁴, some studies which used retrospective individual-level data have examined insurance effects by analyzing relationships between the number of children that have died for women of equal parity and family size and their subsequent fertility outcomes, finding that mothers' with repeated experience of child deaths were more likely to progress to higher parities than their counterparts that had not lost a child.^{55,56} Although research in sSA has generated insight on the effect of son preference⁵⁷, maternal education attainment⁵⁸, and socio-economic status⁵⁹ on fertility change, we are not aware of studies that assess whether these factors moderate the magnitude of associations between replacement and insurance effects of child loss and parity progression.

3. Study hypotheses

This study draws upon longitudinal data collected from health and demographic surveillance systems on the individual reproductive and child survival trajectories of mothers and children in three rural districts of Tanzania between 2000 and 2015. It treats exposure to insurance conditions, i.e., mothers' motivation to become pregnant that arises due to her ever having lost a child before

the onset of a given birth interval, and replacement conditions, i.e., intention to replace a child that died during a given birth interval, as independent factors that influence parity progression. We hypothesize first that although both conditions accelerate parity progression, replacement motivations will be more pronounced. Importantly, we also calculated the period child mortality rates in the cluster of communities in which individual mothers resided at the onset of a given birth interval and tested our second hypothesis, that the positive differences in the prevailing context of child mortality in individuals' environments, i.e., a dimension of insurance effects, hasten future childbearing, although less appreciably than replacement effects and insurance effects that arise from mothers' personal experience of child loss. Then we assess high order interactions between determinants that we conjecture moderate the fertility response to child mortality. Our third hypothesis is that that child sex preferences moderate women's tendency to hasten childbearing when placed under replacement, but not insurance conditions. Fourth and fifth, we hypothesize that the replacement response to child loss will be more pronounced among mothers with relatively high levels of education and from households that are relatively wealthy compared to counterparts with fewer years of schooling and that are from poorer households, respectively; while, conversely, that exposure to the insurance condition will especially accelerate parity progression among mothers that received less schooling and whose households are relatively poor. Finally, we examine whether the prevailing context of child deaths during the period surrounding the onset of a given birth interval moderates the fertility response to child loss under both insurance and replacement conditions. We hypothesize that mothers' residence in a cluster of communities in which the period child mortality rate is relatively low will diminish the effect of insurance motivations on childbearing compared to residence in settings where child mortality rates are

relatively high; however, that the child mortality context will exert no effect on the replacement response to child loss.

4. Methods

The data for this study come from the Ifakara and Rufiji Health and Demographic Surveillance Systems (HDSS) managed by the Ifakara Health Institute in Morogoro and Pwani regions of Tanzania, respectively.^{60,61} Morogoro is a landlocked region in the southern part of the country, and Pwani is on the country's central Indian Ocean coast. Prior to 2010, the sentinel areas observed by the Ifakara HDSS encompassed 36 communities in two districts separated by the Kilombero river, Kilombero and Ulanga, and 38 communities in Rufiji. In 2010, the HDSS extended surveillance operations to encompass an additional 32 communities in Kilombero and 5 additional communities in Rufiji. From 2010-2015, the Ifakara HDSS collectively included approximately 238,000 people in both Kilombero and Ulanga districts, and the Rufiji HDSS observed a sentinel population of approximately 124,000. The Ifakara and Rufiji populations are predominantly rural and rely on subsistence farming, with small peri-urban areas with businesspeople and traders.

Between 2000 and 2015, both HDSS collected data continuously in their respective surveillance sites throughout the year, with all households visited every four months. Information collected includes data compiled through periodic censuses undertaken every 1-2 years to enumerate old and new households and communities as they arose in the sentinel areas over time, and routine core updates, both of which included data on socio-demographic characteristics of household members and episodes of birth, death, and in- and out-migration episodes.

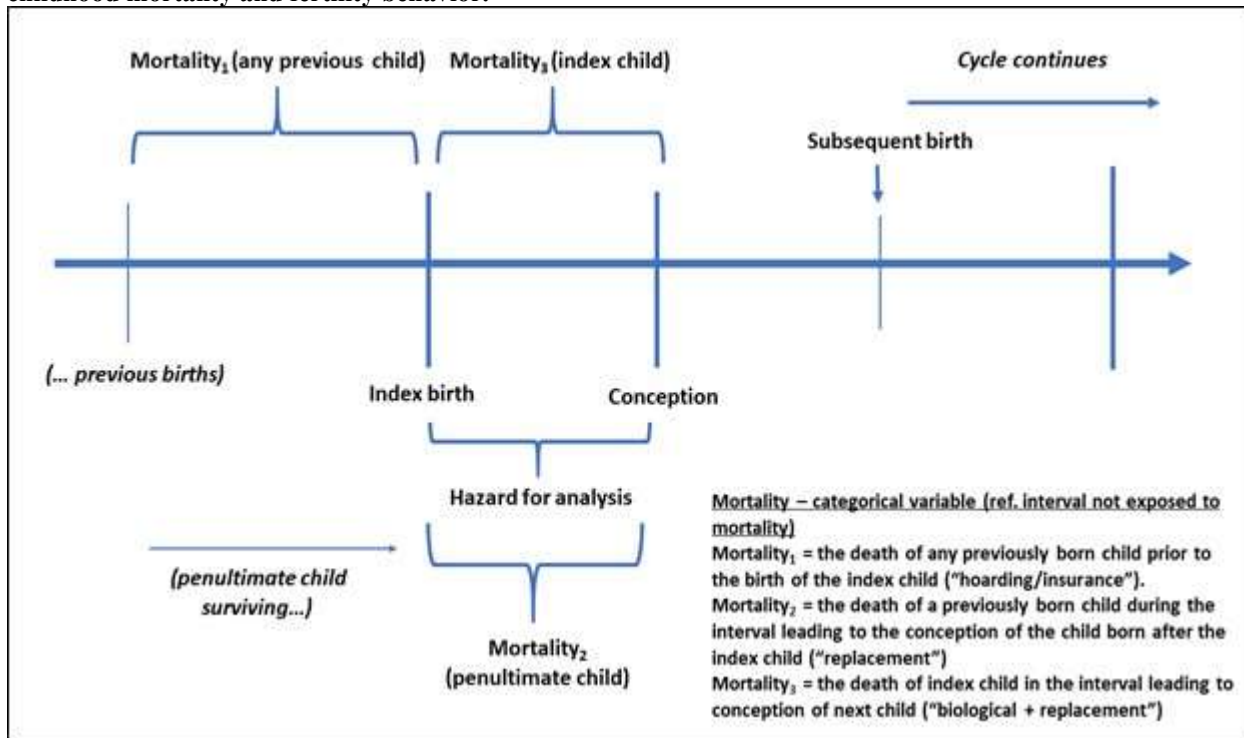
The study includes a subset of the data available from the period it reflects, 2000 to 2015. Included are reproductive histories of the cohort of women that were born between 1981-1984, who were residents of households in the respective HDSS areas and were 15-18 (n=15,291) at the start of the

longitudinal follow ups that obtained information on their fertility and childbearing, the incidence of child mortality and women's subsequent time until future pregnancy. The age range of women that was selected intends to minimize the risk of unobserved childbearing among members of the cohort. For the purposes of this article, all births were considered, whether singleton or multiple births, if they were of children of mothers registered by the HDSS (n=25,762). Of this number of children, 52 index children were removed from the final analysis because their deaths occurred during the pregnancy that closed their subsequent birth interval. Such deaths cannot be counted as exposure events since they occurred after the response. The number of children included in the final analysis was 25,710. The data were manipulated to ensure that person-months of observation of postpartum women accounted for repeat pregnancies within the same individual, mothers' possible physical exit and re-entry into the sentinel population and exit due to loss to follow up, death or right censoring. The latter occurred by December 31, 2015 to mothers whose postpartum trajectory surpassed the follow up period for which data were available and were thus living in the sentinel areas when the last core update took place. Linking the observations of mothers and children noted during different updates of the HDSS was made possible by the unique identifier assigned to both that remained with them until their permanent exit from the cohort due to death or administrative censoring. The duration of a subsequent birth interval was calculated by subsetting data sets by mothers' accumulated number of births over the 15 years and, within each subset, sorting each by birth order and mothers' unique identifier and inserting simple formulas for each set of births for each mother that calculated time between births and conception.

The analysis presented here is based on the interval between a given birth (the "index birth") and the next conception, employing the strategy proposed by Hill et al. (2001) in a paper on child

insurance and replacement effects in Zimbabwe and Senegal, and a methodology used by Hossain et al. (2007) in a similar study conducted on data from Bangladesh.^{62,63} See Figure 3.1.

Figure 3.1: The independent variable categorizing child death to assess alternative pathways between childhood mortality and fertility behavior.



This analysis defined the independent variable for the main effect categorically by classifying every observed birth interval in the data according to the type of child death to which it was exposed based on the hypothesis that it influences the hazard of future childbearing (see Figure 1): (1) No child mortality (m_0), (2) the death of a child prior to the birth of the index child, i.e., “insurance effect” (representing scenarios in which parents may “hoard” children as “insurance” against future child mortality because previous personal experiences of child loss that pre-date their most recent pregnancy condition them to expect that they may lose a child again) (m_1), (3) the death of a previously born child during the interval leading to the conception of the child born after the index child, i.e. “volitional replacement” (m_2) and (4) the death of the index child in the interval leading to the conception of the next child, “biological and volitional replacement” (m_3).

Although the incidence of biological replacement caused by the truncation of lactation and postpartum amenorrhea following the death of a child has been established, because breastfeeding data were not recorded in the HDSS, decomposing the effects of the variable we use to represent this factor (m_3) into behavioral and biological components is not possible in this analysis. The other variables used in the model are described in Table 1. Of note, approximately 12% of observations had missing data for household wealth status. We generated “missingness maps” to understand whether the absence of these values was more prevalent for different levels of our predictor or other salient covariates. We concluded that the missingness was distributed at random in the study population. To avoid removing potentially valuable data, we addressed this by imputing these values using multiple imputation through chained equations, found in the *mice* package in *R Studio*.

The pace of childhood mortality reduction in the period of our analysis, the “MDG era” of 2000-2015, during which Tanzania achieved MDG 4, was remarkable. Indeed, analysis of HDSS data on the entire study population in Ifakara and Rufiji for this period indicate that under-five mortality declined by 45 percent from 2000-15.⁶⁴ Nevertheless, fertility decline was much less appreciable. According to the same data source, between these years the period total fertility rate declined from 5.5 to 5.0 births per woman aged 15-49. See Table 3.1.

Table 3.1: Descriptive Statistics for the Study Population, 2000-2015

	2000	2015	Percentage
Under-five mortality rate (per 1,000 live births)	124	70	
Total fertility rate (per woman of reproductive age)	5.5	5.0	
Mean age of mothers at birth of index children	26	27	
Mean years of school of mothers at time of index child’s birth	4.62	6.18	
Percentage of birth intervals associated with:			
Prior child dies before birth of index child (m_1)			8.89%
Prior child died during the interval before the conception of the child born after the index child (m_2)			0.91%
Death of index child during the interval leading to the conception of the subsequent child (m_3)			6.53%

The persistence of relatively high fertility levels despite contemporaneous, and large, reductions in child mortality challenges the classical theory that posits a causal connection between mortality decline and subsequent fertility transition. The analysis that follows investigates the volitional context of the impact of child loss on the time until subsequent pregnancy during this period of rapid reduction in childhood mortality, first by testing the hypothesis of the behavioral response to child mortality, i.e., insurance and replacement (intentional and biological + intentional), and then by estimating the conditionality of insurance and replacement associations between child loss and fertility on factors that might modify these effects: child sex, maternal educational attainment, household wealth status, and the wider ‘child mortality context’ of fertility decision making. Table 3.2 describes all the variables that we used in our analysis (Models 1-5).

Table 3.2: Description and definition of the variables used in the study.

Variable	Levels	Description
Child-level covariates		
Birth order	First born, 2, 3, 4, 5 + (numeric)	The order in which the index child falls in terms of the order mother’s childbearing. Also, the parity of the mother at the time of index child’s birth.
Birth status	Singleton, multiple birth (binary/categorical)	Whether the index child was a single birth or multiple (i.e., twin, triplet).
Sex of index child	Male, female (binary/categorical)	Sex of index child
Birth year	2000-2015 (Numeric)	Year in which index birth intervals start.
Previous birth interval duration	Numeric (months)	The duration of time that had elapsed between the birth of the index child’s closest older sibling and the index child.
Mother/household level covariates		
Mother	Unique identifier	Unique identifier of every mother that links mothers across repeat observations during the follow up.
Maternal age	<20, 20-24, 25-29, 30-34, 35+ (categorical)	Mothers’ age at birth of index child.
Marital status	In union, not in a union (binary)	Mother was married or in union at birth of index child
Mother’s educational attainment	None, Primary level schooling only, secondary +.	Mothers’ highest level of education attained.

Household wealth	Highest to lowest quintile (1-5)	Household wealth quintiles based on household assets and basic amenities in the year of index child's birth.
Contextual-level covariates		
Demographic Surveillance System	Ifakara or Rufiji (binary/categorical)	The sentinel area under surveillance of one of the two DSS in which the index child was born.
Child mortality context	Numeric, continuous	Contextual-level child mortality rates in the cluster of communities in which each birth interval under analysis was nested from 2000-2015.

To estimate the child mortality context, which the DTT hypothesizes influences childbearing, period child mortality rates were calculated for the geographic area of communities in the sentinel areas in which households under surveillance were clustered for each two-year period of the cohort. This was to create a measure that represented the small-area child mortality rate that prevailed in the context in which each mother resided during the time that led to the start of each index birth interval in our analysis. The contours of the geographic clusters, five in total (three in Ifakara and two in Rufiji) were defined before the analysis for the purpose of managing HDSS operations. The values that emerged from constructing this variable were then assigned to every observation in the data, to denote variation in the child mortality context in which birth intervals were embedded between clusters and over time.

Analysis and visualization of descriptive relationships in the data and non-parametric Kaplan-Meier survival curves were used to explore the data. The latter was reviewed together with results from the Schoenfeld test of proportionality to see whether the Cox proportional hazards model was an appropriate statistical approach. Because multiple covariates marginally failed to satisfy the proportional hazards assumption, we elected to use a Weibull parametric hazard regression model (Equation 1) to estimate the effects of the exposure variables (m_1 , m_2 and m_3 , ref. m_0):

$$\ln(h(t/x, z) = p(t^{p-1}) + \sum_{j=1}^J \beta_j X_{ij} + \sum_{k=1}^K \gamma_k Z_{ik} + \sum_{j=1}^J \sum_{l=1}^3 \delta_l X_{ij} * mort_{il} + \sum_{k=1}^K \sum_{l=1}^3 \eta_l Z_{ij} * mort_{il} + \epsilon_{ij} + \epsilon_i \quad (1)$$

Weibull regression models applied in this analysis capture the underlying hazard of parity progression that is known to be small during postpartum amenorrhea but tends to accelerate with advancing time and decline as fertility increases. This intuitive appeal was corroborated by findings from assessing model fit in terms of its Akaike Criterion (AIC) value, which proved superior to others reported by the same model when fit with lognormal, loglogistic and exponential distributions (AIC = 95,857). In this model, t is the number of months from the onset of the index birth interval to the time of the subsequent pregnancy that closes the interval, the time to the end of the study period (i.e., right-censorship at the conclusion of the follow up), or the time to loss to follow up. The conditional hazard, $h(t/x, z)$ defines the risk of pregnancy at time t conditional on the set of maternal characteristics X and child characteristics Z . Weibull distribution parameter, p , defines the shape of the underlying hazard as time continues. The vector of variables represented by X_{ij} define the J background characteristics of individual mother i , i.e., mother age at birth of the index child, maternal education attainment, marital status, wealth status (of household), and HDSS sentinel area of residence; and the variable Z_{ik} comprises the birth order, year of birth, sex, birth status (singleton or multiple birth), previous birth interval of the index child, and the child mortality context of the index birth interval. The mortality variables represent the indicators of insurance and replacement effects. β and γ are vectors of parameters to be estimated by the maximum likelihood; δ and η are vectors of parameters for the terms representing interaction between the three mortality categories, respectively, and mother, index child and contextual characteristics, X and Z , also to be estimated through maximum likelihood. Interactions test the hypotheses that the different responses to child loss are conditional on child sex, maternal education, household

wealth, and the child mortality context in which birth intervals elapsed from 2000-2015. ε_{ij} and ε_i are error terms for the within- and between-mother effects, respectively. Frailty options were specified to reflect the assumed distribution of heterogeneity considering the likelihood of correlation in the data due to the clustering of repeat pregnancies within individual birth histories, and individual birth histories clustered within communities and districts. In the end, the base model included a frailty term for individual mother and accounted for geographic clustering by incorporating district of residence as a fixed effect. The Weibull model yields results interpretable as the “accelerated failure time” (AFT), i.e., the mean change in the characteristic subsequent birth interval duration associated with differences between those exposed to child mortality (m_1 , m_2 , and m_3) and no child mortality (m_0), i.e., the accelerated (or decelerated) survival time associated with each category of child loss. Subsequently, we fit a series of models which were built upon the base model to investigate the higher order interactions of child sex, maternal education, household wealth quintile, and child mortality context. Altogether, five models were fit for this analysis. All statistical analysis were conducted using the *survreg()* function in the *survival* package in *R Studio* version 4.0.3. We report conversions of the raw coefficients reported by the AFT models as the ratio of the hazards of childhood mortality for unit differences in the predictors. For this conversion, we used the *ConvertWeibull()* function from the *SurvRegCensCov* package.

5. Results

5.1 Sample Characteristics and Descriptive Results

The characteristics of the 25,762 births that took place in the study areas between 2000 and 2015 are in Table 3.3. Noticeable is the relatively high proportion of intervals that were associated with first births, 60% (n = 15,434). This reflects the young age structure of the female population that was subset for the analysis. Mortality rates were calculated for sub-categories of childhood

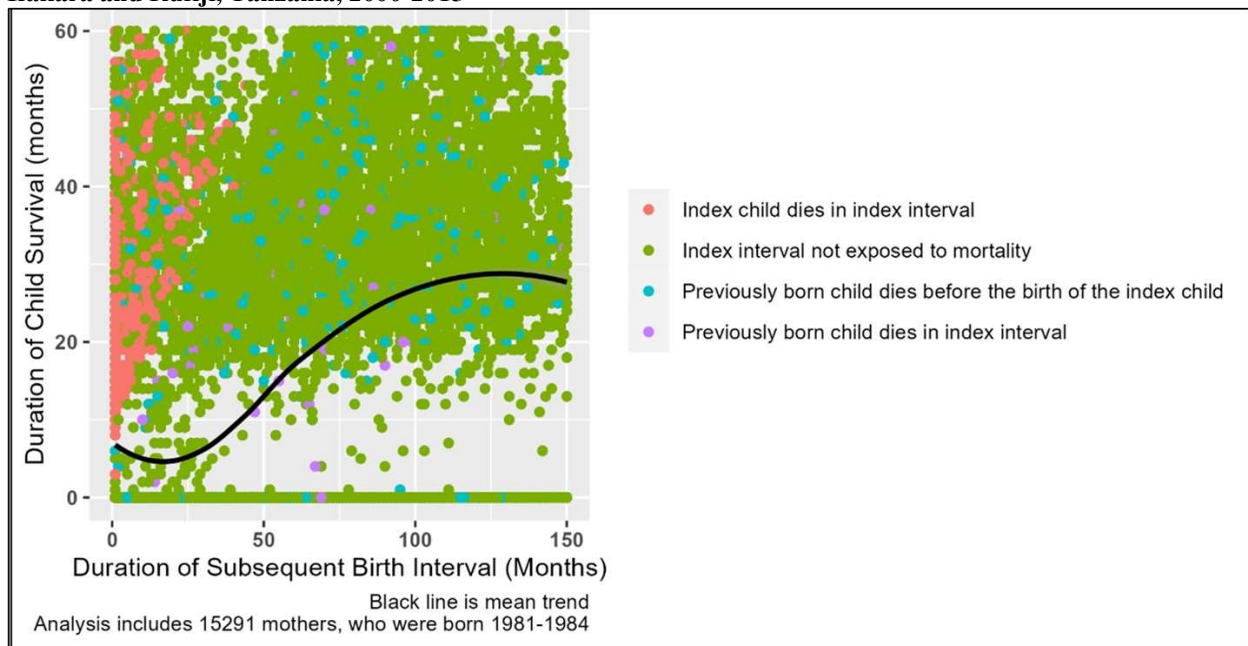
Table 3.3: Births and mortality rates (deaths per 1,000 person years) by covariates of children in the Ifakara and Rufiji HDSS, 2000-2015

	Births		Neonatal mortality (<1 month)		Postneonatal mortality (1-11 months)		Early childhood mortality (12-24 months)		Late childhood mortality (24-60 months)	
	N	%	Count	Rate	Count	Rate	Count	Rate	Count	Rate
Overall	25762*	100	694	26.9	600	29.5	261	14.9	187	5.5
Birth order										
1	15434	60.0	431	28.2	395	33.2	176	17.6	102	5.4
2	5818	22.6	167	28.0	143	30.2	58	13.9	44	5.1
3	2691	10.4	58	21.5	39	17.6	13	6.4	30	7.0
4	1209	4.7	26	21.3	15	14.7	10	11.1	9	5.4
5-8	610	2.4	12	44.8	8	16.9	4	10.9	2	4.2
Birth status										
Singleton	25653	98.5	626	24.7	584	29.8	260	15.0	187	5.5
Multiple	399	1.5	68	170.4	16	61.4	1	14.2	0	0
Child sex										
Male	12983	50.4	378	29.1	308	30.0	138	15.6	99	5.8
Female	12779	49.6	316	24.7	292	28.9	123	6.2	88	5.3
Maternal age										
<20	2593	10.0	79	30.5	99	50.5	34	21.3	20	6.1
20-24	8022	31.1	214	26.7	245	38.2	110	19.0	75	5.7
25-29	8856	34.3	212	23.9	171	23.5	84	12.3	73	5.2
30-34	6100	23.6	182	29.8	85	18.3	33	10.0	19	5.8
35+	191	0.7	7	36.6	0	0	0	0	0	0
Marital status										
In-union	19001	73.8	467	24.6	400	26.1	186	13.6	142	5.1
Unmarried	6761	26.2	227	33.6	200	40.0	75	19.6	45	7.2
Maternal education										
None	10931	42.4	265	24.2	216	25.2	111	15.6	90	7.1
Primary	3325	12.9	93	28.0	108	41.4	45	20.2	23	5.2
Secondary +	11506	44.7	73	6.3	276	30.1	105	12.9	74	4.4
Wealth status**										
First	4036	17.9	104	25.8	80	24.7	43	15.4	19	3.5
Second	4538	20.1	133	29.3	104	28.7	41	12.7	32	4.8
Third	4959	22.0	138	27.8	116	29.0	57	15.9	30	4.0
Fourth	4539	20.1	111	24.5	119	33.1	41	12.9	51	7.8
Fifth	4470	19.8	113	25.3	130	36.9	53	17.9	46	8.0
Birth year										
2000-2004	7417	28.8	211	28.4	263	45.5	109	21.9	68	6.3
2005-2009	7889	30.6	187	23.7	201	31.1	83	13.8	73	5.1
2010-2015	10449	40.6	296	28.3	136	16.7	69	10.7	46	4.3
DSS										
Ifakara	15623	60.6	496	31.7	384	31.8	152	15.0	80	4.2
Rufiji	10139	39.4	198	19.5	216	26.1	109	14.9	107	7.1

employing person-time data (counts per 1,000 person-years). Sub-categories are neonatal (< 1 month), postneonatal (1-11 months), early childhood (12-24 months) and late childhood (24-60 months). See Table 3.3. Note that Table 3.3 reflects the women in this analysis, not the entire population.

Figures 3.2 and 3.3 illustrate the relationship between parity progression and each level of our predictor, m_0 - m_3 . Figure 3.2 illustrates patterns in birth interval closure given changes in the survival duration of an index child. Not surprisingly, longer life spans of index children tend to correlate with longer periods until mothers start childbearing.

Figure 3.2: Relationship between subsequent birth interval length and child survival - 25,710 children born in Ifakara and Rufiji, Tanzania, 2000-2015



At a descriptive level it appears that child mortality during the subsequent birth interval and before conception tends to hasten parity progression most emphatically, which lends support to the hypothesis that replacement motives (volitional and biological) exert the greatest influence on the association of interest. Figure 3.3 also compares the distribution of durations until closure for all index birth intervals for the period of observation in both surveillance sites. What is

noteworthy, again, is the appreciably lower median interval duration associated with the condition representing both volitional and biological replacement. The volitional-only replacement condition that is represented by the deaths of previous born children during the index child’s subsequent birth interval has a median that approximates that of the insurance condition that is represented by the deaths of any previous born children prior to the birth their index sibling. The average subsequent birth intervals associated with the levels of the predictor, m_0 , m_1 , m_2 and m_3 , were 37.3, 35.3, 34.75 and 28.81 months, respectively.

Figure 3.3: Duration of subsequent birth intervals by mortality exposure type – 25,710 children born in Ifakara and Rufiji, Tanzania, 2000-2015

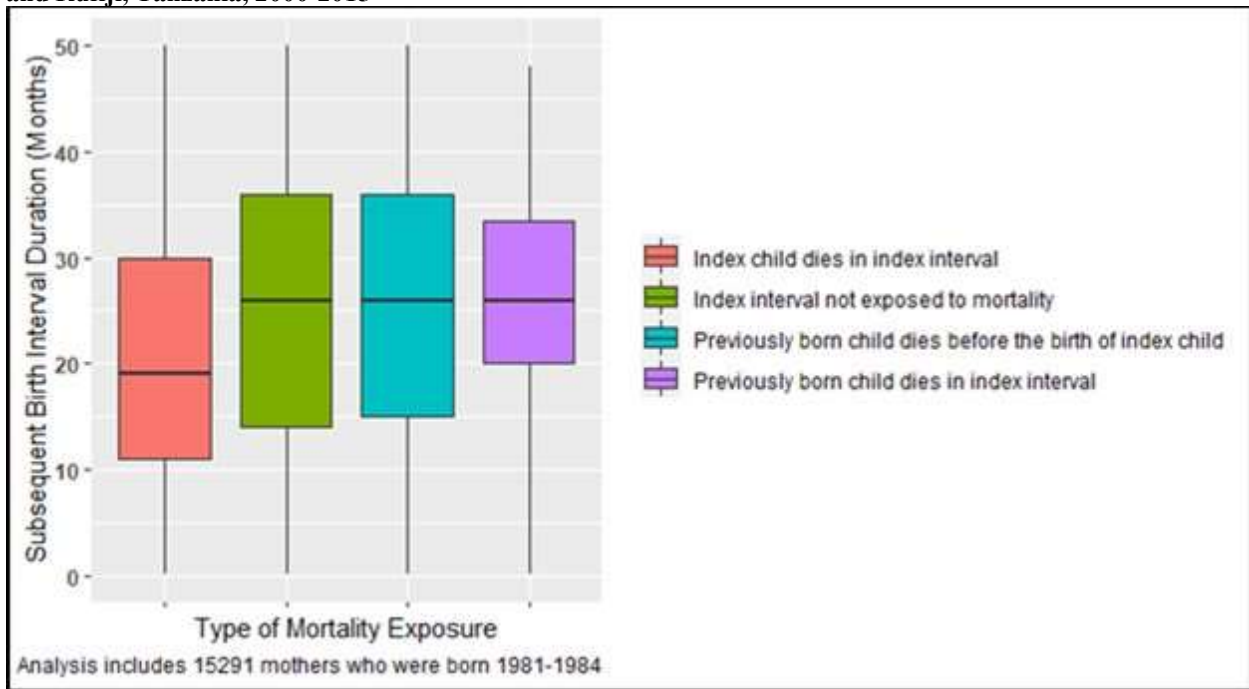
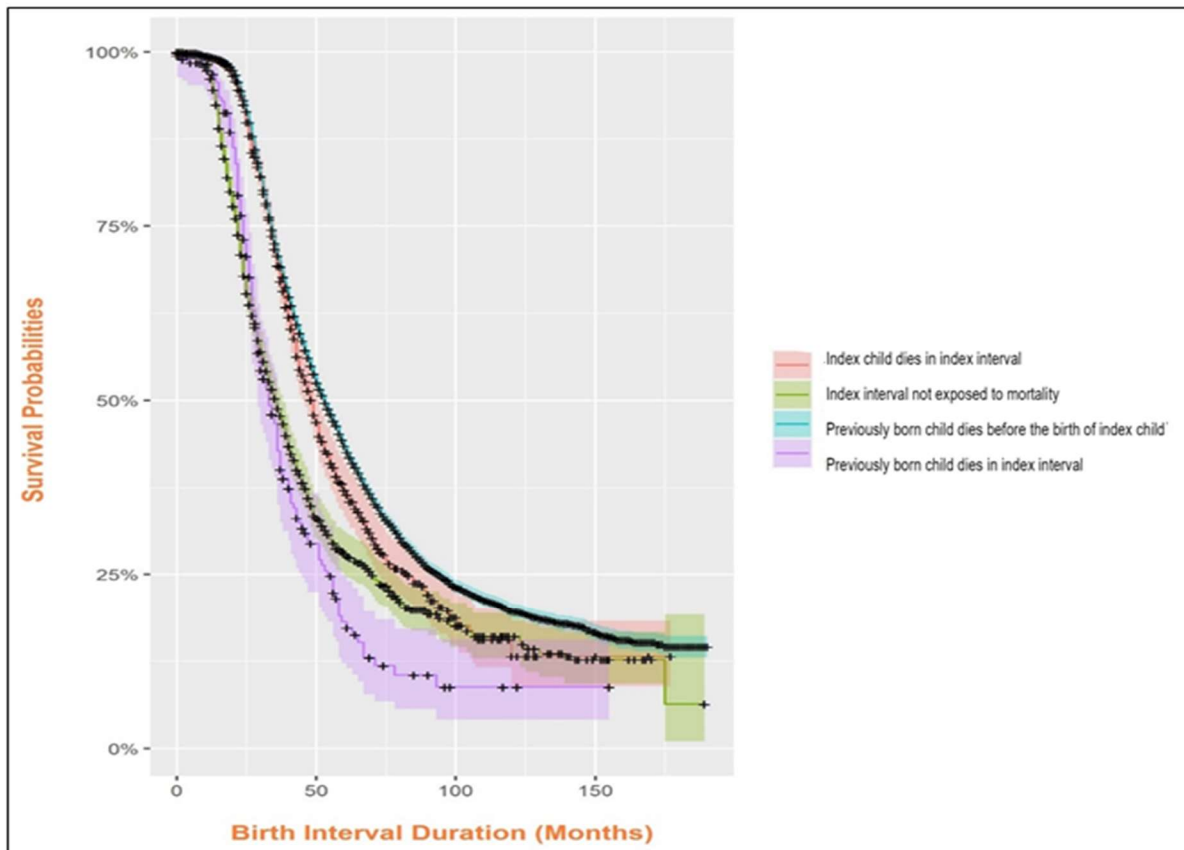


Figure 3.4 illustrates the Kaplan-Meier survival functions that estimate probabilities of parity progression of mothers associated with their exposure to the different child outcome classifications. From it, we observe that “survival probabilities” tend to be highest (i.e., a tendency for slower parity progression) among birth intervals that were not exposed to any type of mortality (the m_0 condition). However, survival probabilities tend to be the lowest among birth intervals

exposed to m_2 and m_3 conditions, which suggests that the pace of parity progression may be higher for births subjected to replacement motivations compared to those subjected to insurance motivations or no mortality at all.

Figure 3.4: Kaplan-Meier Survival Functions of Risk of Birth Interval Closure Associated with Child Survival



5.2 Multivariate Analysis of the Fertility Response to Childhood Mortality

In Table 3.4, hazard ratios for the parameter of Model 1 are presented for the main effects of the child mortality indicators on the hazard of closing the index-birth interval (Model 1), for the interaction of mortality indicators with sex of the index child (Model 2), maternal education attainment, (Model 3), and household wealth status (Model 4). Finally, Model 5 assesses if cluster-level child mortality rates in the year prior and birth year of index children moderates the association between child death and parents' subsequent fertility behavior.

Table 3.4: Hazards for the Main and Interaction Effect of Child Mortality (m_1, m_2, m_3 , ref: m_0) on Parity Risk Adjusting for Covariates.

Covariate (main effects)	Relative Risk				
	Model 1	Model 2	Model 3	Model 4	Model 5
Mortality (ref: no mortality)					
m_1 (Previous child dies before index birth)	1.46***	1.38**	1.51***	1.83**	1.86***
m_2 (Previous child dies in index interval)	3.89***	4.52***	4.19***	4.95***	2.91***
m_3 (Index child dies in index interval)	4.69***	4.37***	4.26***	4.15***	3.96***
Child sex (ref: female)					
Male	0.95*	0.94**	0.95**	0.95*	0.95*
Household wealth status (ref. fifth quantile, i.e., poorest)					
First	0.36***	0.36***	0.36***	0.35***	0.36***
Second	0.50***	0.50***	0.50***	0.51***	0.50***
Third	0.65***	0.65***	0.65***	0.65***	0.65***
Fourth	0.86***	0.86***	0.86***	0.87*	0.86***
Mother age at birth of index child (ref: <20)					
20-24 years	0.73***	0.73***	0.73***	0.73***	0.73***
25-29 years	0.76***	0.76***	0.76***	0.76***	0.76***
30+ years	0.98	0.98	0.98	0.98	0.98
Mother educational attainment (ref: none)					
Primary	0.78***	0.77***	0.78***	0.78***	0.77***
Secondary	0.63***	0.63***	0.62***	0.63***	0.63***
Marital status (ref: married)					
Not married	0.40***	0.39***	0.40***	0.40***	0.40***
Multiple birth status (ref. no)					
	1.18	1.18	1.19	1.15	1.21
Birth order	0.36***	0.36***	0.36***	0.36***	0.36***
Birth year	1.16***	1.16***	1.16***	1.16***	1.16***
Child mortality context	1.07***	1.07***	1.07***	1.08***	1.08***
Previous birth interval duration (month)	1.00***	1.00***	1.00***	1.00***	1.01***
District (ref: Kilombero)					
Rufiji	2.54***	2.54***	2.54***	2.55***	1.78***
Interaction terms					
Child loss (m_1, m_2, m_3)*child sex					
m_1 (Previous child dies before the birth of index child) *child sex(male)		1.10			
m_2 (Previously child dies in index interval)* child sex(male)		0.73 [†]			
m_3 (Index child dies in index interval)* child sex(male)		1.15 [†]			

	Relative Risk Ratios				
	Model 1	Model 2	Model 3	Model 4	Model 5
Child loss (m_1, m_2, m_3)*maternal education					
m_1 (Previous child dies before the birth of index child)*Mother Education (primary)			1.05		
m_2 (Previous child dies in index interval)* Mother Education (primary)			0.57*		
m_3 (Index child dies in index interval) * Mother Education (primary)			0.96		
m_1 (Previous child dies before the birth of index child)*Mother Education (secondary)			0.92		
m_2 (Previously child dies in index interval)* Mother Education (secondary)			0.96		
m_3 (Index child dies in index interval)* Mother Education (secondary)			1.26**		
Child loss (m_1, m_2, m_3)*household wealth status					
m_1 (Previous child dies before the birth of index child)*Quintile1				0.74*	
m_2 (Previously child dies in index interval)* Quintile 1				1.43	
m_3 (Index child dies in index interval)*Quintile1				1.55**	
m_1 (Previous child dies before the birth of index child)*Quintile2				0.67*	
m_2 (Previously child dies in index interval)* Quintile 2				0.74	
m_3 (Index child dies in index interval)*Quintile2				0.97	
m_1 (Previous child dies before the birth of index child)*Quintile3				0.73*	
m_2 (Previously child dies in index interval)* Quintile 3				0.59 [†]	
m_3 (Index child dies in index interval)*Quintile3				1.41**	
m_1 (Previous child dies before the birth of index child)*Quintile4				0.85	
m_2 (Previously child dies in index interval)*Quintile4				0.77	
m_3 (Index child dies in index interval)*Quintile4				0.94	
Child loss (m_1, m_2, m_3)*child mortality context					
m_1 (Previous child dies before the birth of index child) *child mortality context					0.80**
m_2 (Previously child dies in index interval) *child mortality context					1.11
m_3 (Index child dies in index interval) *child mortality context					1.08***

***p<0.001, **p<0.01, *p<0.05, †= 0.05<p<0.10

Findings yielded by Model 1 indicate that as birth order increases, the risks of birth-interval closure decline monotonically. Similarly, this risk declines monotonically for level increases in household wealth status classification. Parity progression tends to be slower for intervals for which the index child is male. After adjustment for all other covariates, women's risk of birth interval closure tends to occur more precipitously among women with no education compared to peers with primary and secondary level schooling. In addition, Model 1 indicates that the risk of pregnancy tends to increase over time, which is likely a cohort effect that arises from the tendency to accelerate childbearing after marriage. Risks of parity progression are notably higher in Rufiji compared with the population resident in the sentinel areas of the Ifakara HDSS.

Results for mortality hazard ratios reported by Model 1 are consistent with the hypotheses of insurance and replacement effects on childbearing. In Table 3.4, the main effects it produced for all three mortality predictors are positive and statistically significant, especially indicators m_2 and m_3 , whose hazards were approximately four-fold greater than that of the reference condition, indicating a pronounced replacement effect that reflects both behavioral and biological responses to child loss (HR: 3.89, 95% CI: 3.22-4.69; HR: 4.69, 95% CI: 4.33-5.08). The main effect analysis reports a substantially smaller, but statistically significant, hazards ratio for parity progression associated with women's exposure to the loss of a previous born child during prior to the onset of the index interval (HR: 1.46, 95% CI: 1.32-1.61), which implies a smaller, but meaningful insurance effect. With regards to the main effect of the child mortality context in which birth histories are nested, Model 1 reports that for unit-increases in levels of mortality in women's surrounding environs during the time of conception and the onset of each birth interval are associated with 1.07 times higher hazards of birth interval closure (HR: 1.07, 95% CI: 1.02-1.21). Taken as a set of main effects, the hazard ratios reported from the first model in Table 3.4

demonstrate a prominent and consistent relationship between childhood mortality and fertility, supporting the hypothesis that some combination of insurance and replacement effects are operative.

The interaction effects reported in the second section of Table 2 for Models 2-5 help understand the role of socio-economic factors and the child mortality context on the main effect relationships reported by Model 1. Model 2 yielded two marginally significant ‘hazard ratio ratios’ implying that sex preferences may affect fertility behaviors after child loss. Whereas no such effect was detected with respect to insurance motivations, m_1 , Model 2 reports that the tendency for volitional replacement (occurring when a child elder to the index child dies) is diminished if the index child is a son (HRR=0.73, 95% CI: 0.51-1.05, $p=0.072$). Conversely, this analysis suggests that the combined behavioral and biological replacement condition, which occurs after the loss of the index child, is stronger if the index child was a son (HRR=1.15, 95% CI: 0.98-1.34, $p=0.093$). With regards to the moderating effect of maternal education attainment, contrary to expectations, Model 3 reports that the behavioral replacement effect (m_2) is less pronounced among women with some primary education, compared to their counterparts that had no schooling (HRR = 0.57, 95% CI: 0.32-0.99). In addition, and in alignment with our hypothesis, Model 3 results suggest that the combined replacement effect tends to be stronger among women with some secondary education compared women with no schooling at all (HRR=1.26, 95% CI: 1.06-1.49).

An intriguing finding regarding the insurance effect is reported by Model 4 which assess for effect modification of household wealth status on the main effect associations. The data reported that the risk of conception given exposure to insurance condition was 0.74 times lower among women resident in households in the highest wealth quintile compared to women in the lowest (HRR=0.74, 95% CI: 0.53-1.00). Conversely, the analysis reveals that women’s risk of pregnancy immediately

after death of a child (compared with that of women that experienced no child loss), i.e., the combined replacement condition, m_3 , was 1.55 times greater among women in the highest wealth quintile compared with women with the same exposure in the lowest wealth quintile (HRR = 1.55, 95% CI: 1.20-1.99). As Table 4 narrates, these patterns play out consistently comparing birth interval durations of women in the second and third household wealth categories to women in the poorest.

The final model in the analysis, Model 5, reported significant findings with respect to the interaction effect of child mortality context on the main effect relationship on insurance motivation (m_1) on childbearing. It reports in settings whose contextual child mortality rates differ by one-unit, the risk of parity progression given mothers' loss of any previous child before the birth of the index child is 0.80 times lower in the higher mortality settings than it is in lower mortality settings (HRR = 0.80, 95% CI: 0.74-0.86, $p < 0.001$). Although more research is required to fully understand this finding, that insurance motivations are weaker in relatively high child mortality contexts does not conform to the expectation that insurance behavior is constant as demographic change progresses or is pronounced in settings and at times when mortality is relatively high. With respect to the effect of child mortality context on the parity risks associated with m_2 and m_3 conditions, Model 5 results indicate that the combined replacement condition effects on parity progression are relatively pronounced among birth interval trajectories nested in relatively high child mortality settings compared to the same relationship when situated in relatively low child mortality settings (HRR = 1.08, 95% CI: 1.02-1.15).

6. Discussion

This analysis uses longitudinal data of exceptional quality to examine the effects of childhood mortality on women's fertility behavior. It does so, first, by determining whether the effect of

directly experiencing child loss affects the individual childbearing trajectories of mothers differentially if it operates through “insurance” mechanisms, i.e., the motivation to pursue family formation shaped by fears or the expectation that child loss may ever occur again, or “replacement” tendencies, i.e., the impetus to have another child to replace one that had just been lost. Secondly, the analysis assessed whether these main effect relationships were moderated by social determinants and the wider context of child mortality.

As expected, the main effects demonstrate a pronounced and consistent relationship between child mortality and fertility and are consistent with the broad tenet of the DTT, which hypothesizes that both insurance and replacement effects shape reproductive behavior and decision making during the demographic transition. In this case, the latter exerted a much larger influence over parity progression even in the absence of its biological component. In addition, we also observed that residing in small areas with relatively high child mortality levels was associated with hastened parity progression. This suggests that among women directly exposed to the same mortality conditions, their reaction to the overarching mortality context still conditions fertility behavior, albeit to a lesser extent than does their response to personal experiences with child loss.

The first interaction model, which tested whether the sex of the index child moderated the influence of child loss on fertility behavior gleaned notable insight on child replacement effects, even though the findings were marginally significant only. Compared to mothers whose most recent born is a daughter, those whose most recent born was son, when faced with the loss of an elder child, are, generally, slower to replace the child through future fertility. However, within the same comparison, if mothers are instead faced with losing their most recent born (i.e., the index child), mothers who lose a son tend to replace it more rapidly. These findings are consistent with those

from similar studies which report that couples with sons are less likely than those with daughters to continue childbearing, and that death of male children enhances replacement.^{63,65,66}

Comparison of m_1 and m_3 main effects between mothers from wealthier households to those from the poorest give evidence that the fertility response to child loss, no matter women's underlying motivational disposition, varies according to their socio-economic position. Specifically, Model 4 indicates that the effects of ever losing a child on women's reproductive course are, relatively speaking, less enduring for women from the highest wealth quintile. In other words, the risk of fertility as insurance against childhood mortality given a prior experience that raises expectations of that prospect is significantly more prominent among mothers with the least resources. Conversely, with respect to replacement tendencies in the immediate aftermath of losing a child, the analysis found that this impetus was significantly more intense among women from wealthiest households relative to those from the poorest. The former finding suggests that heightened and more lingering effects of insurance motives are linked to household resource constraints, which, in the context of rural Tanzania, imply such conditions as higher risks of childhood morbidity, agrarian dependencies that encourage child labor participation and discourage school attendance, subjection to traditional fertility norms and pressures, lower access to maternal education and economic opportunities, and poor nutrition and sanitation.³⁷ This is consistent with results reported elsewhere in the literature. Based on a similar investigation conducted decades ago in Thailand, Hashimoto and Hongladarom (1981) report findings that underscore the importance of insurance-motivated fertility response to child mortality in poor settings where health, nutritional and sanitary conditions are inadequate.⁶⁷ Similarly, in their analysis of Demographic and Health Survey data in Ghana, Novignon et al. (2019) demonstrate that family size preferences of women when faced with child mortality risk were highest among women with the least economic bargaining power.⁶⁸

As the results indicate, in our study population there is a tendency for more pronounced replacement effects among women from wealthier households compared to those from the poorest. Previous studies attribute this to the practice of fertility regulation, including modern contraceptive use, which are generally more prevalent among wealthier families compared to peers from lower socioeconomic positions.⁶⁹ Other studies which address the demographic transition from the perspective of evolutionary theory interpret positive associations between replacement fertility and socio-economic status more broadly in terms of an enabling process through which wealth – e.g., availability of disposable income and valuable assets – helps women and couples to adapt to changes life circumstances, including reallocating resources for parental investment in rearing a new child in the early aftermath of losing a previously born one.⁷⁰ This, as a corollary, may tend to enhance replacement fertility behaviors among wealthy households to a level which exceeds that of poorer households that have also experienced child loss.

The fifth model tested a core tenet of the DTT by assessing whether the level of childhood mortality in women's and couples' environment – i.e., throughout their immediate and surrounding communities – moderated their pace of pursuing childbearing given the experience of child loss. These results are also illuminating. While the main effect findings generally affirm DTT assumptions that child mortality accelerates parity progression, the interactions of child mortality context on the m_1 condition challenges a corollary of that expectation, which is that insurance behaviors will decline as a function of improvements in child survival that occur in the wider population and allay parental fears and perceptions of risk.⁷¹ To the contrary, Model 5 reveals a tendency in this study population for the effect of insurance conditions to be significantly stronger in clusters in which child mortality was lowest compared with the same effect in settings where it is highest. This implies that experiencing child loss imparts perceptions of vulnerability and risk

that are impervious to improvements in the survival context of family formation that make the recurrence of child loss decreasingly probable. This interpretation corroborates those reported from several earlier pieces of research on the demographic transition in sSA which depict family building strategies that are determined in the face of adversity, contingency and, above all, the “absence of regularity”.⁷² Frank (1987) reflects on the endurance of high childbearing desires of women in sSA and attributes it, in part, to the sensitivity of family size ideation to women’s and couples’ earlier personal experiences of child death and survival.⁷³ Studies that sought to explain why perceptions of vulnerability endure and have focused on the endemicity of infectious disease that continues to exist, particularly in rural settings, and the effect of childhood morbidity on long term health, cognitive and physical development of children, which, it follows, lowers the marginal return on parental investment on offspring and helps to perpetuate preferences for large families.²¹

Alternatively, the persistence of insurance effects amidst wider improvements in child survival in Tanzania suggests that fertility intentions that are conditioned by child loss, rather than diminish in an enabling environment, come to affect fertility behavior through mechanisms not emphasized by proponents of the DTT. An alternative explanation for this applies the notion of “delayed replacement”, which posits that women bear an extra child or children at the end of a reproductive span in order to compensate for child loss that could have occurred near the beginning.⁴¹ Scholars of the African fertility transition have made similar observations, attributing the progressive lengthening of birth intervals seen throughout the region over years to decisions of women to postpone childbearing in response to other sources of uncertainty, which in the case of this study may compound anxieties about child mortality, including concerns with respect to relationship stability, finances and money, employment, and housing.^{18–20,74,75} Whereas their underlying drivers and mechanisms remain an important subject for future research, this analysis suggests that robust

insurance motivations and the resilience of large family size preferences are factors that may help explain the relatively slow pace of fertility change in Tanzania.

The analysis reported by Model 5 brings into focus important insights on the conditionality of replacement behaviors after child loss on the child mortality context in which individual childbearing trajectories are nested. Unlike other studies that have estimated effects of replacement that are accentuated as demographic transitions progress toward low child mortality regimes^{63,67}, this study, which assesses for effect modification not by the stage of a population change trajectory, but by actual measures of variation in areal child mortality rates between years and geographic clusters, finds that replacement effects are more pronounced in settings and times in which levels of child death are relatively high. Whereas additional and deeper research into this relationship is required to fully understand its drivers and mechanisms, reflecting on this observation is apropos and can help shape future studies. Previous investigations of child replacement and the demographic transition have attributed inflections of behavioral replacement patterns at points when child mortality is decreased to the co-occurrence of child mortality reduction with fertility decline and higher levels of modern contraceptive use, i.e., increases in women's and couples' practice of controlling fertility.^{41,76,77} In contrast, our results unfolded in rural Tanzania in which, as child mortality declined, fertility reduction was slow and increases in contraceptive use unappreciable.⁷⁸ It follows that forthcoming studies ought to investigate fertility regulation in the context of low or declining child mortality, in particular the conditions and factors that shape whether or not the former is elevated as a result of the latter, and how this plays out at the individual level given direct experiences with child loss. Our observation that family formation practices did not adapt as expected in the advent of trends believed to help precipitate reproductive change is not new to African demography. Bledsoe et al. (1994) made similar observations of rural Gambian

communities' response to the introduction of western contraceptive methods. Contrary to expectations of a transition from natural to modern fertility regimes, their study population was trying through highly intentional actions to maintain natural fertility birth intervals by blending short-term, parity-specific use of modern contraception with more longstanding birth spacing strategies.⁷⁹ This illustration of the robustness of traditional fertility intentions to modernizing influences seems applicable to the context of rural Tanzania decades later.

It is important also to acknowledge that this study has limitations. First, while this analysis aimed at maximizing the utility of demographic data to operationalize and examine theoretical perspectives that underlie the DTT, doing so invites risk of measurement bias that is inherent in using quantitative indicators to represent complex, emotional and deeply personal responses to traumatic events. Whereas the demographic data used to construct the exposure in this analysis can affirm that women experienced child loss at particular times in their life course, there is risk in assuming that this instigated a specific psychological predisposition toward the prospect of fertility. Doing so presents a foremost limitation of this study. Second, as Pebley et al. (1979) remarked in their examination of the effects child mortality on reproductive volitions in Guatemala, child mortality experiences affecting a woman's fertility decisions are not only those of her own childbearing years, but also those of her mother's childbearing years, and that these influences are manifested at difference life stages, which suggests that mortality declines must occur over two generations to make a significant impact on women's desire for additional children.⁸⁰ Casterline (2017) makes a similar case, asserting that cohorts entering the reproductive years will increasingly feel assurance that survival through childhood is highly likely and frame their reproductive strategies accordingly, and that confidence about child survival could well have a transformative effect on fertility demand in Africa during the next decade via cohort succession.⁸¹

Although this analysis leverages high quality longitudinal data that cover a long period of appreciable mortality reduction, more years of observation may be required to observe the final effects of child survival improvements on fertility behavior in Tanzania. In addition, the sole data source for this analysis, the Ifakara and Rufiji HDSS, only tracked a limited range of demographic indicators, even though a richer evaluation of mortality-fertility dynamics requires more data on, for example, the proximate determinants of fertility; attitudes and behaviors related to contraception; socio-cultural and economic determinants of fertility and childrearing practices. Thus, future research ought to embed more elaborate methods of survey and qualitative research to enable a more complete examination of the fertility response to child mortality in Tanzania and similar settings.

7. Conclusion

In conclusion, our analysis demonstrates that the experience of child loss in the context of rural Tanzania during the first 15 years of the 21st century are associated with accelerations in parity progression, and that the demographic role of replacement effects are particularly pronounced relative to the effect of insurance motivations, which are smaller but also statistically significant. As noted above, future research should adopt a deeper focus on exploring the effect modification on these main effect associations that our Models 4-5 detected with respect to the differential effects of child loss on future fertility comparing women that differ with respect to socio-economic positions and child mortality contexts. Similar studies in rural Tanzania will be relevant in the years to come to assess whether the effects of child survival improvements, in turn, beget fertility change in subsequent cohorts of women of reproductive age.

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Chapter 5:

Conclusions

This dissertation contains three manuscripts that reflect upon the causes and consequences of child mortality in the context of health systems strengthening and demographic change in rural Tanzania. The sentinel areas of the Ifakara and Rufiji HDSS between 2000 and 2015 was the setting for this exploration. This was appropriate given the concentration of multi-level primary health care interventions there during that time and the availability of data from health facility assessments, household surveys, qualitative health systems appraisals and demographic surveillance, which offer complementary insights into the determinants, mechanisms and impacts of child mortality. The motivations for pursuing this research were to advance our understanding of the programmatic drivers and processes that shape child mortality risk, and the effects of childhood mortality on the demographic transition in rural sub-Saharan Africa. Findings from this dissertation can inform strategies to accelerate child mortality reduction and maximize the impact of improved child survival on longer term health development in Tanzania and similar settings.

Summary

In Aim 1, we reduced cross-sectional indicators of maternal, newborn and child health (MNCH) service availability and readiness at primary health care (PHC) facilities into catchment-level scales of implementation strength. We found that variation in the effective coverage of MNCH was mostly explained by between-facility differences in terms of general readiness (staffing, infrastructure and management inputs and practices) and the provision of preventive health care for mothers and children, followed by the provision of curative care for sick children and care for women during labor and delivery. We linked the implementation strength scales to longitudinal data on the survival of children born in facilities' catchment areas and performed analysis which

reported that children nested in contexts of relatively high implementation strength tended to have a lower risk of dying during childhood. These effects were most appreciable with respect to the strength of sick child care service delivery, followed by the strength of facilities' general readiness to provide MNCH and their delivery of preventive maternal and child health care interventions. The implications of these results for health systems researchers and planners in Tanzania are two-fold. First, establishing an environment of strong PHC implementation is associated with higher chances of survival during childhood. To sustain child mortality reduction or instigate that process in settings where child mortality remains high, health systems should recognize the need for evidence-based interventions that reflect the spectrum of services along the MNCH continuum of care. Our results also underscored the importance of general facility readiness to effective coverage and child survival. As a corollary, health system strengthening strategies should balance their emphasis on evidence-based interventions and the underlying needs for appropriate staffing, management inputs and processes and infrastructure at facilities. The second implication relates to our ability to validate our implementation strength scales by demonstrating their positive association with the survival of children, irrespective of where and if they actually received health care. This suggests that routine facility data can be utilized to measure health systems strengthening outputs and draw inferences about the health impacts of implementation. Health system researchers and planners should consider our methodology for measuring implementation and ways in which it can be incorporated into routine monitoring and evaluation of health systems' performance.

In Aim 2, we triangulated analyses of quantitative household survey data with qualitative analysis of process evaluation data from a cluster-randomized pragmatic trial of the impact of community health workers (CHW) on MNCH and child survival. We reported on the effects of CHW on

MNCH service utilization behaviors, childhood morbidity and access to timely and appropriate sick child care. The CHW program reduced incidence of childhood diarrheal, respiratory, and febrile illness and improved access to timely and appropriate sick childcare; however, there was no effect on MNCH service utilization behaviors. The positive outcomes were achieved through implementation mechanisms that triggered high levels of acceptability of CHW among community-members, and motivation and confidence of the CHW. Implementation factors that generated these effects related to the engagement of communities in program startup; the training, remuneration and configuration of supervisory support to the CHW from the local health system and communities. The lack of program effects on MNCH service utilization were attributed to the fragile health systems context, which was replete with lapses in the availability of needed care at facilities and timely resupplying of working materials to CHW, and the cost and complexity of the intervention vis-à-vis local health systems absorptive capacity for strategic change. These findings are relevant to CHW interventions for several reasons. First, despite an adverse systems context, community-based health programs can improve child health if program implementers engage communities in cocreating interventions, imparting the belief that interventions originated in communities and are adapted to address needs and realities that are specific to community contexts. This underscores the importance of integrating intervention components, such as CHW recruitment, selection, training, and deployment, with existing community structures and processes. Second, our analysis underscored how CHWs' effectiveness was shaped by the program's multi-faceted approach to enhancing their abilities and motivation to serve their communities and health systems. Crucially, strategies to optimize CHW effectiveness should blend intervention features that build CHW technical competency, promote their meaningful and constructive relationships with supervisors and support actors from the community, avail CHW

with compensation, and give CHW professional affiliations and opportunities for growth within the health system. Third, to maximize the impact of CHW programs, implementers need to establish an enabling environment for CHW productivity and success. This should start with acknowledgment of how health systems and communities need to adapt to become able to integrate and deploy CHW effectively. In addition, programs should adopt a broader focus on strengthening and aligning health system and community capacities to perform core functions and routinize the strategic changes associated with extending the PHC system to the community-level.

In Aim 3, we examined the impact of child mortality on fertility. Doing so is important since mortality and fertility processes are the driving forces governing population changes. Therefore, knowledge about the way in which mortality changes are linked to fertility is crucial for the design of policies and programs that influence the course of population change. This guided our use of demographic surveillance data on childbearing and survival that were compiled by the Ifakara and Rufiji Health and Demographic Surveillance Systems from 2000-15. We coded index birth intervals to instantiate theoretical propositions of the effect of child loss on parity progression. The first was a “replacement condition” in which childbearing is motivated by a desire to replace an index child that died; and the second, “insurance conditions” which represent the “hoarding” instincts of parents whose childbearing is motivated by fears that arise from ever having lost a child before or residing in clusters where child mortality is prevalent. We conducted generalized hazard regression analyses, which reported that women’s exposure to replacement and, to a lesser degree, insurance motivations were associated with accelerated time to conception during future birth intervals. Investigation of high-order interactions suggested a tendency for insurance effects to be greater in low mortality communities, for replacement effects to be stronger in high mortality community contexts, and for wealthier families to exhibit a weaker insurance response, but

stronger replacement response, to childhood mortality compared to poorer families. The main effect associations, generally, conform to expectations raised by decades of demographic theory. However, the results also illuminate the robustness of traditional fertility intentions despite broad-based improvements in child survival in the study population. This challenges conventional demographic theory which purports that insurance motivations attenuate, and that replacement effects accentuate, in societies as child mortality becomes less prevalent over time. Altogether, the results indicate that child mortality reduction may accelerate a transition to lower fertility regimes in Tanzania and similar settings; however, this is likely to occur gradually and via cohort succession. The mechanisms through which child mortality affects fertility behavior are more complex than hypothesized by classical demographic theory and affected by socio-economic insecurity and contingencies that persist in the lives of women contemporaneously with improvements in child health. Thus, improved child survival in our study population, while it translates to increases in uptake of birth spacing, is unlikely to change how women frame their broader family formation strategies. As a corollary, and as observed in Tanzania and similar settings from 2000-15, fertility patterns that are contemporaneous with child mortality reduction, may stall or experience only minor inflections. For child mortality reduction to accelerate fertility transition, there is a need for programs that, on one hand, enhance women's opportunities and abilities to adapt their fertility intentions, and meet them; and, on the other hand, address the drivers of contingency and uncertainty in women's lives that shape their reproductive choices and high fertility in the population.

Future Directions

This dissertation has identified numerous areas in which future research is needed. The lessons from Aim 1 research illuminate the need for additional research on the determinants and

mechanisms of health systems strengthening. Our results revealed the general domains of MNCH service availability and readiness that contribute to implementation strength and validated a methodology for using routinely available data to measure implementation by demonstrating its association with child mortality. This raises important questions about how health care planning, management and delivery process, and contextual factors, generate implementation strength, and inequities in this outcome within local health systems. Future research should address these topics from multiple perspectives and by using a combination of research methods. For example, we need research that explains potential associations between the domains and constructs of implementation strength, such as those that we identified in our analysis, the dynamics of these relationships, how they produce inequalities in implementation strength within health systems, and the effect of coverage inequities on service utilization. To do so, scholars should consider ways to use model-based multivariate statistical methods, such as factor analysis and structural equation modelling, to understand the mechanisms through which health systems become stronger and more accessible, and thereby help elucidate the causal pathway between health systems strengthening and access to care. Triangulating this with qualitative research is especially important, as is incorporating client and health worker perspectives. In our study, we reduced multiple indicators on the structural aspects of service quality into gradients of implementation strength; however, more knowledge is needed on how to measure dimensions of process quality and health worker performance, and their impacts on facilities' overall quality of care. Similarly, future research should examine implementation strength from the perspective clients, addressing questions about the aspects of health care quality that clients value the most and the mechanisms through which they contribute to client satisfaction and shape decision-making on where and when to seek care. Whereas Aim 1 of this dissertation underscores the importance of longitudinal data on population

outcomes for estimating the impacts of health systems strengthening, a gap which it could not fill concerns the absence of similar data on implementation strength. Understanding the dynamics of implementation strength and the factors that cause it to fluctuate temporally are needed to develop approaches to sustain the impacts of health systems strengthening and ensuring better long-term outcomes for populations served. Finally, our study linked prospective data on child survival to the measures of implementation strength ecologically, based on where they lived, not where they received health care. Future research is needed that compares both approaches to linking health systems and population data. Such studies can report on the level of agreement between measures of effective coverage and MNCH outcomes derived from individual-level and ecological linking strategies, identify biases, and help identify better ways to measure the impacts of health systems strengthening.

Through Aim 2 research, we generated evidence on the determinants and mechanisms of CHW effectiveness in relation to mixed results that were posted by a cluster-randomized pragmatic trial. Our analysis revealed that the positive effects associated with CHW deployment were generated through mechanisms that triggered high levels of acceptability of CHW among the residents of intervention communities. The success of these mechanisms was moderated by strength and involvement of community leadership structures in introducing and supporting the CHW. Additional research is needed to better understand the mechanisms of CHW program acceptability and the processes and contexts in which they are triggered, and to refine and test theories to improve the selection and implementation of CHW interventions and their impact on health and behavior change. There is also a need for more evidence on ways to bolster the strength and participation of community leaders in CHW program management and implementation. An important finding of our analysis was that the process of selecting interventions for the CHW

service delivery package did not sufficiently engage communities nor include some services that they valued. In the future, scholars of community-based health care should investigate the feasibility and effectiveness of strategies that integrate existing citizen accountability structures into primary health care planning and implementation. Our qualitative analysis illuminated some of the influences on CHW performance. Future research should build on these lessons and collect more detailed information on the factors that affect CHW productivity, motivation, and quality. Mixed method approaches should be considered for this investigation, including descriptive and model-based multivariate methodologies (e.g., PCA, factor analysis, structural equation modelling), to measure dimensions of contextual and programmatic influences and understand potential associations between them and how they are related to proximal outcomes of CHW, such as coverage and quality. In addition to exploring these issues from the health system perspective, future research could add additional value by measuring CHW perceptions of their work environment and support systems to identify their needs and the mechanisms through which such factors influence their confidence, motivation, and job satisfaction. Aim 2 research findings illustrated how characteristics of health systems, including lapses in core systems functionality and readiness to deliver routine services, condition the ways in which health system and community stakeholders perceive, value, and adopt evidence-based interventions. In the future, researchers should explore these relationships in greater depth. In particular, these studies should direct attention to the multi-level barriers and facilitators of organizational adoption and behavior change, the mechanisms through which communities and systems learn and adapt, and the need for strategies to enable those processes in the context of extending PHC interventions to the community-level.

Findings from Aim 3 research, which examined the fertility response to child mortality in our study area between 2000 and 2015, suggested that family formation in the aftermath of child loss did not adapt as expected in the advent of child survival improvements. Whereas demographic theories and previous research generated an expectation that “insurance effects” i.e., the tendency to hasten childbearing in response to anticipating child loss, would be less pronounced, and “replacement effects”, i.e., the tendency to hasten parity progression to replace a child that just died, would be more pronounced, in settings where child mortality is relatively rare, our analysis reported that the opposite occurred in our study population. Although we conclude that these outcomes may need more time to conform to expectations, there is a need for research to interrogate these findings more deeply and generate evidence that can be used to design programs that meet the needs of women in transitional societies, such as Tanzania. Understanding the durability of “insurance effects” on fertility, and role of socio-economic position in this pattern, is particularly important. Specifically, future research ought to identify the factors that sustain the potency of such insurance motivations, e.g., financial circumstances and insecurity, women’s relationships and social networks, experiences with the health system, and the mechanisms through which these influences shape fertility preferences and family planning behaviors. Implementation science has a key role in this, as knowledge translation is needed to design family planning interventions that are attuned to women’s circumstances and tailored to enhance women’s opportunities, intentions, and abilities to adapt and act on their reproductive goals. In addition, future studies should reflect on our findings that “replacement fertility” was less pronounced in settings with relatively low levels of child mortality. This is contrary to the hypothesis that child mortality reduction precipitates the adoption of fertility regulation practices – both to volitionally avoid pregnancy when it is unintended through the practice of family planning, and to hasten parity progression at times when

having more children is desirable. Forthcoming research should adopt a deeper focus on understanding fertility behavior and contraceptive use in the context of child loss in transitional societies, such as rural Tanzania, to see whether the patterns suggested by our analysis bear out across settings. Findings can inform efforts to strengthen family planning programs so that they better meet the needs of women and families in societies where increasing assurance of child survival may make the practice of modern family planning more appealing.

Appendices

1. Chapter 2: Supplementary Files

S1.1 Association between child mortality risk and MNCH implementation strength using three approaches for addressing missing data for covariates on household SES and mothers' years of schooling.

Model 1: Complete case analysis omitting covariates with missing data.

Model 2: Addresses missing data by imputing community-level median values of household SES and mothers' years of schooling where data are missing.

Model 3: Addresses missing data by using multiple imputation with chained equations.

Associations between child mortality risk and implementation strength scores (n=8999)

	Model 1		Model 2		Model 3	
	HR	95% CI	HR	95% CI	HR	95% CI
IS scores						
IS score 1	0.61*	0.39, 0.97	0.62*	0.39, 0.96	0.61*	0.39, 0.96
IS score 2	0.53*	0.30, 0.97	0.54*	0.30, 0.99	0.54*	0.30, 0.99
IS score 3	0.51	0.20, 1.30	0.48	0.19, 1.24	0.48	0.19, 1.24
Child sex						
Female	-	-	-	-	-	-
Male	1.25*	1.05, 1.48	1.25*	1.05, 1.49	1.25*	1.05, 1.49
Birth order						
No. children (cont.)	1.00	0.88, 1.13	1.00	0.88, 1.13	1.00	0.88, 1.13
Previous birth interval						
Months (cont.)	1.00	0.99, 1.00	1.00	0.99, 1.00	1.00	0.99, 1.01
Subsequent birth interval						
Months (cont.)	1.01***	1.01, 1.02	1.01***	1.00, 1.02	1.01***	1.00, 1.02
Mother age at birth						
Years (cont.)	1.01	0.99, 1.02	1.01	0.99, 1.02	1.01	1.00, 1.02
Mother marital status at birth						
Married/in union	-	-	-	-	-	-
Single	1.43**	1.19, 1.71	1.43**	1.19, 1.72	1.43**	1.19, 1.72
Mother number years of schooling						
Year of schooling (cont.)	-	-	0.98	0.95, 1.01	0.97	0.95, 1.01
Household SES at birth (quintile ranking)						
Fifth	-	-	-	-	-	-
Fourth	-	-	0.87	0.62, 1.22	0.79 [†]	0.60, 1.03
Third	-	-	1.06	0.87, 1.69	0.99	0.75, 1.29
Second	-	-	1.06	0.75, 1.51	0.95	0.71, 1.26
First	-	-	0.97	0.67, 1.41	0.93	0.70, 1.25
Distance to nearest health facility						
Kilometers (cont.)	1.04 [†]	1.00, 1.07	1.14 [†]	0.99, 1.32	1.14 [†]	0.98, 1.32

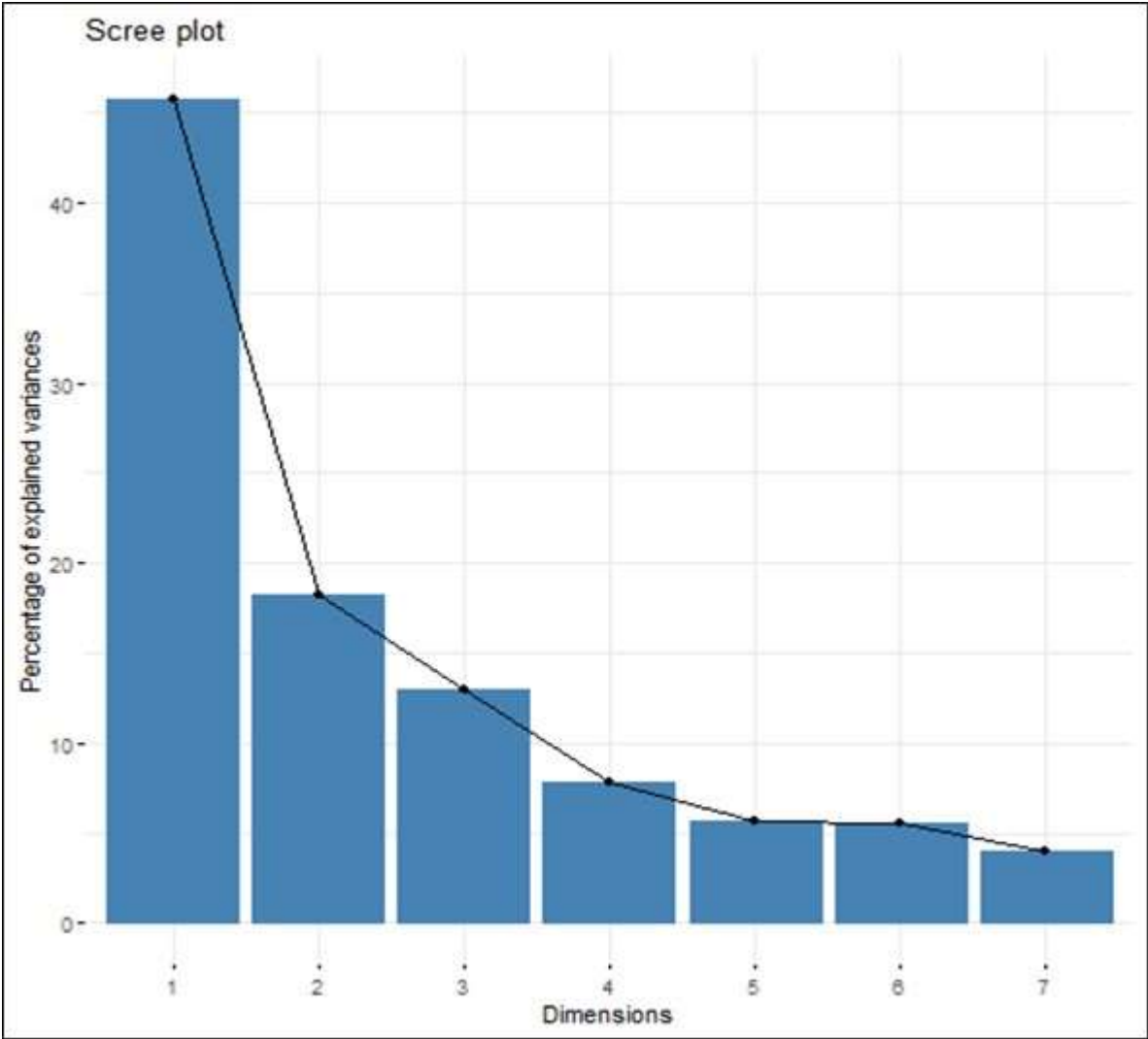
HDSS zone						
Ifakara Expansion	-	-	-	-	-	-
Ifakara Rural	0.83	0.58, 1.20	0.85	0.58, 1.23	0.85	0.58, 1.22
Ifakara Urban	1.35	0.87, 2.10	1.25	0.81, 1.95	1.25	0.81, 1.95
Rufiji Rural	1.08	0.68, 1.73	1.12	0.70, 1.81	1.12	0.70, 1.81
Rufiji Urban	0.93	0.53, 1.64	0.95	0.54, 1.68	0.95	0.54, 1.68

‡ = p-values < 0.1, * = p-values < 0.05, 0.** = p-values < 0.01, *** = p-values < 0.001

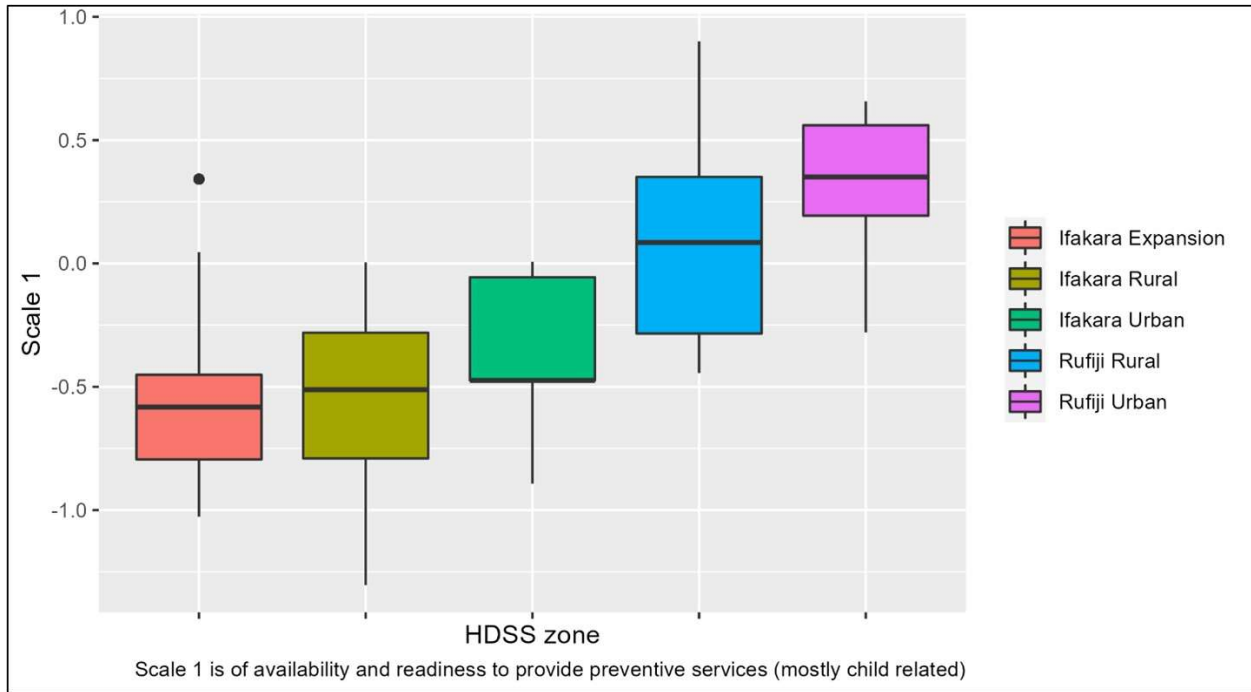
S1.2 Effective coverage indices (median and range) for primary health care facilities (n = 56)

	Ifakara Rural	Ifakara Urban	Ifakara Expansion	Rufiji Rural	Rufiji Urban
General (staffing, management, infrastructure)	0.71 (0.25, 0.99)	0.71 (0.61, 0.99)	0.75 (0.57, 0.99)	0.46 (0.33, 0.72)	0.72 (0.58, 0.99)
Family planning	0.28 (0.06, 0.50)	0.18 (0.06, 0.43)	0.23 (0.08, 0.49)	0.14 (0.00, 0.44)	0.21 (0.08, 0.43)
Antenatal care	0.29 (0.16, 0.49)	0.26 (0.12, 0.47)	0.23 (0.10, 0.48)	0.13 (0.05, 0.64)	0.23 (0.14, 0.70)
Intrapartum care	0.25 (0.08, 0.49)	0.39 (0.15, 0.54)	0.34 (0.15, 0.57)	0.33 (0.14, 0.56)	0.45 (0.20, 0.75)
Postnatal care	0.12 (0.00, 0.30)	0.15 (0.06, 0.28)	0.17 (0.08, 0.28)	0.16 (0.00, 0.38)	0.26 (0.09, 0.37)
Preventive childhood services	0.82 (0.27, 0.99)	0.72 (0.27, 0.99)	0.68 (0.22, 0.90)	0.53 (0.28, 0.83)	0.63 (0.27, 0.90)
Sick childcare	0.30 (0.13, 0.54)	0.36 (0.17, 0.69)	0.33 (0.15, 0.48)	0.21 (0.13, 0.45)	0.30 (0.19, 0.46)

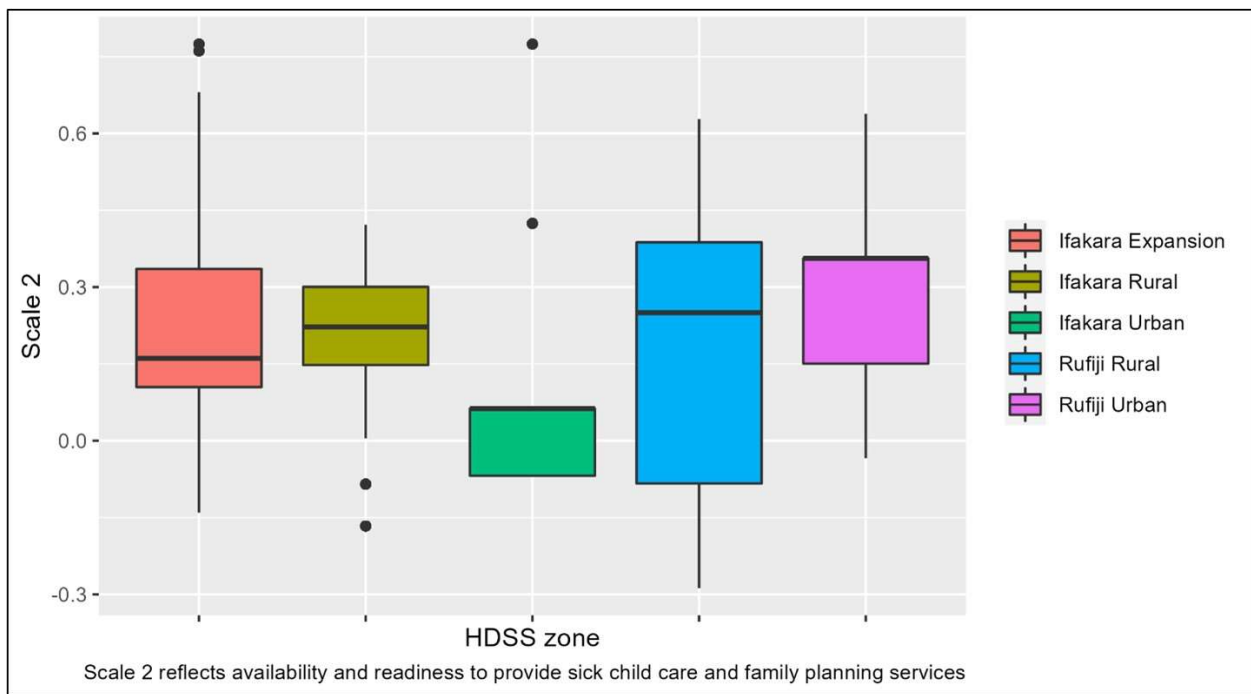
S1.3 Figure 3: Scree plot reported by the principal components analysis of effective coverage indices.



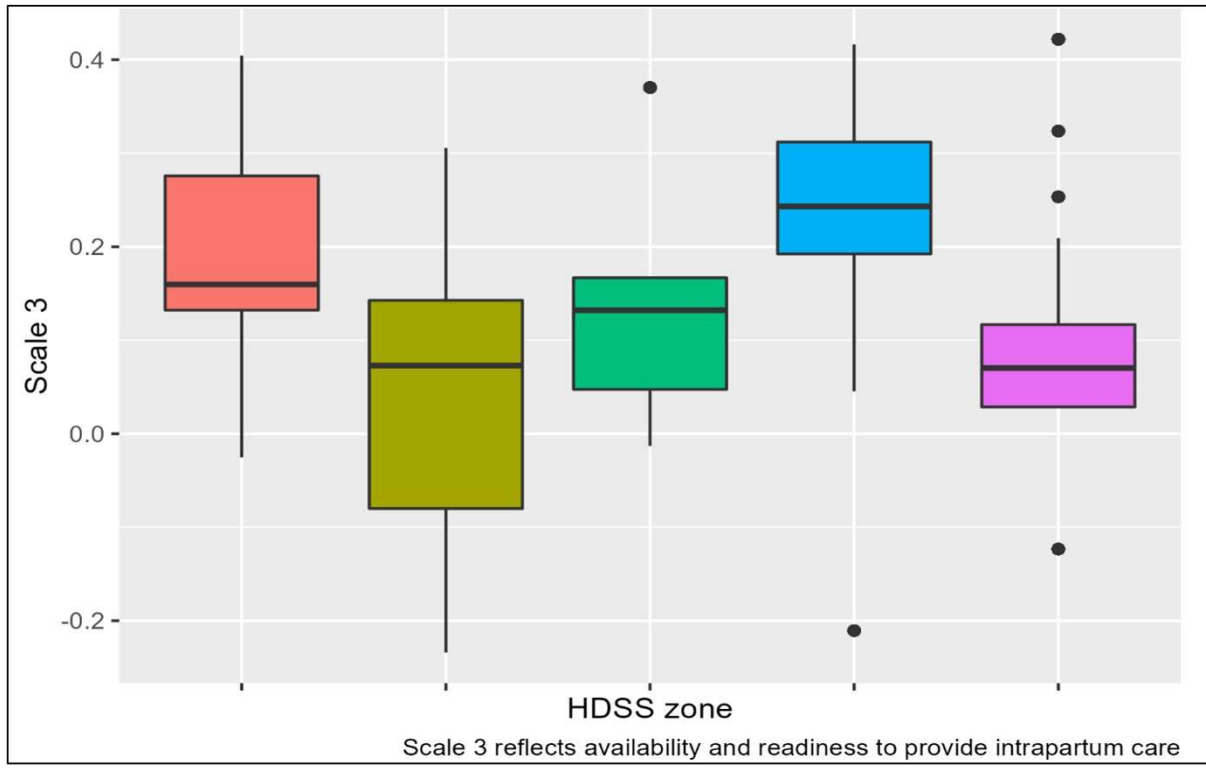
S1.4a Distribution of implementation strength scale 1's median values across geographic zones within local health systems in the study area.



S1.4b Distribution of implementation strength scale 2's median values across geographic zones within local health systems in the study area.



S1.4c Distribution of implementation strength scale 3's median values across geographic zones within local health systems in the study area.



S1.5 Association between newborn (< 1 month), infant (< 12 months) and child (≤ 60 months) mortality and implementation strength.

Associations between childhood mortality risks and implementation strength scores reported from Model 3 (IS scores, mother-, child- and contextual-level covariates) (n=8,999)

	Newborn mortality		Infant mortality		Child mortality	
	HR	95% CI	HR	95% CI	HR	95% CI
IS scores						
IS score 1 (ANC, PNC and preventive child care)	0.59	0.28, 1.22	0.64	0.36, 1.12	0.61*	0.39, 0.96
IS score 2 (sick child care)	0.45	0.17, 1.19	0.48 [†]	0.23, 1.02	0.54*	0.30, 0.99
IS score 3 (intrapartum care)	0.65	0.14, 3.05	0.45	0.14, 1.49	0.48	0.19, 1.24
Child sex						
Female	-	-	-	-	-	-
Male	1.10	0.85, 1.43	1.12	0.92, 1.37	1.25*	1.05, 1.49
Birth order						
No. children (cont.)	1.20*	1.01, 1.43	1.13	0.97, 1.30	1.00	0.88, 1.13
Previous birth interval						
Months (cont.)	1.00	0.99, 1.00	1.00	0.99, 1.00	1.00	0.99, 1.01
Subsequent birth interval						
Months (cont.)	1.01**	1.00, 1.02	1.01* **	1.00, 1.02	1.01* **	1.00, 1.02
Mother age at birth						
Years (cont.)	1.00	0.98, 1.00	1.00	0.99, 1.02	1.01	1.00, 1.02
Mother marital status at birth						
Married/in union	-	-	-	-	-	-
Single	1.51**	1.15, 2.00	1.34* *	1.09, 1.67	1.43* *	1.19, 1.72
Mother number years of schooling						
Year of schooling (cont.)	1.00	0.96, 1.05	0.98	0.94, 1.01	0.97	0.95, 1.01
Household SES at birth (quintile ranking)						
Fifth	-	-	-	-	-	-
Fourth	1.01	0.67, 1.54	0.86	0.62, 1.19	0.79 [†]	0.60, 1.03
Third	1.19	0.77, 1.81	1.18	0.86, 1.61	0.99	0.75, 1.29
Second	0.98	0.63, 1.55	1.00	0.72, 1.40	0.95	0.71, 1.26
First	1.03	0.48, 2.24	1.03	0.73, 1.46	0.93	0.70, 1.25
Distance to nearest health facility						
Kilometers (cont.)	0.99	0.78, 1.25	1.05	0.87, 1.25	1.14 [†]	0.98, 1.32
HDSS zone						
Ifakara Expansion	-	-	-	-	-	-
Ifakara Rural	0.67	0.37, 1.19	0.65 [†]	0.42, 1.02	0.85	0.58, 1.22
Ifakara Urban	1.07	0.53, 2.19	1.29	0.75, 2.23	1.25	0.81, 1.95
Rufiji Rural	0.84	0.39, 1.81	0.78	0.43, 1.41	1.12	0.70, 1.81
Rufiji Urban	0.76	0.31, 1.88	0.68	0.33, 1.39	0.95	0.54, 1.68

[†] = p-values < 0.1, * = p-values < 0.05, ** = p-values < 0.01, *** = p-values < 0.001

2. Chapter 3: Supplementary Files

S2.1 CONSORT checklist items for reporting pragmatic trials

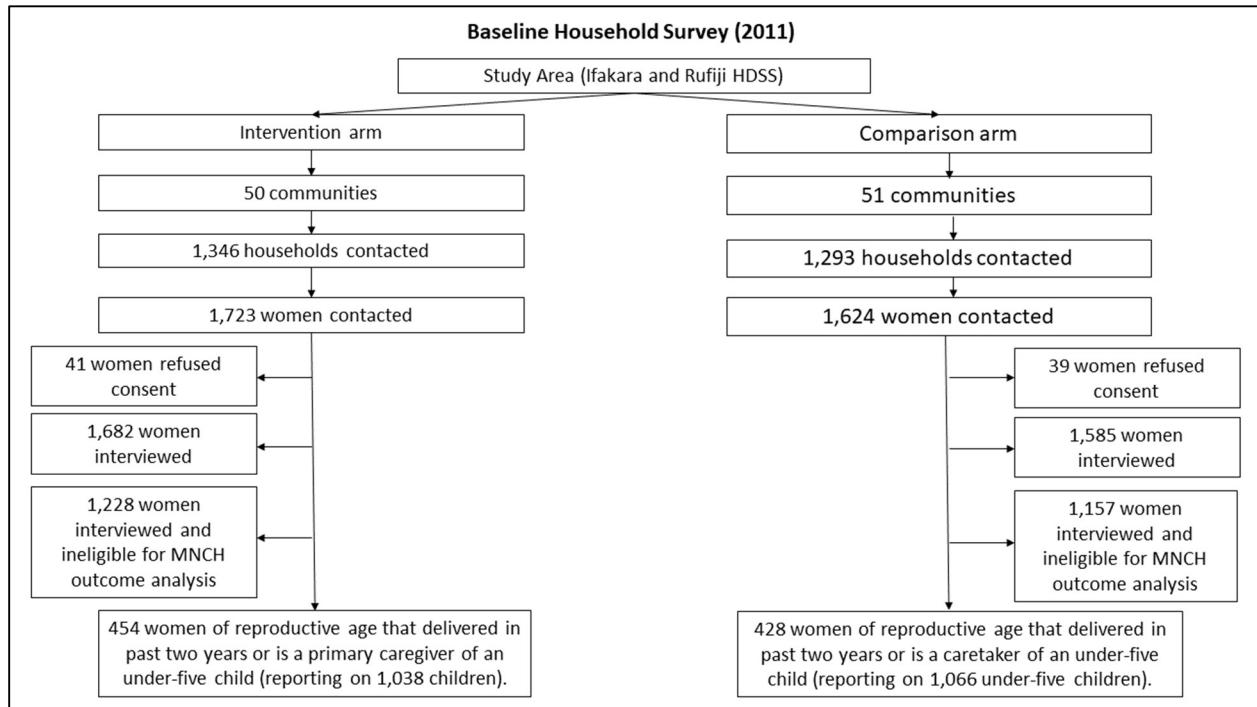
Section	Item	Standard CONSORT description	Extension for pragmatic trials	Page
Title and abstract	1	How participants were allocated to interventions (eg, “random allocation,” “randomised,” or “randomly assigned”)		42-43
Introduction				
Background	2	Scientific background and explanation of rationale	Describe the health or health service problem that the intervention is intended to address and other interventions that may commonly be aimed at this problem	45-46
Methods				
Participants	3	Eligibility criteria for participants; settings and locations where the data were collected	Eligibility criteria should be explicitly framed to show the degree to which they include typical participants and/or, where applicable, typical providers (eg, nurses), institutions (eg, hospitals), communities (or localities eg, towns) and settings of care (eg, different healthcare financing systems)	49-50, 52, 54-55
Interventions	4	Precise details of the interventions intended for each group and how and when they were actually administered	Describe extra resources added to (or resources removed from) usual settings in order to implement intervention. Indicate if efforts were made to standardise the intervention or if the intervention and its delivery were allowed to vary between participants, practitioners, or study sites	47-50

Section	Item	Standard CONSORT description	Extension for pragmatic trials	Page
			Describe the comparator in similar detail to the intervention	51
Objectives	5	Specific objectives and hypotheses	5	46
Outcomes	6	Clearly defined primary and secondary outcome measures and, when applicable, any methods used to enhance the quality of measurements (eg, multiple observations, training of assessors)	Explain why the chosen outcomes and, when relevant, the length of follow-up are considered important to those who will use the results of the trial	43
Sample size	7	How sample size was determined; explanation of any interim analyses and stopping rules when applicable	If calculated using the smallest difference considered important by the target decision maker audience (the minimally important difference) then report where this difference was obtained	51-52, 55
Randomisation—sequence generation	8	Method used to generate the random allocation sequence, including details of any restriction (eg, blocking, stratification)		50-51
Randomisation—allocation concealment	9	Method used to implement the random allocation sequence (eg, numbered containers or central telephone), clarifying whether the sequence was concealed until interventions were assigned		50-51
Randomisation—implementation	10	Who generated the allocation sequence, who enrolled participants, and who assigned participants to their groups		50-51

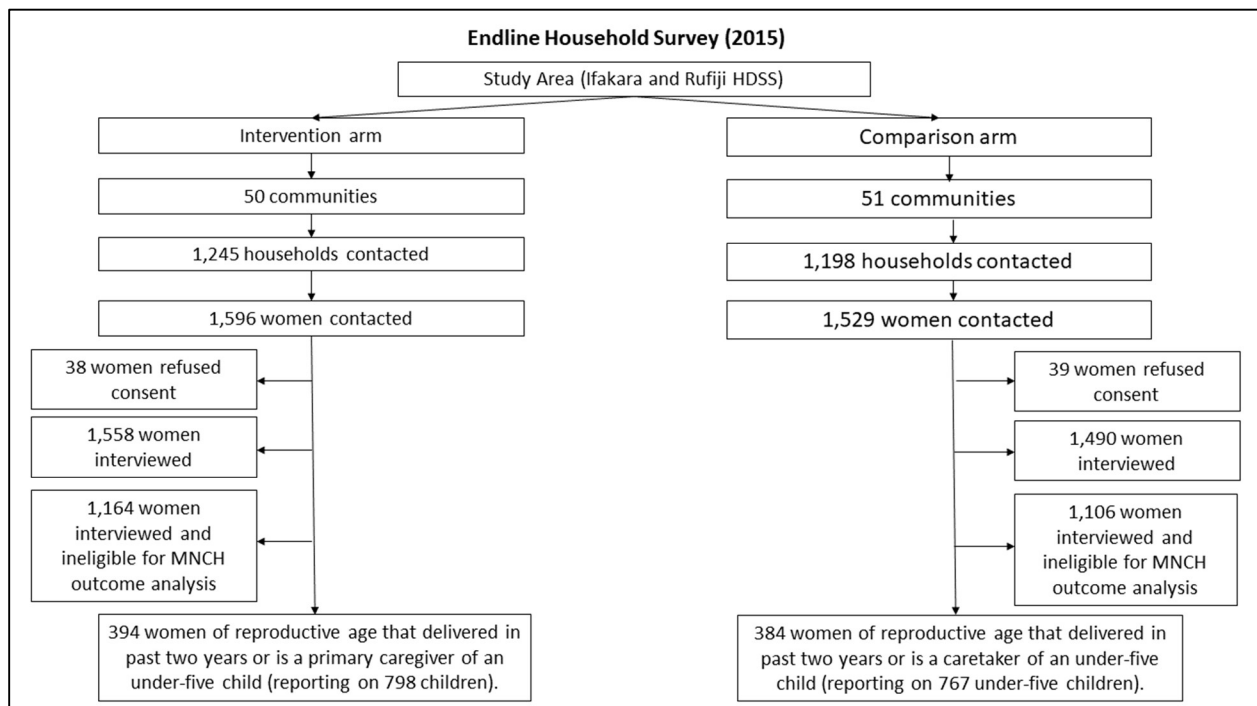
Section	Item	Standard CONSORT description	Extension for pragmatic trials	Page
Blinding (masking)	11	Whether participants, those administering the interventions, and those assessing the outcomes were blinded to group assignment	If blinding was not done, or was not possible, explain why	50-51
Statistical methods	12	Statistical methods used to compare groups for primary outcomes; methods for additional analyses, such as subgroup analyses and adjusted analyses		53-54
Results				
Participant flow	13	Flow of participants through each stage (a diagram is strongly recommended)—specifically, for each group, report the numbers of participants randomly assigned, receiving intended treatment, completing the study protocol, and analysed for the primary outcome; describe deviations from planned study protocol, together with reasons	The number of participants or units approached to take part in the trial, the number which were eligible, and reasons for non-participation should be reported	52 (and Supplemental File 2.2a-b)
Recruitment	14	Dates defining the periods of recruitment and follow-up		51, 54
Baseline data	15	Baseline demographic and clinical characteristics of each group		Supplemental File 2.3, 47
Numbers analysed	16	Number of participants (denominator) in each group included in each analysis and whether analysis was by “intention-to-treat”; state the results in absolute numbers when feasible (eg, 10/20, not 50%)		58 (Table 3), 54 (Table 2)

Section	Item	Standard CONSORT description	Extension for pragmatic trials	Page
Outcomes and estimation	17	For each primary and secondary outcome, a summary of results for each group and the estimated effect size and its precision (eg, 95% CI)		56-58 (quantitative) 58-68 (qualitative)
Ancillary analyses	18	Address multiplicity by reporting any other analyses performed, including subgroup analyses and adjusted analyses, indicating which are prespecified and which are exploratory		56-57
Adverse events	19	All important adverse events or side effects in each intervention group		
Discussion				
Interpretation	20	Interpretation of the results, taking into account study hypotheses, sources of potential bias or imprecision, and the dangers associated with multiplicity of analyses and outcomes		70-76
Generalisability	21	Generalisability (external validity) of the trial findings	Describe key aspects of the setting which determined the trial results. Discuss possible differences in other settings where clinical traditions, health service organisation, staffing, or resources may vary from those of the trial	75-76
Overall evidence	22	General interpretation of the results in the context of current evidence		70-76

S2.2a Participant flow diagram (baseline)



S2.2b Participant flow diagram (endline)



S2.3 Socio-demographic balance of baseline sample (2011)

Outcome		Baseline (2011)		
<i>Mother-level indicators</i>		<i>Intervention (n=454)</i>	<i>Comparison (n=428)</i>	<i>p</i>
1	Age (mean, range)	27 (15, 46)	28 (15, 48)	<0.001
2	Educational attainment (% , n)			0.34
	Did not study	27 (123)	30 (129)	
	Primary only	70 (318)	66 (283)	
	Some secondary and higher	3 (13)	4 (16)	
3	Marital status (% , n)			0.11
	Married	85 (386)	83 (355)	
	Not married	15 (68)	17 (73)	
4	Wealth status (quintile) (% , n)			0.07
	First	21 (95)	16 (68)	
	Second	22 (100)	23 (98)	
	Third	28 (127)	27 (116)	
	Fourth	18 (82)	19 (81)	
	Fifth	11 (50)	15 (65)	
5	Parity (mean, range)	3.6 (1, 12)	3.8 (1, 14)	<0.001
6	Distance to nearest health facility			0.37
	<5 kilometers	49 (222)	52 (223)	
	5-10 kilometers	38 (173)	36 (154)	
	≥10 kilometers	13 (59)	12 (51)	
<i>Child-level indicators</i>		<i>Intervention (n=1038)</i>	<i>Comparison (n=1066)</i>	
1	Age (months) (mean, range)	32 (4, 59)	34 (3, 57)	0.54
2	Sex			0.41
	Male	50 (514)	50 (537)	
	Female	50 (524)	50 (529)	
3	Primary caregiver			0.79
	Parent	94 (972)	94 (1004)	
	Other	6 (66)	6 (64)	