Documentation of a fully integrated epidemiological-demographic-macroeconomic model of Malaria: The case of Ghana

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Abstract

We develop a novel and fully integrated epidemiological-demographic-macroeconomic EDM-malaria simulation model framework for modelling of *P. falciparum* malaria transmission in Ghana. Our model framework represents a milestone, as the first fully integrated EDM model framework for any type of infectious disease. The complex specification and integration of regional epidemiological-demographic models within a malaria-focussed macroeconomic Computable General Equilibrium model is fully described and documented, and ideas are outlined for future applications to investigate the interplay between macroeconomic and health disease burdens, to measure the health and economic impacts of economic growth and malaria interventions, and to study the importance (or lack thereof) of the general omission of proper epidemiological underpinnings and integration of economic incentive feedback effects in the existing literature on macroeconomic assessment of infectious disease.

1. Introduction

The current study presents and documents a fully integrated and dynamically-recursive epidemiological-demographic-macroeconomic (EDM) simulation model of *P. falciparum* malaria transmission in Ghana during 2015-34. So far, there has been a complete lack of proper empirical tools for combined and consistent assessment of macroeconomic and health disease burdens and policy analysis of interventions, associated with infectious diseases such as malaria. A variety of approaches have been applied for macroeconomic health burden assessment of HIV/AIDS epidemics including neoclassical growth and overlapping generations models (Cuddington 1993a, 1993b; Cuddington and Hancock 1994; Bell, Devarajan and Gersbach 2003, 2004, 2006; Young 2005; Bell, Bruhns and Gersbach 2006; Johansson 2007; Roe and Smith 2008; Ventelou et al. 2008), and multi-sector Computable General Equilibrium (CGE) models (Kambou, Devarajan and Over 1992; Arndt and Lewis 2000, 2001; Arndt 2006; Jefferis et al. 2008; Ventelou et al. 2008; Thurlow, Gow & George 2009). In contrast, the malaria-focussed macroeconomic health burden literature is narrow (Ashraf, Lester and Weil 2009; Pattanayak et al. 2009; Anthoff & Tol 2012; Tol 2013). Some HIV/AIDS studies have employed epidemiological satellite models to specify health burden shocks, but no malaria or HIV/AIDS studies fully account for economic feedback effects by integrating epidemiological models (of HIV/AIDS transmission) within the macroeconomic assessment tools. The lack of proper epidemiological underpinnings is also characteristic of the Anti-Microbial Resistance literature (Smith et al. 2005) and the Pandemic Flu literature (Keogh-Brown & Smith 2008, Smith, Keogh-Brown et al. 2009; Keogh-Brown, Smith et al. 2010; Keogh-Brown, Wren-Lewis et al. 2010; Smith & Keogh-Brown 2013).

By specifying a fully integrated and dynamically-recursive EDM-malaria transmission model for Ghana, based on a core malaria-focussed macroeconomic CGE model and with detailed feedback effects between economic and epidemiological models via private demand for malaria interventions, we aim to create a model-consistent health and macroeconomic model framework which can be used to investigate the interplay between health and macroeconomic disease burdens, measure the health and economic impacts of malaria interventions, and study the importance (or lack thereof) of the general omission of proper epidemiological underpinnings and integration of economic incentive feedback effects in the existing literature. Ghana is a typical Sub-Saharan African (SSA) country where malaria is hyperendemic, infections are dominated by the virulent *Anopheles* mosquito vector-borne *Plasmodium falciparum* parasite, and transmission, in most areas except for the savannah region, occurs all year round, putting the entire population of 27.2 million (2015) at risk (WHO 2016). In spite of donor-supported scaling-up, the reported numbers of suspected out-patient department malaria cases increased from 3.1-3.5 million during 2001-2008 (NMCP 2009) to 8.1-11.1 million per year during 2010-14 (NMCP 2015). While the jump is likely to

reflect improved monitoring, the numbers indicate that the health disease burden remains high and that the control of malaria continues to represent a critical challenge to Ghanaian authorities.

In order to consistently measure future health and macroeconomic disease burdens and impacts of malaria interventions, and to study the importance of economic incentive feedback effects, we integrate a macroeconomic dynamically-recursive CGE model framework, calibrated on the basis of a recently developed 2004 malaria-focussed Social Accounting Matrix (MalSAM) (Jensen, Keogh-Brown et al. 2012), with a MacDonald-Ross compartment model of malaria transmission (Anderson and May 1991), which has been extended to account for human super-infections (Dietz 1988). We purposefully constructed the 2004 MalSAM data set, and calibrated our CGE model, to include 19 household categories stratified according to (1) rural-urban location, (2) coastal-forest-savannah eco-region location, and (3) low-medium-high malaria prevalence district location. By capturing regional variation in transmission intensities, this stratification allowed us to construct and match 19 epidemiological and demographic models thereby ensuring that regional variations in malaria transmission are captured endogenously within our model. The 19 regional demographic models also capture interregional and international migration flows and accompanying changes in population exposure. The full EDM-malaria model framework allows us to undertake policy analyses with model-consistent macroeconomic and clinical health outcome indicators, and to produce consistent macroeconomic and health burden assessments with decompositions across macroeconomic cost components and economic incentive mechanisms. The model is set up to assess future policy scenarios and disease burdens over the two coming decades (2015-34), defined as the dynamically accumulating policy impacts or future burdens avoided by current interventions or elimination of malaria transmission.

To our knowledge, the current EDM-malaria model is the first fully-integrated empirical EDM model framework for any type of infectious disease. In our model framework, economic incentives affect regional epidemiological and clinical outcomes, and *inter alia* macroeconomic and health disease burdens, in two ways: (1) demand for prevention and treatment interventions, and (2) migration between Ghana regions with varying malaria transmission intensities. In turn, clinical outcomes, in the form of uncomplicated malaria episodes and excess malaria mortality, drive macroeconomic feedback effects on regional labour markets as well as private and government expenditure patterns for malaria-related composite intervention commodities. We specifically distinguish between pecuniary macroeconomic impacts/economic disease burdens and non-pecuniary human disease impacts/health disease burdens. The focus on pecuniary outcomes is particularly important for undertaking sound and sustainable public (malaria) funding allocations in an otherwise capital-constrained developing country context, while non-

pecuniary (malaria) health indicators are critical for effectively pursuing the overarching policy objective of enhancing human welfare.

The rest of the paper is organized so as to fully document the individual epidemiological, demographic and macroeconomic models, and the transmission mechanisms which links them together: The macroeconomic dynamically-recursive CGE model framework for Ghana, including parametrization and calibration to our 2004 malaria-focussed macroeconomic SAM database, is presented and described in section 2; the epidemiological model equations and their parametrization are presented and described in section 3; the demographic model equations and their parametrization are presented and described in section 4; the specification and parametrization of our effective labour supply equations and health intervention equations linking the epidemiological, demographic and macroeconomic models are presented in respectively sections 5 and 6; while conclusions are offered in section 7.

It should be noted that our economy-wide dynamically-recursive Computable General Equilibrium (CGE) model is based on the static 'IFPRI standard model' (Löfgren, Lee Harris and Robinson 2002). In what follows, model equations are only presented if they represent (1) fully new equations, or (2) adaptations of existing standard model specifications. For all other equations, please consult the documentation of the standard model (ibid.) We also adopt the notation from the documentation of the standard model in terms of variables, parameters, and sets (ibid.)

2. Malaria-focussed SAM database and CGE model calibration

Malaria-focussed Social Accounting Matrix (SAM) data base

The calibration of our malaria-focussed Computable General Equilibrium (CGE) model was based on a 2004 malaria-focussed Social Accounting Matrix (MalSAM) for Ghana (Jensen, Keogh-Brown et al. 2012). The 2004 Ghana MalSAM data set was constructed with the explicit purpose of providing a data structure which captures the diversity of *P. falciparum* malaria transmission across Ghana and thereby allows for constructing an integrated Epidemiological-Demographic-Macroeconomic (EDM) model framework which can be applied for appropriate integrated analyses of malaria epidemiology and macroeconomic outcomes.

The 2004 Ghana MalSAM was constructed on the basis of a previously established 2004 Ghana SAM (Jensen, van den Andel & Duncan 2008). The original 2004 Ghana SAM included 175 activities, 139 commodities, 22 factor types (1 capital factor and 21 labour factors distinguished by rural-urban location, coastal-forest-savannah eco-region location, and low-medium-high skill levels), and 21 household types (distinguished by rural-urban location, GAMA-coastal-forest-savannah eco-region location, and low-medium-high education of head of household; Greater Accra Metropolitan Area (GAMA) was classified as a separate urban region without rural areas).

For our current purposes, we needed to distinguish between gender types of labour factors (due e.g. to the gender-specific differences in absenteeism due to female caregiving for malaria-sick children). Our 2004 MalSAM therefore includes 43 different factor types including 1 capital factor and 42 labour factor types (distinguished by rural-urban location, coastal-forest-savannah eco-region location, low-medium-high skill levels, and male-female gender types). Data for disaggregation of labour value added between gender types were obtained from the 2004 Ghana Supply and Use Tables (van den Andel 2007).

The household classification of the original 2004 Ghana SAM was also inappropriate for our current purposes. One of the key transmission mechanisms between our epidemiological and macroeconomic models link impacts on households' labour factor ownership and effective labour force participation (see section 5). In order to properly capture regional differences in malaria transmission (see section 3), it was deemed necessary (and sufficient) to keep the Greater Accra Metropolitan Area (GAMA) as a separate household type, and classify the rest of Ghana into 18 household types according to (1) rural-urban location, (2) coastal-forest-savannah eco-region location, and (3) low-medium-high malaria prevalence district location (within each region). The two former rural-urban and eco-region location classifications were derived from the original SAM data asset, while the malaria prevalence classification was achieved by defining two district level threshold values, 33% and 47%, resulting in an equal sharing of Ghana's 110

districts (2005 administrative classification) into low transmission districts (37), medium transmission districts (36), and high transmission districts (37). The disaggregation into 19 households, categorized according to malaria prevalence rates, was achieved by linking malaria prevalence data from the Malaria Atlas Project (Gething et al. 2011) with Ghana household survey data from the 2005/06 GLSS5 data set (GSS 2008) (see section 3.3 for additional details about the mapping of epidemiological and economic data).

While the definition of our two threshold values for malaria prevalence ensured a balanced overall distribution of low, medium, and high prevalence districts, distributions within eco-regional household types were, as expected, less balanced with overrepresentations of low transmission districts in coastal region households (18 of 27), medium transmission districts in forest region households (24 of 47), and high transmission districts in savannah region households (27 of 36). The new factor and household account typologies, which were developed for the 2004 Ghana MalSAM, were retained in the final aggregated 2004 Ghana MalSAM as outlined in Table A.1 (annex A).

The 2004 Ghana MalSAM retained the 175 activity and 139 commodity accounts from the original 2004 Ghana SAM (Jensen, Keogh-Brown et al. 2012). However, for the purposes of CGE modelling and in order to reduce the complexity of our EDM-malaria model framework, we decided to aggregate these accounts into 10 activity and commodity accounts. Separate health activity ('a10') and health commodity ('c10') accounts were retained in order to allow for modelling of health interventions and health system costs, and separate agricultural, industry and service sectors were also retained. The activity and commodity accounts, which were retained in the final aggregated 2004 Ghana MalSAM, are also outlined in Table A.1 (annex A).

Computable General Equilibrium (CGE) model specification and calibration

The economy-wide dynamically-recursive Computable General Equilibrium (CGE) model, presented in this paper, is based on the 'IFPRI standard model' (Löfgren, Lee Harris and Robinson 2002). This is a well-known and widely applied comparative static, single country, open economy, multi-sector CGE model which has recently been used in health and trade-related applications by some of the authors (e.g. Smith, Keogh-Brown et al. 2009; Lock, Smith et al. 2010, Jensen, Keogh-Brown, Smith et al. 2013). The model accounts for different types of agents including producers/enterprises, private households, the government sector and the foreign sector.

There are several reasons why our CGE model approach is a superior tool for the purpose of analysing economic analysis of an infectious disease such as malaria. In particular, the integrated framework allows for (1) measuring the economic values of commodities, services and employment at which the net economic costs of malaria illness and malaria interventions are assessed, and it also allows for (2)

specification of endogenous interactions and feedback effects between the CGE model and other sub-models including private intervention demands (see section 6) and private migration decisions (see section 4) which links economic outcomes to demographic impacts and malaria exposure, and subsequently feeds back into the calculation of economic values and net economic costs. In terms of modelling, our wage-driven private migration specifications were accommodated by our disaggregation of labour factor use in production, discussed above, which allowed for modelling of gender- and region-specific wage levels. In terms of policy interventions, we adapted our framework to be able to handle both benefit- and cost-sides. The benefit-side involves health-related changes in labour supplies due to reduced absenteeism of sick adults and (female) caregivers for sick children, while the cost-side involves increased private and government expenditures of treatment and prevention interventions and administrative and laboratory services.

In order to capture all of the above-mentioned migration- and intervention-related transmission mechanisms, our simulation framework needed to include (as a minimum): (1) multiple production sectors to capture the production and supply of health services, and multiple production factors to capture the variations in employment patterns and resulting variations in gender- and region-specific wage-levels, (2) endogenous goods and factor prices (to capture important GE effects from e.g. health-related demand- and supply-shocks to goods and factor markets including the dynamic wage impact of economic development over time), (3) separate private and government accounts (to capture changes in private and government sector income and expenditure patterns), and (4) a dynamic specification (to capture future benefits and costs associated with investment and capital accumulation, and long-term health impacts on the labour force). Our dynamically-recursive multi-sector Computable General Equilibrium (CGE) model framework shares all of the above features, and therefore allows for capturing all of the required transmission mechanisms.

As noted above, the CGE model for the Ghana economy was calibrated on the basis of the aggregated 2004 MalSAM dataset with 10 production activities and 10 retail commodities. A further three commodities were created specifically to allow for analysing private treatment and prevention intervention demand for respectively ITN and ACT commodities and for an additional ACT-related 'composite malaria treatment' commodity (see equation (6.6'') for the specification of demand for intervention-related composite health services). The former ITN and ACT commodities were assumed to be imported, while a Leontief production specification was invoked to specify the supply of the 'composite ACT treatment' commodity (the 'composite ITN prevention' commodity was assumed to be identical with the ITN input), implying fixed input shares of (1) ACT drugs, and (2) administrative and laboratory services. The latter services were

assumed to be supplied by the domestic health sector. Value breakdowns were based on non-AMFm ACT prices and laboratory costs from an Affordable Medicines Facility-malaria (AMFm) evaluation study (Bate et al. 2012) and from a KNUST hospital study (Dontwi, Dedu & Aboagye 2013) (see Table 6.1).

In line with the abovementioned demands for composite intervention commodities, our CGE model is based on the fundamental principles of profit maximization among producers and utility maximization among households. Production (apart from composite intervention commodities) is specified as a Constant Elasticity of Substitution (CES) function of aggregate intermediate input demand (individual commodity input demands are determined by a Leontief specification) and aggregate factor input demand (individual factor input demands are determined by a CES specification). Standard elasticity values were used for the top-level production specification (0.8) and the bottom-level factor input demand specification (0.6). Trade between domestic and foreign agents is specified as a function of relative prices (determined by the real exchange rate), based on an Armington CES specification on the import side and a Constant Elasticity of Transformation (CET) specification on the export side. Standard trade elasticity values were applied on the import side (0.8) and on the export side (1.6).

On the consumer side, our model relies on a standard Linear Expenditure System (LES) for household demand. The LES demand system was calibrated to the 10 commodities from our aggregate 2004 MalSAM (Table A.1, annex A), and subsequently extended to include private composite intervention commodity demand (see the extended LES demand system equations (6.6')-(6.6'') in section 6) and derived demands for prevention interventions (ITNs) and treatment interventions (ACTs) (see input demand equations (6.8')-(6.8'') in section 6). Calibration of the full 12 commodity LES demand system was ensured by extracting 'ITN' and 'composite malaria treatment' expenditure patterns from the aggregate 'health' commodity 'c10'. The extension of the LES demand system to include private demand for malaria-related composite intervention commodities is a key specification in our integrated EDM model, which allows for endogenous feedback effects from the economic model to the epidemiological model.

As is customary, the LES specification assumes that the Frisch parameter, for our 12 commodities, is based on the development level of Ghana (in the CGE model literature, Frisch parameters are typically derived from an econometric relationship with GDP per capita, estimated by Lluch, Powell and Williams (1977): $-36 * GDPcap^{-0.36}$. Based on the 2010 Ghana GDP per capita (1,283US$), we assumed that the Frisch parameter is -2.74 for Ghana, and, based on the standard assumption of income elasticities of +1.0, we calibrated household-specific autonomous consumption levels for all commodities except for the two malaria-related composite intervention commodities where income elasticities were derived from the literature (see Table 6.2 and equations (6.18)-(6.19) for details; see section 6.3 for additional discussion).

The original 'standard model' (Löfgren, Lee Harris and Robinson 2002) relies on fixed factor income shares to compute household factor income aggregates. This specification is not satisfactory, however, when region-specific population and workforce levels are tracked and household-specific workforce compositions are likely to change. The addition of a set of regional demographic models therefore prompted us to extend the core 'standard model' with a set of explicit factor ownership equations in order to keep track of household-specific factor ownership and thereby to ensure that household income generation, within the fully integrated EDM-malaria model framework, is model-consistent (see section 5 for technical details on equation specifications).

With the addition of standard factor-updating equations for labour and capital, we finally derived our dynamically-recursive malaria-focussed Ghana CGE model. The framework solves recursively for annual equilibria, and this is consistent with our high-frequency discrete time epidemiological model specification (see discussion in section 3 below). The dynamically-recursive CGE model had to be run forward from 2004 (the SAM base year) to 2015 (the base year of the 2015-2034 simulations). To ensure that the model mirrored the 2015 Ghana economy, we targeted key macroeconomic aggregates (nominal and real GDP) over the period 2004-2010. Furthermore, we created reasonable counterfactual growth paths for respectively 2011-2015 and 2015-2034 by targeting 2006-2010 historical Ghana growth rates for nominal GDP (25.4% p.a.) and real GDP (6.6% p.a.). The counterfactual real growth rates matches the recent real GDP growth experience in Ghana (IMF 2016, 2017) and the implied GDP deflator (our numeraire price index), which grows at 17.4% p.a., matches recent inflation experience (ibid.)

For our baseline 2015-2034 projections (not shown), we apply the historical Ghana growth rates, described above, and employ a standard neo-classical model closure implying that flexible prices are clearing all goods and factor markets and that a flexible real exchange rate is clearing the current account of the balance of payments. Alternative structuralist CGE model approaches with a focus on market imperfections exist (see e.g. Taylor 1983; Robinson 1991; Agénor, Izquierdo and Jensen 2007), but the neo-classical closure with explicit imposition of resource constraints was considered to be appropriate for our long term health- and labour-focussed baseline simulations. Our baseline projections also uses on a balanced macro closure (fixed government demand-to-absorption ratio) which ensures a relatively unchanged composition of absorption (private and government consumption vs. investment) over the projection period. Our Net Present Value (NPV) calculations of economic outcomes, which use a real discount rate of 5.0% p.a., is based on a nominal discount rate of 22.4% p.a. (consistent with the 17.4% p.a. inflation rate). The 5.0% p.a. real discount rate mirrors recent real interest rates on domestic Ghanaian public debt: 4.3-4.7% p.a. in 2013-14 (IMF 2016).

3. Epidemiological model equations and parametrization

In this section, we specify a full set of bi-weekly discrete time epidemiological models stratified over our 19 malaria-focussed household types (section 3.1), and we also model clinical health outcomes as closed-form piece-wise linear specifications based on 'seasonal transmission'-corrected lookup tables derived from the Swiss Tropical Institute model (section 3.2). All epidemiological specifications are parameterized in section 3.3, while endogenous variables and exogenous parameters are listed and defined in Annex B.

3.1. Household-specific epidemiological models for malaria transmission

The household-specific epidemiological sub-models of our EDM-malaria model framework are calibrated to model clinical outcomes of *Plasmodium falciparum* parasites. *P. falciparum* is the dominant type of malaria parasite in Ghana and Sub-Saharan Africa (SSA). It is transmitted by female Anopheles mosquitoes (main vector: Anopheles Gambiae), and it is the most virulent form of malaria infection with the highest rates of complications and mortality.

The epidemiological model framework relies on a standard MacDonald-Ross compartment model (Anderson and May 1991), which has been expanded with a Ditz specification for modelling of super-infections (Dietz 1988), i.e. multiple infections with different types of parasites. While the literature specify continuous time models, we specify our epidemiological model framework in discrete time to match the discrete time specifications of our other macroeconomic and demographic sub-models (see sections 2 and 4).

The epidemiological model employs the so-called "reversible catalytic model" for modelling of malaria prevalence and superinfections. The origin of this approach can be traced back to Muench (1959) and subsequent malaria-applications include Bekessy, Molineaux, and Storey (1976) and Drakeley et al. (2005). In our case, the reversible catalytic model framework allows us to derive the difference equation (3.1), essentially the general solution to the Poisson-distributed multiplicity of super-infections (see derivation in section 3.4.1), with the multiplicity of malaria infections ($N_{\mathrm{h,tbw}}$) as state variable:

$$(3.1) \qquad N_{h,tbw+1} = \left(N_{\mathrm{h,tbw}} - \frac{\bar{\lambda}_h^S * \lambda_{h,tbw}^{FOI}}{\bar{\mu}^S}\right) * \exp\left(-\bar{\mu}^{\mathrm{S}}\right) + \frac{\bar{\lambda}_h^S * \lambda_{h,tbw}^{FOI}}{\bar{\mu}^S}, \forall h \in H, tbw \in TBW$$

where the dynamic evolution of $N_{\mathrm{h,tbw}}$ is governed by the force of infection ($\lambda_{h,tbw}^{FOI}$) corrected for the arrival rate of super-infections ($\bar{\lambda}_h^S$), and the clearance rate of super-infections ($\bar{\mu}^{\mathrm{S}}$). State variable difference equations are specified for each of our regional household types ($h \in H$) and each bi-weekly time period ($tbw \in TBW$). Based on a simple model of super-infection, where infected individuals are

assumed to harbour at least one clone (Dietz 1988), the human malaria prevalence rate ($p^H_{h,tbw}$) can be expressed, as specified in equation (3.2), as a simple monotonic function of the multiplicity of malaria infections ($N_{h,tbw}$):

(3.2) $$p^H_{h,tbw} = 1 - \exp(-N_{h,tbw}) \,, \forall h \in H, tbw \in TBW$$

Due to the above monotonic relationship, our state equation can be re-stated with $p^{\mathrm{H}}_{\mathrm{h,tbw}}$ as the state variable (derivation is included in section 3.4.2):

(3.1') $$p^H_{\mathrm{h,tbw+1}} = 1 - \left(1 - p^H_{\mathrm{h,tbw}}\right)^{\exp(-\bar{\mu}^S)} \exp\left(\left(\frac{\bar{\lambda}^S_h * \lambda^{FOI}_{h,tbw}}{\bar{\mu}^S}\right) * (\exp(-\bar{\mu}^S) - 1)\right) \,, \forall h \in H, tbw \in TBW$$

Either of the two state equations (3.1 or 3.1') can be used (in conjunction with the static equation 3.2) as the basis for our dynamic epidemiological model specification.

The above set of difference equations with discrete time steps governs the dynamic evolution of our epidemiological model. The discrete time step specification is particularly useful since the other sub-models also employ discrete time steps. The economic and demographic sub-models rely on annual time steps t (sections 2 and 4). However, this is not useful for our epidemiological model, since the malarial parasites and mosquito vectors have fairly short life cycles of around two weeks. Instead, it was decided to rely on bi-weekly discrete time intervals in the epidemiological model. The epidemiological model therefore solves for 26 bi-weekly time periods ($tbw \in TBW$) every time the other sub-models solve for one annual time period ($t \in T$).

The asymmetrical length of time periods has implications for the modelling of feedback effects between the epidemiological model, on the one hand, and the economic and demographic models, on the other. We decided to use the regional human malaria prevalence rates from the final 26th bi-weekly time period of each year ($p^H_{h,'tbw26'}$) as the $p^H_{h,t}$ value for each household strata ($h \in H$) and corresponding annual time period ($t \in T$). This value was also used as starting value for the $p^H_{h,tbw}$ state variable for the following year. Similarly, we decided to use the regional entomological inoculation rates from the final 26th bi-weekly period of each year ($EIR_{h,'tbw26'}$) as the basis for calculating annual regional clinical outcome indicators (see section 3.2, below), which are subsequently used as inputs in the annual macroeconomic and demographic models for each household strata ($h \in H$) and corresponding annual time period ($t \in T$).

The epidemiological model is known to converge relatively quickly towards equilibrium. Hence, the "final 26th bi-week" convention means that the annual demographic and macroeconomic simulations, de facto,

are based on equilibrium p_h^H and EIR values. This focus on "annual equilibrium" is consistent with our economic methodology, since the dynamically-recursive CGE model also solves recursively for annual equilibria.

The remaining part of the epidemiological model consists of static but time-dependent equations without time dynamics. In order to compute mosquito malaria prevalence rate ($p_{h,t}^M$), we employ a reduced-form static equilibrium specification (Smith and McKenzie 2004), presented in equation (3.3), where the prevalence of infectious mosquitoes is assumed to remain in equilibrium within a given period (but allowed to change between time periods with the human prevalence rate $p_{h,t}^H$):

$$(3.3) \quad p_{h,tbw}^M = \frac{\bar{a}*\bar{c}*p_{h,tbw}^H}{\mu_{h,t}^M+\bar{a}*\bar{c}*p_{h,tbw}^H} * \exp\left(-\mu_{h,t}^M * \bar{\tau}^{\text{incub}}\right), \forall h \in H, tbw \in TBW$$

This equilibrium specification of $p_{h,tbw}^M$ depends, in addition to $p_{h,tbw}^H$, on the human feeding rate of female mosquitoes ($\bar{a}$), the infectiousness of humans to mosquitoes ($\bar{c}$), the malaria parasite incubation period ($\bar{\tau}^{\text{incub}}$), and the mosquito mortality rate ($\mu_{h,t}^M$) which is an endogenous parameter since it is affected by endogenous coverage rates of malaria interventions (see equation (6.18) in section 6). Again, static time dependent equilibrium equations for mosquito malaria prevalence rates ($p_{h,tbw}^M$) are specified for each of our regional household types ($h \in H$).

Our static part of the model is closed by adding definitions of key indicators including the force of infection ($\lambda_{h,tbw}^{FOI}$) and the entomological inoculation rate ($EIR_{h,tbw}$) in questions (3.4)-(3.5):

$$(3.4) \quad \lambda_{h,tbw}^{FOI} = \bar{b} * EIR_{h,tbw}, \forall h \in H, tbw \in TBW$$

$$(3.5) \quad EIR_{h,tbw} = m_{h,t} * \bar{a} * p_{h,tbw}^M, \forall h \in H, tbw \in TBW$$

where the force of infection ($\lambda_{h,tbw}^{FOI}$) is the product of the infectiousness of mosquitoes to humans ($\bar{b}$) and the Entomological Inoculation Rate ($EIR_{h,tbw}$), and where $EIR_{h,tbw}$ is the product of the mosquito prevalence rate ($p_{h,tbw}^M$), the human feeding rate of female mosquitoes ($\bar{a}$) and the number of female mosquitoes per person ($m_{h,t}$) which is an endogenous parameter since it is affected by endogenous coverage rates of malaria interventions (see equation (6.19) in section 6).

A consideration was made to include, within the model, empirical modelling (estimation and parametrization) of the infectiousness of mosquitoes to humans ($\bar{b}$). Not every infective mosquito bite leads to a blood stage infection, but few systematic analyses of the relation between $\lambda_{h,tbw}^{FOI}$ and $EIR_{h,tbw}$ exist in the literature. Three candidate models were considered including the classical approach with

constant rate of infectiousness b, and two other models (Smith et al. 2006): (1) a model assuming saturation of $\lambda_{h,tbw}^{FOI}$ at high $EIR_{h,tbw}$, and (2) another model with additional development of pre-erythrocytic immunity upon repeated exposure, leading to a (potentially non-monotonic) reduction of the force of infection in older hosts. While preliminary results indicated that a model, such as that given in equation (3.6'), with variable rate of infectiousness ($b_{h,t}$) but without pre-erythrocytic immunity, was most consistent with underlying data, it was decided that the statistical basis was not sufficiently developed to include this specification within the current model framework:

(3.6') $$b_{h,tbw} = b^{min} + \frac{\left(1-\bar{b}^{min}\right)}{\left(1+\frac{EIR_{h,tbw}}{\overline{eir}^{b}}\right)}, \forall h \in H, tbw \in TBW$$

In principle, our epidemiological model measures the true human malaria prevalence ($p_{h,tbw}^{H}$), i.e. the proportion of people harbouring detectable or undetectable parasites. Such a variable would not be directly comparable to normal measures of observed malaria prevalence, detectable by microscopy or PCR-based methods. Recent research has made advances into estimation of detectability relations (Bretscher et al. 2010). In order to produce a comparable variable of expected human malaria prevalence rate detectable by microscopy, we experimented with including, within the model, two additional empirical equations (3.7')-(3.8') which measure respectively the correction factor for detectability of malaria infection by microscopy ($q_{h,t}$) as a function of the Entomological Inoculation Rate ($EIR_{h,tbw}$), and the observed human malaria prevalence rate, also known as slide prevalence ($sp_{h,tbw}^{H}$):

(3.7') $$q_{h,tbw} = \bar{q}^{max} + \frac{\left(\bar{q}^{min}-\bar{q}^{max}\right)}{\left(1+\frac{\overline{eir}^{q}}{EIR_{h,tbw}}\right)}, \forall h \in H, tbw \in TBW$$

(3.8') $$sp_{h,tbw}^{H} = q_{h,t} * p_{h,tbw}^{H}, \forall h \in H, tbw \in TBW$$

In the end, we also decided to exclude specifications (3.7')-(3.8') due to insufficient statistical basis.

Finally, for the purposes of analysing the potential for future malaria elimination, we employed a model-consistent formula for calculating basic reproduction numbers (R_h^0) (Smith & McKenzie 2004) to derive household-specific relationships for control reproduction numbers ($R_{h,tbw}^{C}$):

(3.9) $$R_h^0 = \frac{\bar{m}_h * a^2 * b * c}{\bar{\mu}_h^M * \bar{\mu}^S}, \forall h \in H$$

(3.10) $$R_{h,tbw}^{C} = \frac{m_{h,tbw} * a^2 * b * c}{\mu_{h,tbw}^M * \bar{\mu}^S}, \forall h \in H, tbw \in TBW$$

where ($\bar{m}_h$, $\bar{\mu}_h^M$) are epidemiological parameter values associated with 0% coverage rates of preventive interventions (see section 6.2 for further discussion).

For presentational purposes, the implementation of our epidemiological simulation model includes the human malaria prevalence rate ($p_{h,tbw}^H$) as state variable. Hence, our simulated model includes the following equations: (3.1'), (3.2), (3.3), (3.4), (3.5), and (3.10). This set of equations includes one state variable equation (3.1') for $p_{h,tbw}^H$ and five static time dependent model equations without dynamics for the determination of the remaining five endogenous variables: the multiplicity of infections ($N_{h,tbw}$), mosquito malaria prevalence rate ($p_{h,t}^M$), the force of infection ($\lambda_{h,tbw}^{FOI}$), the entomological inoculation rate ($EIR_{h,tbw}$), and the controlled reproduction number ($R_{h,tbw}^C$).

Distinct epidemiological models were calibrated for each of the 19 regional households in our integrated model framework, and a full account of the region-specific calibrated parameter values are presented in section 3.3.

3.2. Clinical outcome specifications

It is well-known that clinical outcomes from *P. falciparum* infection are intimately related to the intensity of transmission measured by the Entomological Inoculation Rate. E.g. it has been argued that "there is strong evidence both from molecular typing and from patterns of seasonality in morbidity that clinical malaria is normally caused by newly invading parasites, and the most severe symptoms generally accompanying the first peak of parasite density after infection. It follows that in the short term, any reduction in EIR will decrease the incidence of clinical episodes in proportion to the effect on the force of infection" (Smith, Killeen et al. 2004). However, the literature also generally finds that "Reductions in transmission intensity … also reduce immunologic stimulation, and this may have longer term effects, in particular resulting in shifts of the peak in the age incidence profiles to older ages…" (ibid.) It was therefore decided to include an empirical relation which links age-specific clinical outcomes to EIR-levels, and, thereby, allows for measuring household- and region-specific clinical outcomes based on endogenously determined EIR-levels (and the age composition of households).

We chose to model the relation between EIR and age-specific clinical outcomes through a set of piece-wise linear specifications based on simulated lookup tables. The Swiss Tropical Institute (STI) model (Smith et al. 2008) is a well-known and reputable epidemiological model, which allows for capturing non-linearities in the relation between EIR-levels and age-specific clinical outcomes. We decided to use this model to simulate a set of eight lookup tables for eight distinct EIR-values (see equations (3.9)-(3.10); empirical characteristics of the parametrization is discussed in section 3.3.) Our baseline choice of piece-wise linear

specifications based on lookup tables (as well as the choice of simulating eight lookup tables) was made, in an attempt to capture the underlying non-linearities within a parsimonious specification. For modelling purposes and in order to allow for sensitivity analyses, we also include an alternative set of continuous polynomial approximations, which are fitted to mirror the piece-wise linear STI lookup table specifications (see equations (3.9')-(3.10')). The modelling of malaria-related clinical outcomes is focussed on two measures: (1) morbidity as proxied by the age-specific number of uncomplicated malaria episodes per person per year ($\tau_{h,age,t}$), and (2) mortality as measured by age-specific excess mortality rates ($\mu_{h,age,t}$).[1]

The actual specification of the piece-wise linear functions for the two clinical outcome measures relied, specifically, on two sets of age-specific lookup tables, including (1) the number of uncomplicated malaria episodes per person per year ($Lookup^{\tau}_{age,l}$), and (2) the excess mortality risk per person per year ($Lookup^{\mu}_{age,l}$), tabulated at eight different equidistant $\log_{10}$-linearized EIR values with intervals of 0.5 ($l \in L$). The lookup table parameters were used as the basis for our piece-wise linear interpolation specification (and, hence, modelling of) household- and region-specific numbers of uncomplicated malaria episodes per person per year ($\tau_{h,age,t}$) and excess malaria-related mortality rate ($\mu_{h,age,t}$).

In order to implement piece-wise linear specifications, endogenously, within our model framework, we developed a family of functional forms, presented in equations (3.9)-(3.10), which are suitable for our purposes of modelling clinical outcomes over an (EIR) biomarker with bounded support set (L):

$$\tau_{h,age,t}\left(EIR_{h,t}\right) = \bar{\alpha}^{\tau} * \sum_{l=\underline{L}}^{\overline{L}-1} \prod_{m \neq l} \frac{\left(\left[\frac{f\left(EIR_{h,t}\right)}{\Delta}\right]\Delta - m\right)}{l-m} * \left(\left(\frac{f\left(EIR_{h,t}\right)}{\Delta} - \left[\frac{f\left(EIR_{h,t}\right)}{\Delta}\right]\right)\overline{Lookup}^{\tau}_{age,l} + \left(\left[\frac{f\left(EIR_{h,t}\right)}{\Delta} + 1\right] - \frac{f\left(EIR_{h,t}\right)}{\Delta}\right)\overline{Lookup}^{\tau}_{age,l+1}\right), \forall h \in H, age \in AGE, t \in T \quad (3.9)$$

$$\mu_{h,age,t}\left(EIR_{h,t}\right) = \bar{\alpha}^{\mu} * \sum_{l=\underline{L}}^{\overline{L}-1} \prod_{m \neq l} \frac{\left(\left[\frac{f\left(EIR_{h,t}\right)}{\Delta}\right]\Delta - m\right)}{l-m} * \left(\left(\frac{f\left(EIR_{h,t}\right)}{\Delta} - \left[\frac{f\left(EIR_{h,t}\right)}{\Delta}\right]\right)\overline{Lookup}^{\mu}_{age,l} + \left(\left[\frac{f\left(EIR_{h,t}\right)}{\Delta} + 1\right] - \frac{f\left(EIR_{h,t}\right)}{\Delta}\right)\overline{Lookup}^{\mu}_{age,l+1}\right), \forall h \in H, age \in AGE, t \in T \quad (3.10)$$

where f(.) is a random transformation, $\underline{L}$ and $\overline{L}$ represents lower and upper bounds for f(.), and Δ represents the (constant) distance between lookup table f(.)-values. In our case, we simulated lookup tables for eight equidistant $\log_{10}$-linearized EIR values with intervals of 0.5, implying that $f(EIR_{h,t}) =$

[1] Additional clinical outcomes available from the STI model simulations include (1) rate of severe malaria cases, and (2) rate of neurological sequelae cases. The latter clinical outcomes were not included in the current model specification.

$log_{10}(EIR_{h,t})$, Δ=0.5, $\underline{L} = -1$ and $\overline{L} = +2.5$. EIR biomarker values are typically found to range from 0.1 to 100. We therefore chose to define our support set to be $EIR \in [0.1; 316]$ implying that application of the above-mentioned closed-form piece-wise linear functional specifications allows for endogenous calculation of $\tau_{h,age,t}$ and $\mu_{h,age,t}$ clinical outcomes over the entire (required) support set. Critically, the above family of functional forms is also suitable for implementation with the "Nonlinear Programming with Discontinuous Derivatives" (DNLP) solver in our preferred computer program, GAMS.

The scaling parameters in the above specifications (α^{τ}, α^{μ}) also allow for scaling of our clinical outcome specifications to match observed base year clinical outcomes. The scalar nature of the scaling factors implies that (1) the STI clinical outcome levels will be benchmarked to our Ghana-specific country context, but also that (2) the STI clinical outcome patterns across age groups will be retained. Due to (a) country-specific circumstances such as prevention and treatment intervention levels, (b) limitations due to local health system capacity constraints, partial roll-out of RDT and microscopy testing, and limitations in detectability of malaria infections, and (c) infrequent and geographically limited surveys of malaria-related clinical outcomes, there is great uncertainty about the true numbers of malaria cases and malaria-related deaths in Ghana. This is reflected in a relatively erratic set of recent trend estimates of suspected malaria cases in Ghana. Nevertheless, benchmarking is considered to be essential (see discussion in section 3.3).

For technical reasons, and to allow for sensitivity analyses of our piece-wise linear specification, we supplemented the piece-wise linear specifications by several alternative sets of polynomial approximations. The polynomial approximations have a maximum order of seven, but we aim to apply fifth order polynomial approximations when undertaking sensitivity analyses. Specifications are provided in equations (3.9')-(3.10'):

(3.9') $$\tau_{h,age,t}\left(EIR_{h,t}\right) = \bar{\alpha}^{\tau} * \left(\bar{\beta}_{age}^{\tau,1} * EIR_{h,t} + \ \ldots + \bar{\beta}_{age}^{\tau,s} * EIR_{h,t}^{s}\right),$$

$$\forall h \in H, age \in AGE, t \in T, s \leq 7$$

(3.10') $$\mu_{h,age,t}\left(EIR_{h,t}\right) = \bar{\alpha}^{\mu} * \left(\bar{\beta}_{age}^{\mu,1} * EIR_{h,t} + \ \ldots + \bar{\beta}_{age}^{\mu,s} * EIR_{h,t}^{s}\right),$$

$$\forall h \in H, age \in AGE, t \in T, s \leq 7$$

Technically, the continuous specifications are useful, since they allow us to use our preferred (and more efficient) Mixed Complementarity (MCP) numerical algorithm, to solve our integrated model in GAMS. Nonetheless, in order to retain as much information as possible from the original STI lookup tables (and to avoid problems associated with potential fitting of negative clinical outcomes), we only aim to employ the

polynomial approximations for model development[2] and sensitivity analysis purposes (in order to investigate whether future applications can usefully restrict attention to continuous polynomial specifications).

3.3. Parametrization

In line with the macroeconomic calibration methodology (section 2), the epidemiological model was calibrated to an equilibrium solution ($p^H_{h,tbw+1} = p^H_{h,tbw} = \bar{p}^H_{h,tbw_0}$). The calibration of exogenous parameters and initialization of variables and endogenous parameters in the epidemiological model for *P. falciparum* transmission relied on exogenous household-specific information about two variables: human prevalence rates ($\bar{p}^H_{h,tbw_0}$) and Entomological Inoculation Rates ($\overline{EIR}_{h,tbw_0}$); and exogenous information about five parameters: the human feeding rate of female mosquitoes ($\bar{a}$), the infectiousness of infective mosquito bites ($\bar{b}$), the infectiousness of humans to mosquitoes ($\bar{c}$), the mosquito mortality rate ($\bar{\mu}^M_h$), the malaria parasite incubation period ($\bar{\tau}^{\text{incub}}$).

Combined with the epidemiological model specification, reviewed in section 3.1, the above exogenous information on initial parameter and variable values was sufficient to calibrate the remaining exogenous parameter and initialize the remaining five variables. One calibrated parameter: the arrival rate of super-infections ($\bar{\lambda}^{\text{S}}$); and five initialized variables: the number of female mosquitoes per person ($\overline{m}_{h,tbw_0}$), the multiplicity of malaria infections ($\overline{N}_{h,tbw_0,}$), the mosquito malaria prevalence rate ($\bar{p}^M_{h,tbw_0}$), the force of infection ($\bar{\lambda}^{FOI}_{h,tbw_0}$), and the control reproduction number ($\bar{R}^C_{h,tbw_0}$).

Table 3.1 presents the full set of household- and region-specific calibrated and non-calibrated parameters and variables from our 19 epidemiological models. Uniform values were imposed for the five exogenous parameters: The human feeding rate ($\bar{a}$) was set at 0.67 day^{-1} (Filipe et al. 2007), the infectiousness of infective mosquito bites ($\bar{b}$) was set at 0.25 (Filipe et al. 2007), the infectiousness of humans to mosquitoes ($\bar{c}$) was set at 0.05 day^{-1} (Kileen, Ross & Smith 2006), and the incubation period ($\bar{\tau}^{\text{incub}}$) was set at 10 days (Gu et al. 2003), the clonal clearance rate ($\bar{\mu}^{\text{S}}$) was set at 0.078 (=14/180), based on our bi-weekly time interval and an assumed 180 day parasite survival time (Filipe et al. 2007)[3], while the mosquito mortality

[2] In addition, the MCP-solver automatically checks whether our system of equations (the CGE model) is square, and thereby provides an important check in model-development, something which is not available with the non-linear optimization solvers.

[3] Our assumption of a 180 day parasite survival time was set, conservatively, relative to the mean (211.6 days) and the median (215.5 days) survival times observed by Sama, Dietz & Smith (2006).

rate ($\bar{\mu}_h^M$) was set at 1/10 days^{-1}, based on an estimated 10 day average mosquito lifespan derived from a small survey of available empirical evidence (Filipe et al. 2007).[4]

In contrast to the uniform values imposed on our non-calibrated parameters, detailed household- and region-specific values were derived and imposed on our non-calibrated variables: human prevalence rates ($\bar{p}_h^H$) and Entomological Inoculation Rates ($\overline{EIR}_h$). Since level-differences in $\bar{p}_h^H$ and $\overline{EIR}_h$ are critical for measuring regional differences in malaria disease burdens, the household- and region-disaggregation of our data sets and models was defined by our ability to regionally disaggregate $\bar{p}_h^H$ and $\overline{EIR}_h$, and to link this up with our underlying 2005/06 GLSS5 household survey data set (GSS 2008).

Table 3.1. Household-specific epidemiological model parameters and initial variable values

	non-calibrated parameters						Initial variable values$		Calibrated parameters*		Calibrated variable values*			
	a†	b†	c‡	μ^M†	μ^S†	τ^{INCUB}#	EIR$	$p^H$$	m	λ^S	N	p^M	λ^{FOI}	R^C
H01	0.67	0.25	0.05	0.10	0.08	10.00	0.39	0.14	36.29	0.117	0.15	0.02	0.10	26.18
H02	0.67	0.25	0.05	0.10	0.08	10.00	0.84	0.25	43.51	0.108	0.29	0.03	0.21	31.39
H03	0.67	0.25	0.05	0.10	0.08	10.00	2.00	0.37	73.88	0.071	0.46	0.04	0.50	53.30
H04	0.67	0.25	0.05	0.10	0.08	10.00	8.58	0.52	235.72	0.026	0.73	0.05	2.15	170.06
H05	0.67	0.25	0.05	0.10	0.08	10.00	0.95	0.28	45.37	0.106	0.32	0.03	0.24	32.74
H06	0.67	0.25	0.05	0.10	0.08	10.00	1.67	0.34	65.84	0.078	0.42	0.04	0.42	47.50
H07	0.67	0.25	0.05	0.10	0.08	10.00	5.10	0.46	153.97	0.038	0.62	0.05	1.28	111.08
H08	0.67	0.25	0.05	0.10	0.08	10.00	0.84	0.26	42.58	0.111	0.30	0.03	0.21	30.72
H09	0.67	0.25	0.05	0.10	0.08	10.00	2.39	0.38	84.84	0.063	0.49	0.04	0.60	61.21
H10	0.67	0.25	0.05	0.10	0.08	10.00	9.91	0.52	270.06	0.023	0.74	0.05	2.48	194.83
H11	0.67	0.25	0.05	0.10	0.08	10.00	1.15	0.29	51.80	0.095	0.35	0.03	0.29	37.37
H12	0.67	0.25	0.05	0.10	0.08	10.00	2.50	0.38	88.96	0.060	0.49	0.04	0.63	64.18
H13	0.67	0.25	0.05	0.10	0.08	10.00	12.86	0.56	328.72	0.020	0.83	0.06	3.21	237.15
H14	0.67	0.25	0.05	0.10	0.08	10.00	1.40	0.32	58.51	0.086	0.39	0.04	0.35	42.21
H15	0.67	0.25	0.05	0.10	0.08	10.00	3.08	0.41	103.44	0.053	0.53	0.04	0.77	74.63
H16	0.67	0.25	0.05	0.10	0.08	10.00	8.97	0.52	243.73	0.026	0.74	0.05	2.24	175.84
H17	0.67	0.25	0.05	0.10	0.08	10.00	1.36	0.32	56.84	0.089	0.39	0.04	0.34	41.00
H18	0.67	0.25	0.05	0.10	0.08	10.00	3.66	0.42	120.04	0.046	0.55	0.05	0.91	86.60
H19	0.67	0.25	0.05	0.10	0.08	10.00	17.83	0.59	435.84	0.016	0.90	0.06	4.46	314.43

Sources: †Filipe et al. (2007); ‡Kileen, Ross & Smith 2006; #Gu at al. (2003); $Gething at al. (2011); *Own calculations

Detailed maps of 2010 *P. falciparum* values for $\bar{p}_{h,tbw_0}^H$ and $\overline{EIR}_{h,tbw_0}$ at pixel levels of 5x5km were obtained from the Malaria Atlas Project (MAP) (Gething et al. 2011). These maps were manipulated, through use of the ArcGIS program and the overlaying of an own-developed map of 2005/06 Ghana districts (mirroring the basic sampling units of GLSS5), to allow derivation of average 2010 district-level values of $\bar{p}_{h,tbw_0}^H$ and $\overline{EIR}_{h,tbw_0}$ (based on the 2005/06 Ghana district classification). The subsequent categorisation of districts according to low, medium and high levels of $\bar{p}_{h,tbw_0}^H$ allowed for the definition

[4] A small survey of available empirical evidence suggests that *Anopheles Gambiae* average lifespans may vary strongly between 3.6-20 (see appendix A in Chitnis, Hyman & Cushing; 2008). However, based on other evidence (Filipe et al. 2007), we chose a conservative 10 day average mosquito lifespan which implies a daily mosquito mortality rate of 0.10 day^{-1}.

and aggregation of our 19 household types. Finally, the ability for us to link both the 2010 MAP data and the 2005/06 GLSS5 household survey data to the same underlying sampling unit, allowed us to establish a fully consistent epidemiological and economic (SAM) data set with distinct epidemiological and income/expenditure patterns for each of our 19 distinct household types (see section 2 for additional discussion). The average $\bar{p}^{H}_{h,tbw_0}$ and $\overline{EIR}_{h,tbw_0}$ values are given in Table 3.1.[5]

The lookup tables for measurement of clinical outcomes from *P. falciparum* infection were simulated and derived from the Swiss Tropical Institute (STI) model (Smith et al. 2008). As discussed above (section 3.2), clinical outcomes are generally considered to be driven by malaria transmission intensity, proxied by EIR, but, due to (not fully understood) immunization issues, the relationship is also considered to vary by age-group. It was therefore decided to simulate age-specific lookup tables for single year age categories between ages 0-68 (morbidity and mortality rates for ages 69+ were assumed to be similar to the 68 year olds).

Clinical outcomes are also generally thought to vary with the inter-annual variation in transmission intensity. It was therefore decided to simulate separate age-specific lookup tables for four different levels of seasonality in malaria transmission including 5 months, 6 months, 7 months, and 12 months (all year) transmission. Each of our 19 household types were subsequently categorized into a given seasonality group based on information about the length of agricultural growing seasons by ecological region (Oppong-Anane 2006). The household-specific seasonality assumptions are outlined in Table 3.2.

As already explained above, the current model specification employs two sets of 'lookup table' parameters, including (1) the average numbers of uncomplicated malaria episodes per person per year ($\overline{Lookup}^{\tau}_{age,l}$), and (2) the excess mortality risk per person per year ($\overline{Lookup}^{\mu}_{age,l}$), tabulated at eight different equidistant $\log_{10}$-linearized EIR values with intervals of 0.5 (see section 3.2).[6] Surface figures of the lookup tables for morbidity ($\overline{Lookup}^{\tau}_{age,l}$) and excess mortality ($\overline{Lookup}^{\mu}_{age,l}$) associated with *P. falciparum* infection are provided in respectively Figures 3.1 and 3.2 (for 12 month all year transmission).[7]

[5] It should be noted that prevalence data from the MAP data refer to 2-10y age groups. In our model calibration, we assume that these age-specific prevalence rates extend to the broader population. In this context, it should, however, be noted that all clinical outcomes are measured on the basis of household-specific EIR-values. The specification of age-specific prevalence rates is therefore likely to have only minor importance for the measurement of economic impacts and the overall economic disease burden.

[6] Additional sets of lookup tables could be constructed for other clinical outcomes, available from our STI model simulations (see also footnote 1), including (1) rate of severe malaria cases, and (2) rate of neurological sequelae cases. This option was, however, not pursued for the current model specification.

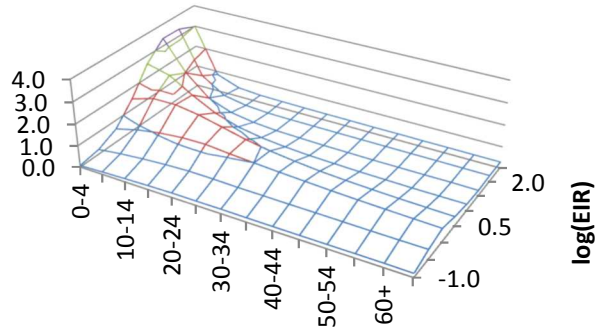
[7] The qualitative nature of the clinical outcome surface figures is robust to variation in seasonality patterns.

Table 3.2. Household-specific seasonality in malaria transmission

Household	Household label	Agricultural growing season[a]	Assumed transmission season
H01	Low prevalence GAMA	250-260 days	12 months
H02	Low prevalence Urban Coastal	250-260 days	12 months
H03	Med prevalence Urban Coastal	250-260 days	12 months
H04	HIgh prevalence Urban Coastal	250-260 days	12 months
H05	Low prevalence Urban Forest	250-260 days	12 months
H06	Med prevalence Urban Forest	250-260 days	12 months
H07	HIgh prevalence Urban Forest	250-260 days	12 months
H08	Low prevalence Urban Savannah	150-200 days	7 months
H09	Med prevalence Urban Savannah	150-200 days	7 months
H10	HIgh prevalence Urban Savannah	150-200 days	7 months
H11	Low prevalence Rural Coastal	250-260 days	12 months
H12	Med prevalence Rural Coastal	250-260 days	12 months
H13	HIgh prevalence Rural Coastal	250-260 days	12 months
H14	Low prevalence Rural Forest	250-260 days	12 months
H15	Med prevalence Rural Forest	250-260 days	12 months
H16	HIgh prevalence Rural Forest	250-260 days	12 months
H17	Low prevalence Rural Savannah	150-200 days	7 months
H18	Med prevalence Rural Savannah	150-200 days	7 months
H19	HIgh prevalence Rural Savannah	150-200 days	7 months

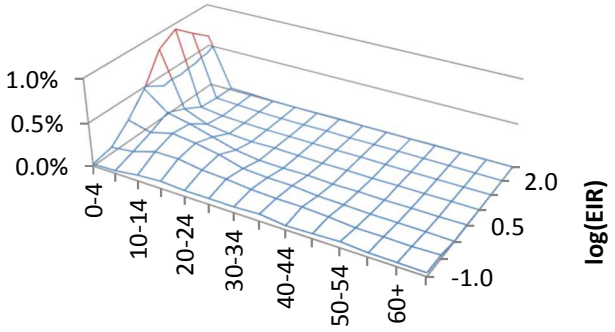
Source: [a]Oppong-Anane (2006); NB: Coastal areas assumed to be belong to transition zones.

Figure 3.1. Malaria morbidity (uncomplicated episodes per person) by 5-year age group and EIR transmission intensity



Source: own simulations of Swiss Tropical Institute model

Figure 3.2. Malaria mortality rates by 5-year age group and EIR transmission intensity



Source: own simulations of Swiss Tropical Institute model

The scaling parameters ($\bar{\alpha}^{\tau}, \bar{\alpha}^{\mu}$) in the piece-wise linear clinical outcome specifications (equations (3.9)-(3.10)) were calibrated to benchmark the simulated STI clinical outcome levels to 2010 Ghana-specific clinical outcomes: (1) 3,694,671 uncomplicated cases, and (2) 1.44% case fatality rate (GHS 2011). The calibration resulted in an (uncomplicated episodes) morbidity scaling factor of 0.152 and an (excess) mortality scaling factor of 1.54 (Table 3.3). Without benchmarking, the raw simulated STI lookup tables would have overestimated Ghana-specific uncomplicated malaria episodes by a factor of 8, and underestimated Ghana-specific excess mortality by one-third.

Table 3.3. 2010 clinical outcome benchmark measures and scaling parameters

	Uncomplicated malaria cases	Case fatality rate
2010 benchmark measures[a]	3,694,671	1.44%
	Uncomplicated malaria episodes ($\alpha^{\tau,SCALE}$)	Excess mortality ($\alpha^{\mu,SCALE}$)
Scaling-parameters[b]	0.152	1.540

Source: [a]GHS (2011); [b]own calculations.

The parameters ($\bar{\beta}^{\tau,s}_{h,age}, \bar{\beta}^{\mu,s}_{h,age}$) for our household- and age-specific polynomial approximations were, subsequently, fitted to our simulated STI lookup tables based on simple standard minimum squared deviation distance metrics.

3.4. Derivation of epidemiological model state variable equations

3.4.1. Derivation of state variable equation for multiplicity (N)

The state variable equation (3.1) for the multiplicity of super-infections ($N_{h,tbw}$) is derived as the general solution to the Poisson distributed stochastic process for $N_{h,tbw}$, i.e. derived from the reversible catalytic model specification of super-infections as given in equation (3.11):

(3.11) $$\frac{dN_{h,tbw}}{dt} = \lambda^{FOI}_{h,tbw} - \bar{\mu}^{S} * N_{h,tbw}\ , \forall h \in H, tbw \in TBW$$

$$\Updownarrow$$

(3.1) $$N_{h,tbw+1} = \left(N_{\mathrm{h,tbw}} - \frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^{S}}\right) * \exp\left(-\bar{\mu}^{\mathrm{S}}\right) + \frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^{S}}\ , \forall h \in H, tbw \in TBW$$

3.4.2. Derivation of state variable equation for true human prevalence (p^{H})

The human prevalence rate $p^{H}_{\mathrm{h,tbw}}$ is equal, as specified in equation (3.12), to the Poisson probability of having at least one (super-)infection ($N_{h,tbw}$), and this relationship allows us, as specified in in equation (3.13), to express $N_{h,tbw}$ in terms of $p^{H}_{\mathrm{h,tbw}}$:

(3.12) $$p^{H}_{\mathrm{h,tbw}} = 1 - \exp\left(-N_{h,tbw}\right)\ , \forall h \in H, tbw \in TBW$$

$$\Updownarrow$$

(3.13) $$N_{h,tbw} = -\log_e\left(1 - p^H_{h,tbw}\right), \forall h \in H, tbw \in TBW$$

The state variable equation (3.1') for $p^H_{h,tbw}$ can now be derived by substituting expression (3.13) into the state variable equation (3.1) for $N_{h,tbw}$:

(3.1) $$N_{h,tbw+1} = \left(N_{\mathrm{h,tbw}} - \frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^S}\right) * \exp\left(-\bar{\mu}^{\mathrm{S}}\right) + \frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^S}, \forall h \in H, tbw \in TBW$$

$$\Updownarrow$$

$$-\log_e\left(1 - p^H_{h,tbw+1}\right) = \left(-\log_e\left(1 - p^H_{h,tbw}\right) - \frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^S}\right) * \exp\left(-\bar{\mu}^{\mathrm{S}}\right) + \frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^S}$$

$$\Updownarrow$$

(3.1') $$p^H_{\mathrm{h,tbw+1}} = 1 - \left(1 - p^H_{\mathrm{h,tbw}}\right)^{\exp\left(-\bar{\mu}^S\right)} \exp\left(\left(\frac{\lambda^{FOI}_{h,tbw}}{\bar{\mu}^S}\right) * \left(\exp(-\bar{\mu}^S) - 1\right)\right), \forall h \in H, tbw \in TBW$$

4. Demographic model equations and parametrization

In this section, we specify a full set of annual demographic models stratified over 1-year age groups ([0-100]), two gender types and our 19 malaria-focussed household types (section 4.1), and we also include a consistently stratified set of wage-driven interregional and international migration specifications based on Harris-Todaro migration specifications (section 4.2). All demographic specifications are parameterized in section 4.3, while endogenous variables and exogenous parameters are listed and defined in Annex C.

4.1. Household- and region-specific demographic models

4.1.1. Aggregate population

The demographic module keeps track of both aggregate and disaggregate Ghanaian population groups ($POP^{H}_{h,t}$, $POP_{h,gen,age,t}$) as indicated by equation (4.1):

(4.1) $$POP^{H}_{h,t} = \sum_{gen,age} POP^{disagg}_{h,gen,age,t} \ , \forall h \in H, gen \in GEN, age \in AGE, t \in T$$

where the disaggregated population groups ($POP^{disagg}_{h,gen,age,t}$) are stratified over 19 household categories ($H = [h01; h19]$), two gender categories ($GEN = \{male, female\}$), 101 one year age groups ($AGE = [0; 100]$), and 20 time periods ($T = [2015; 2034]$).

4.1.2. Non-infant population

Disaggregated non-infant population levels for a given population strata ($POP_{h,gen,age,t}$) is, as specified in equation (4.2), defined as the sum of (1) the population level of the previous age group in the previous year ($POP_{h,gen,age-1,t-1}$) corrected for (2) current deaths ($Deaths_{h,gen,age,t}$) and (3) the change in current net emigration levels ($\Delta POP^{emigr,net}_{h,gen,age,t}$):

(4.2) $$POP_{h,gen,age,t} = POP_{h,gen,age-1,t-1} - Deaths_{h,gen,age,t} - \Delta POP^{emigr,net}_{h,gen,age,t},$$

$$\forall h \in H, gen \in GEN, age \in AGE|_{age \neq 0}, t \in T$$

The key link between epidemiological and demographic outcomes, in our model, is the clinical outcome impact on death rates (see section 3.2). The disaggregated number of deaths in a given population strata are determined by age-specific mortality rates. 'Baseline all-cause mortality rates' ($\bar{\mu}^{all\ cuase}_{h,gen,age,t}$) are calibrated from all-cause mortality levels in the broader population (see section 4.3 below). This implicitly includes 'baseline malaria mortality levels'. In our model, simulated all-cause mortality rates only deviate from 'baseline all-cause mortality rates', when simulated malaria excess mortality rates ($\mu_{h,age,t}(EIR_{h,t})$)

deviate from 'baseline malaria excess mortality rates' ($\bar{\mu}_{h,age,t} = \mu_{h,age,t}(\overline{EIR}_h)$). The number of deaths for a given population strata ($Deaths_{h,gen,age,t}$) is therefore given by equation (4.3):

(4.3) $$Deaths_{h,gen,age,t} = \left(\bar{\mu}^{all\ cause}_{h,gen,age,t} - \left(\mu_{h,age,t}(EIR_{h,t}) - \bar{\mu}_{h,age,t}\right)\right) * POP_{h,gen,age-1,t-1},$$

$$\forall h \in H, gen \in GEN, age \in AGE|_{age\neq 0}, t \in T$$

4.1.3. Infant population

The infant population is defined, in equation (4.4), as the number of births ($Births_{h,gen,t}$) net of infant deaths ($InfDeaths_{h,gen,t}$):

(4.4) $$POP_{h,gen,age,t} = Births_{h,gen,t} - InfDeaths_{h,gen,t},$$

$$\forall h \in H, gen \in GEN, age \in AGE|_{age\neq 0}, t \in T$$

Gender-specific births ($Births_{h,gen,t}$) are modelled, in equation (4.5), by applying gender birth ratios ($sexratio_{gen}$) and age-specific fertility rates ($asfr_{age}$) to the female population in fertile age groups (15-49):

(4.5) $$Births_{h,gen,t} = sexratio_{gen} * \sum_{(gen,age)|gen='female',age\in[15;49]} asfr_{age} * POP_{h,gen,age,t},$$

$$\forall h \in H, gen \in GEN, t \in T$$

while gender-specific infant deaths ($InfDeaths_{h,gen,t}$) for a specific generation are modelled, in equation (4.6):

(4.6) $$InfDeaths_{h,gen,t} = \bar{\mu}^{all\ cuase}_{h,gen,age,t|age=0} * Births_{h,gen,t}, \forall h \in H, gen \in GEN, t \in T$$

4.2. Regional migration specifications

4.2.1. Overall household net migration

Household-specific net immigration ($POP^{migr,net}_{h,gen,age,t}$) is defined, in equation (4.7), as the sum of domestic net immigration ($\Delta POP^{dmigr,net}_{h,gen,age,t}$) and international net immigration ($\Delta POP^{imigr,net}_{h,gen,age,t}$):

(4.7) $$POP^{migr,net}_{h,gen,age,t} = \Delta POP^{dmigr,net}_{h,gen,age,t} + \Delta POP^{imigr,net}_{h,gen,age,t}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$$

International net immigration ($\Delta POP^{imigr}_{h,gen,age,t}$) is, in turn, defined, in equation (4.8), as household-specific immigration ($\Delta POP^{imigr}_{h,gen,age,t}$) net of household-specific emigration ($\Delta POP^{emigr}_{h,gen,age,t}$):

(4.8) $$POP^{imigr,net}_{h,gen,age,t} = \Delta POP^{imigr}_{h,gen,age,t} + \Delta POP^{emigr}_{h,gen,age,t}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$$

4.2.2. International Immigration

International immigration flows ($\Delta POPshr^{imigr}_{h,gen,age,t}$) are computed from modelling of aggregate rural-urban immigrant population stock shares ($POPshr^{imigr,LOC2}_{loc2,t}$) and household-specific immigrant population stocks ($POP^{imigr,H}_{h,t}$). Rural-urban immigrant stock shares are modelled, in equation (4.9), as functions of lagged domestic-to-international wage differentials, based on Harris-Todaro migration specifications:

(4.9) $$ln\left(POPshr^{imigr,LOC2}_{loc2,t}\right) = \alpha^{imigr,LOC2}_{loc2} + \sigma^{imigr,LOC2}_{loc2} * \ln\left(\frac{WF^{LOC2}_{loc2,t-1}}{WF^{ROW}_{t-1}}\right), \forall loc2 \in LOC2, t \in T$$

where the aggregate rural-urban immigrant population stock shares ($POPshr^{imigr,LOC2}_{loc2,t}$) are stratified over two aggregate rural-urban household categories ($LOC2 = \{rural, urban\}$), and where the two location rural/urban ("LOC2") and Rest of the World ("ROW") wage indices ($WF^{LOC2}_{loc2,t}$, WF^{ROW}_{t}) are defined in equations (4.22)-(4.23) below. Parameters include scale parameters ($\alpha^{imigr,LOC2}_{loc2}$) and relative wage-elasticities of immigration ($\sigma^{imigr,LOC2}_{loc2}$).

The aggregate rural-urban immigrant population shares ($POPshr^{imigr,LOC2}_{loc2,t}$) are subsequently multiplied by lagged regional household population totals ($POP_{h,t-1}$), to compute, in equation (4.10), current aggregate immigrant population stocks for our 19 regional household types ($POP^{imigr,H}_{h,t}$):

(4.10) $$POP^{imigr,H}_{h,t} = POPshr^{imigr,LOC2}_{loc2,t|maphloc(h,loc2)} * POP^{H}_{h,t-1}, \forall h \in H, t \in T$$

where the $maphloc(h, loc2)$-mapping maps our 19 household types into rural and urban categories.

The aggregate household-specific immigrant population stocks ($POP^{imigr,H}_{h,t}$) are further disaggregated, in equation (4.11), into age- and gender-specific immigrant population stocks ($POP^{imigr}_{h,gen,age,t}$) based on fixed age- and gender-specific immigrant patterns ($\beta^{imigr}_{h,gen,age}$):

(4.11) $$POP^{imigr}_{h,gen,age,t} = \beta^{imigr}_{h,gen,age} * POP^{imigr,H}_{h,t}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$$

where $\sum_{gen,age} \beta^{imigr}_{h,gen,age} = 1, \forall h \in H$.

Finally, current age- and gender-specific immigration flows are defined, in equation (4.12), as the net increase in household-specific immigrant population stocks:

(4.12) $$\Delta POP^{imigr}_{h,gen,age,t} = POP^{imigr}_{h,gen,age,t} - POP^{imigr}_{h,gen,age-1,t-1}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$$

4.2.3. International Emigration

Our modelling of international emigration flows ($\Delta POP^{emigr}_{h,gen,age,t}$) is equivalent to our modelling of international immigration flows (see section 4.2.2 above). Hence, international emigration flows are again computed from modelling of aggregate emigrant population stocks ($POP^{emigr,H}_{h,t}$), and these stock are derived from rural-urban emigrant stock shares ($POPshr^{emigr,LOC2}_{loc2,t}$) which are again modelled, in equation (4.13), as functions of lagged domestic-to-international wage differentials, based on Harris-Todaro migration specifications:

(4.13) $ln\left(POPshr^{emigr,LOC2}_{loc2,t}\right) = \alpha^{emigr,LOC2}_{loc2} + \sigma^{emigr,LOC2}_{loc2} * \ln\left(\frac{WF^{LOC2}_{loc2,t-1}}{WF^{ROW}_{t-1}}\right), \forall loc2 \in LOC2, t \in T$

where the aggregate rural-urban emigrant population stock shares ($POPshr^{emigr,LOC2}_{loc2,t}$) are stratified over two aggregate rural-urban household categories ($LOC2 = \{rural, urban\}$) and where parameters include scale parameters ($\alpha^{emigr,LOC2}_{loc2}$) and relative wage elasticities of emigration ($\sigma^{emigr,LOC2}_{loc2}$).

The aggregate rural-urban emigrant population shares ($POPshr^{emigr,LOC2}_{loc2,t}$) are subsequently multiplied by lagged regional household population totals ($POP_{h,t-1}$), to compute, in equation (4.14), current aggregate emigrant population stocks for our 19 regional household types ($POP^{emigr,H}_{h,t}$):

(4.14) $POP^{emigr,H}_{h,t} = POPshr^{emigr,LOC2}_{loc2,t|maphloc(h,loc2)} * POP^{H}_{h,t-1}, \forall h \in H, t \in T$

Where the $maphloc(h, loc2)$-mapping again maps our 19 household types into rural and urban categories.

The aggregate household-specific emigrant population stocks ($POP^{emigr,H}_{h,t}$) are further disaggregated, in equation (4.15), into age- and gender-specific immigrant population stocks ($POP^{emigr}_{h,gen,age,t}$) based on fixed age- and gender-specific emigrant patterns ($\beta^{emigr}_{h,gen,age}$):

(4.15) $POP^{emigr}_{h,gen,age,t} = \beta^{emigr}_{h,gen,age} * POP^{emigr,H}_{h,t}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$

where $\sum_{gen,age} \beta^{emigr}_{h,gen,age} = 1, \forall h \in H$.

Finally, current age- and gender-specific emigration flows ($\Delta POP^{emigr}_{h,gen,age,t}$) are defined, in equation (4.16), as the net increase in household-specific emigrant population stocks:

(4.16) $\Delta POP^{emigr}_{h,gen,age,t} = POP^{emigr}_{h,gen,age,t} - POP^{emigr}_{h,gen,age-1,t-1}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$

4.2.4. Net domestic immigration

The modelling of net domestic migration flows ($\Delta POP^{dmigr,net,disagg}{}_{h,gen,age,t}$) follows a similar path to the modelling of international migration flows (sections 4.2.2-4.2.3 above) with a focus on gross domestic migrant population stocks ($POP^{dmigr.loc2}{}_{loc2,t}$). However, in order to maintain consistency (i.e. to meet the zero net domestic migration constraint), the disaggregation of aggregate domestic migrant flows differ somewhat from the international migrant modelling methodology.

Similar to the immigrant and emigrant specifications in equations (4.9) and (4.13), rural-urban gross domestic immigrant stock shares ($POPshr^{dmigr.loc2}{}_{loc2,t}$) are modelled, in equation (4.17), as functions of lagged rural-urban wages differentials based on Harris-Todaro migration specifications:

(4.17) $$ln\left(POPshr_{loc2,t}^{dmigr,LOC2}\right) = \alpha_{loc2}^{dmigr,LOC2} + \sigma_{loc2}^{dmigr,LOC2} * \ln\left(\frac{WF_{'urban',t-1}^{LOC2}}{WF_{'rural',t-1}^{LOC2}}\right), \forall loc2 \in LOC2, t \in T$$

where parameters include scale parameters ($\alpha_{loc2}^{dmigr,LOC2}$) and relative wage elasticities of rural-urban domestic immigration ($\sigma_{loc2}^{dmigr,LOC2}$).

The gross domestic immigrant stock shares ($POPshr_{loc2,t}^{dmigr,LOC2}$) are subsequently multiplied by lagged rural and urban population totals, corrected for current international migration flows and current deaths, to compute, in equation (4.18), rural and urban gross domestic immigrant stocks:

(4.18) $$POP_{loc2,gen,age,t}^{dmigr,LOC2} = POPshr_{loc2,t}^{dmigr,LOC2} * \sum_{h|maphloc(h,loc2)}\left(POP_{h,gen,age-1,t-1} + \Delta POP_{h,gen,age,t}^{imigr,net} - Deaths_{h,gen,age,t}\right), \forall loc2 \in LOC2, gen \in GEN, age \in AGE, t \in T$$

Recognizing that net domestic migration between rural and urban areas has to be zero, we use the gross domestic immigrant population stocks ($POP_{loc2,gen,age,t}^{dmigr,LOC2}$) to compute, in equation (4.19), net domestic immigrant stocks for rural and urban areas ($POP_{loc2,t}^{dmigr,net,LOC2}$):

(4.19) $$POP_{loc2,gen,age,t}^{dmigr,net,LOC2} = POP_{loc2,gen,age,t}^{dmigr,LOC2} - POP_{loc2p,gen,age,t|loc2p\neq loc2}^{dmigr,LOC2}, \forall loc2 \in LOC2, gen \in GEN, age \in AGE, t \in T$$

We further disaggregate, in equation (4.20), these rural-urban net domestic immigrant stocks ($POP_{loc2,gen,age,t}^{dmigr,net,LOC2}$) into household-specific net domestic immigrant stocks ($POP_{h,gen,age,t}^{dmigr,net}$) based on fixed immigrant patterns across rural and urban household types ($\beta_{h,gen,age}^{dmigr}$):

(4.20) $POP^{dmigr,net}_{h,gen,age,t} = \beta^{dmigr}_{h,gen,age} * \sum_{loc2|maphloc(h,loc2)} POP^{dmigr,net,LOC2}_{loc2,gen,age,t}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$

where $\sum_{h|maphloc(h,loc2)} \beta^{dmigr}_{h,gen,age} = 1, \forall loc2 \in LOC2, gen \in GEN, age \in AGE.$

Finally, net domestic immigration flows are defined, in equation (4.21), as the net increase in household-specific domestic immigrant population stocks:

(4.21) $\Delta POP^{dmigr,net}_{h,gen,age,t} = POP^{dmigr,net}_{h,gen,age,t} - POP^{dmigr,net}_{h,gen,age-1,t-1}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$

4.2.5. Aggregate wage indices for migration determination

The relative wage indices which are used in the migration specifications (equations (4.9), (4.13) and (4.17)), are based on two types of wage indices including (1) a domestic wage index type for rural and urban workers, and (2) an international wage index type for foreign workers. The domestic wage index type is defined for rural and urban workers, and computed on the basis of household-specific factor endowments ($QFH_{h,flab,t}$) multiplied by average labour factor wages ($WF_{flab,t}$) from the macroeconomic CGE model:

(4.22) $WF^{LOC2}_{loc2,t} = \sum_{h,flab|maphloc(h,loc2)} WF_{flab,t} * \frac{QFH_{h,flab,t}}{\sum_{h1,flab1|maphloc(h,loc2)} QFH_{h1,flab1,t}}, \forall h \in H, gen \in GEN, age \in AGE, t \in T$

where factors are stratified over labour factor types ($flab \in FLAB$).

The international wage index (WF^{ROW}_t) for workers from the Rest of the World (ROW) is computed, in equation (4.23), as a fixed (in real value terms) international wage ($\overline{WF}^{ROW}$) multiplied by the exchange rate (EXR_t) from the macroeconomic CGE model:

(4.23) $WF^{ROW}_t = \overline{wf}^{ROW} * EXR_t, t \in T$

4.3. Parametrization

The calibration of the demographic model involved the exogenous imposition of (interpolated) quinquennial UN parameter values for fertility rates, while age- and gender-specific mortality rates were calibrated to ensure consistency with quinquennial UN population projections for 2000-2100, derived from the World Population Prospects, 2010 revision (UN 2013). Specifically, the 2000-2010 baseline data were joined with the 2010-2100 medium scenario projections into a combined 2000-2100 Ghana population dataset, and this dataset, combined the household and census survey data (GSS 2003, GSS 2008, GSS 2012b) (see details below), formed the basis for initializing key variables of our demographic model including population levels ($POP_{h,gen,age,t}$) and population deaths ($Deaths_{h,gen,age,t}$). The latter two data

series were initially used to calibrate non-infant all-cause mortality rates ($\bar{\mu}^{all\ cuase}_{h,gen,age,t|age>0}$). Combined with (interpolated) UN parameter assumptions about gender birth ratios ($sexratio_{gen}$) and age-specific fertility rates ($asfr_{age}$), these data were subsequently used to derive and initialize gender-specific births ($Births_{h,gen,t}$) and (residually) infant deaths ($InfDeaths_{h,gen,t}$). The projections of infant deaths were, finally, used to calibrate infant all-cause mortality rates ($\bar{\mu}^{all\ cuase}_{h,gen,age,t|age=0}$).

The 2000-2100 population projections for Ghana were only available at quinquennial time intervals, at the national level, and only for 5-year age groups. Linear interpolation was used to disaggregate to annual population projections for 1-year age groups, and regional population shares, obtained from the 2005/06 GLSS household survey (GSS 2008), was combined with domestic and international migration patterns, based on information from the 2000 and 2010 Ghana Censuses (GSS 2003, GSS 2012b), to derive regional household-specific disaggregate population projections ($POP_{h,gen,age,t}$) and disaggregate population deaths ($Deaths_{h,gen,age,t}$). While our model framework is specifically designed to simulate over the next 20 year period (2015-34), we present population projections for our 19 household types for the next 35 year period (2010-2050) in Figure 4.1, and they demonstrate that the Ghana population will continue to expand, rapidly, and that urbanization is likely to remain a prominent feature of Ghana society until 2050 with GAMA seeing the greatest population growth.

The migration module of our demographic model framework (section 4.2) is focused on calculating household net migration (section 4.2.1) from respectively (1) international migration (sections 4.2.2 and 4.2.3) and (2) domestic migration (section 4.2.4). The calibration of international migration patterns relied on a combination of (1) UN assumptions about net international migration patterns over 2010-2050, (2) region-specific immigration and emigration patterns from the 2000 Ghana Census (GSS 2003), and (3) a survey of internal and international migration elasticities.

For domestic migration, the UN population projections do not provide information about domestic regional migration patterns. Hence, the calibration of domestic migration patterns between our 19 distinct regional household types had to rely on reasonable assumptions about future domestic migration patterns. Based on numbers from the 2000 and 2010 Census Reports (GSS 2003, GSS 2012b), it was clear that the urban population share had grown from 32.0% in 1984, to 43.8% in 2000, and to 50.9% in 2010. This amounts to growth rates of 0.73%-points p.a. during 1984-2000, and 0.71 %-points p.a. during 2000-2010. Given the very high levels of urbanization of the past 25 years, it is however clear that future urbanization rates (within our extended time horizon 2010-2050 and beyond) will have to be smaller. Instead of assuming unchanged domestic migration patterns, we therefore assumed that rural-urban migration rates will

decline linearly from 0.71 %-points p.a. in 2010 to 0.0%-points p.a. in 2100. This assumption implies that urbanization levels will stabilize beyond 2100 at around 82.6%. Based on this assumed counterfactual domestic migration pattern, we proceeded to compute domestic rural-urban migration patterns for each of our 19 distinct geographical areas based on regional domestic migration patterns from the 2000 Ghana Census (GSS 2003).

After initialization of the immigration, emigration, and domestic migration patterns for our 19 regional household types (and after subsequent derivation of regional population projections), we calibrated the level parameters of our three migration specification types ($\alpha_{loc2}^{imigr,LOC2}$, $\alpha_{loc2}^{emigr,LOC2}$, $\alpha_{loc2}^{dmigr,LOC2}$) based on (1) relative rural and urban (and international) wage levels derived from the macroeconomic CGE model (section 4.2.5), and (2) relative wage elasticities of migration ($\sigma_{loc2}^{imigr,LOC2}$, $\sigma_{loc2}^{emigr,LOC2}$, $\sigma_{loc2}^{dmigr,LOC2}$). Parameters for age- and gender-distribution of migrants within households ($\beta_{h,gen,age}^{imigr}$, $\beta_{h,gen,age}^{emigr}$, $\beta_{h,gen,age}^{dmigr}$) were calibrated to migration patterns in the initial year (2000).

The non-calibrated demographic model parameters, i.e. relative wage elasticities ($\sigma_{loc2}^{imigr,LOC2}$, $\sigma_{loc2}^{emigr,LOC2}$, $\sigma_{loc2}^{dmigr,LOC2}$), are presented in Table 4.1, and they include (1) one Ghana-specific relative income elasticity of internal rural-urban migration (0.675) (Tsegay 2007), and (2) one relative income elasticity of international immigration (-1.41) (Clark, Hatton & Williamson 2007); It was, furthermore, assumed that the relative income elasticity of international emigration is the reverse of the specified immigration elasticity (+1.41). The elasticity estimates were specifically chosen since (1) the internal rural-urban migration elasticity is Ghana-specific (Tsegay 2007), and (2) the empirical specifications of both studies (Tsegay 2007; Clark, Hatton & Williamson 2007) were consistent with our Harris-Todaro type semi-elasticity specification of relative migrant stocks (Equations (4.9), (4.13), (4.17)).[8]

Table 4.1. Demographic model parameters: Relative wage elasticities of migration

	Relative wage elasticity of migration
Internal migration[a]	
- internal migration	0.675
International migration[b]	
- emigration	-1.41
- immigration	1.41

Source: [a]Tsegai (2007); [b]Clark, Hatton & Williamson (2007)

[8] The former Ghana-specific study (Tsegay 2007) employed an empirical probit model, but provided a marginal elasticity estimate (0.675) which, under first-order approximation, fits our semi-elasticity specification, while the latter international cross-section study (Clark, Hatton & Williamson 2007) employs an empirical specification which is equivalent to our international migration specifications.

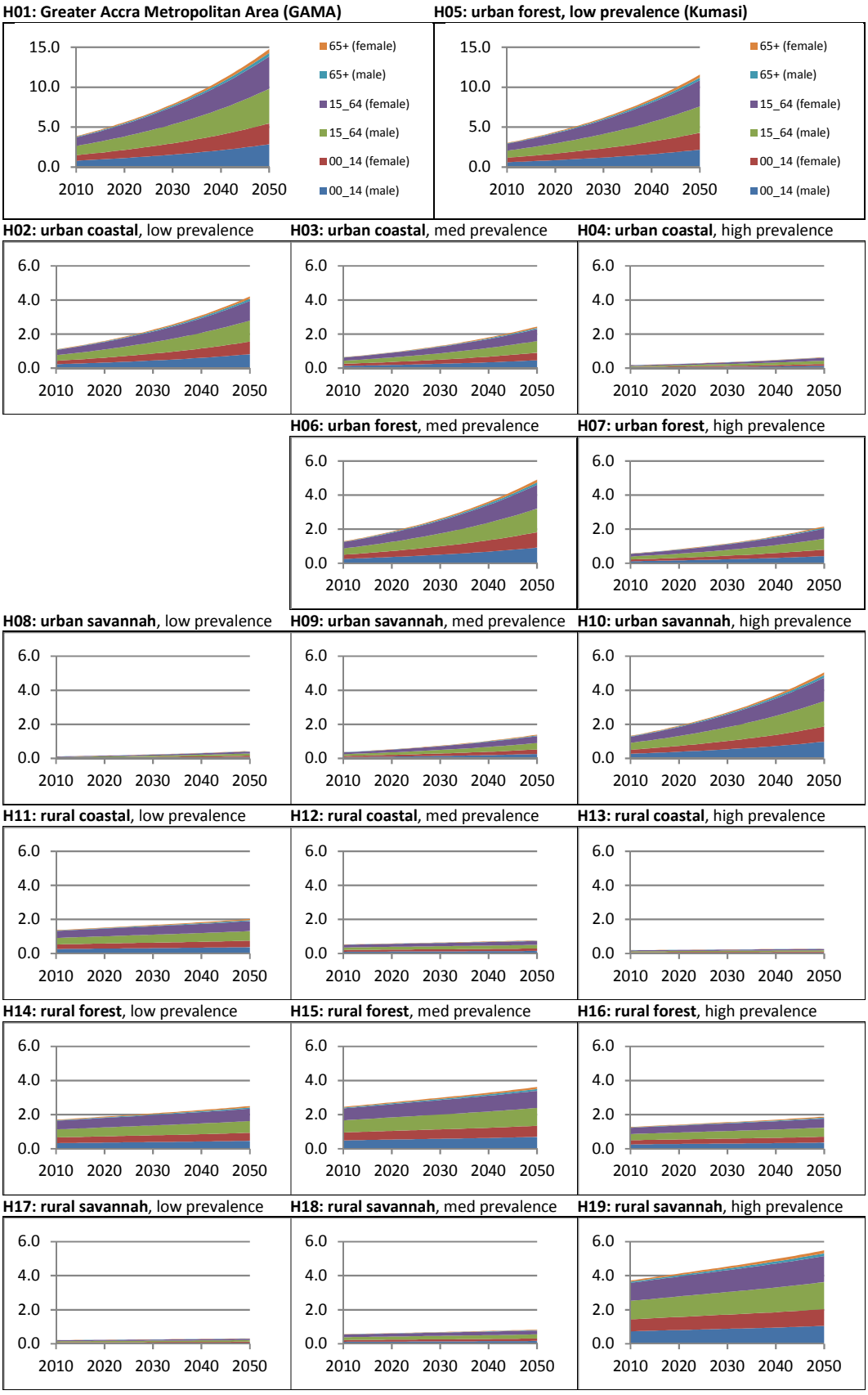


Figure 4.1. 2010-2050 Demographic projections for Ghana (millions); by gender and age group

It should be noted that a historical study of internal migration between Ghanaian regions (Beals, Levy & Moses 1967) finds evidence of higher relative income elasticities of migration (absolute values ε [1.4; 2.9]) compared to our preferred point estimate (0.675). In contrast, more recent studies of international migration (Grogger & Hanson 2011; Ortega & Peri 2012) find indications of lower relative income elasticities of international migration (absolute values ε [0.3; 0.8]) compared to our preferred estimate (1.41). It should also be noted that all studies, including our two preferred studies, are focusing on relative income differentials (as opposed to the wage-differential specification in our model), and that only Tsegay (2007) models household-level decisions (all other studies analyse regional or national cross section or panel data sets with a focus on regional or national per capita income).

5. Effective labour supply equations and parametrization

In this section, we outline a full set of effective labour supply specifications for our 42 labour factor types (section 5.1) and we also outline a full set of labour factor ownership specifications for our 42 labour factor types and stratified over our 19 malaria-focussed household types (section 5.2). All labour supply and ownership specifications are parameterized in section 4.3, while endogenous variables and exogenous parameters are listed and defined in Annex D.

5.1. Aggregate effective labour supply

The labour market, which keeps track of individual workers, are stratified according to three dimensions: (1) regional household type ($h \in H$), (2) labour ($flab \in FLAB$), and (3) time period ($t \in T$). The aggregate effective supply of a given labour factor type ($QFS^{eff}_{flab,t}$) is defined, in equation (5.1), as the sum over all households' effective supplies of that type of labour ($QFH^{eff}_{h,flab,t}$):

(5.1) $$QFS^{eff}_{flab,t} = \sum_h QFH^{H,eff}_{h,flab,t} \ , \forall flab \in FLAB, t \in T,$$

where households' effective labour supplies ($QFH^{eff}_{h,flab,t}$) are stratified over 19 household categories ($H = [h01; h19]$), 42 labour factor types ($FLAB = [f01; f42]$), and 20 time periods ($T = [2015; 2034]$).[9] The 42 labour factor categories are spanned by rural/urban and ecological region location (7 types), gender types (2 types) and skill levels (3 types) (see Table F.1 in Annex F for details).

5.2. Household effective labour factor ownership

Individual households' effective supply of a given labour type ($QFH^{eff}_{flab,t}$) is defined, in equation (5.2), as that households' total labour factor participation ($QFH^{H}_{h,flab,t}$) net of malaria-related labour-supply impacts of adult morbidity ($QFH^{H,morb,adult}_{h,flab,t}$) and child morbidity ($QFH^{H,morb,child}_{h,flab,t}$):

(5.2) $$QFH^{H,eff}_{h,flab,t} = QFH^{H}_{h,flab,t} - QFH^{H,morb,adult}_{h,flab,t} - QFH^{H,morb,child}_{h,flab,t} \ , \forall h \in H, flab \in FLAB, t \in T.$$

Individual households' labour factor participation for individual labour factor types ($QFH_{h,flab,t}$) are computed, in equation (5.3), on the basis of household-specific working age population levels ($POP_{h,gen,age,t|age\in[15;64]}$) corrected for gender-specific participation rates ($PRate_{gen}$) and household- (and gender-specific) labour factor skill shares ($SklShr_{h,flab}$):

[9] Note that Table E.1 in Annex E highlights the (partially) household-specific nature of labour factor types, where households with the same regional location (7 types), but not necessarily with the same malaria prevalence levels, are assumed to own the same types of gender- and skill-specific labour factors (6 types).

(5.3) $QFH^{H}_{h,flab,t} = SklShr_{h,flab} * \sum_{gen|mapgflab(gen,flab),age|age\in[15;64]} PRate_{gen} * POP_{h,gen,age,t}$,

$\forall h \in H, flab \in FLAB, t \in T,$

where the $mapgflab(gen,flab)$-mapping maps our 42 labour factor types into male and female gender categories.

The malaria-related labour supply impact of adult morbidity ($QFH^{morb,adult}_{h,flab,t}$) refers to incapacitated sick adults who are forced to reduce their number of work days (absenteeism). The labour supply impact of adult morbidity is computed, in equation (5.4), on the basis of the total number of household-specific uncomplicated malaria episodes for working age individuals ($\sum_{age|age\in[15;64]} \tau_{h,age,t}(EIR_{h,t}) * POP_{h,gen,age,t}$) corrected for gender-specific participation rates ($PRate_{gen}$) and labour factor skill shares ($SklShr_{h,flab}$), and multiplied by the morbidity rate measured by the average amount of reduced worktime (years/episode) per uncomplicated episode ($MRate_{h,t}$):

(5.4) $QFH^{H,morb,adult}_{h,flab,t} = MRate_{h,t} * SklShr_{h,flab} * \sum_{gen|mapgflab(gen,flab)} PRate_{gen} *$
$\left(\sum_{age|age\in[15;64]} \tau_{h,age,t}(EIR_{h,t}) * POP_{h,gen,age,t}\right), \forall h \in H, flab \in FLAB, t \in T.$

where the morbidity rate ($MRate_{h,t}$) is endogenous due to the endogenous ACT coverage rates (see equation (6.20) in section 6).

Finally, the malaria-related labour supply impact of child morbidity ($QFH^{morb,child}_{h,flab,t}$) refers to (female) caretakers for sick children who are forced to reduce their number of workdays (absenteeism). Similar to adult morbidity, the labour supply impact of child morbidity ($QFH^{morb,child}_{h,flab,t}$) is calculated, in equation (5.5), on the basis of the total number of household-specific uncomplicated malaria episodes for children ($\sum_{age,gen|age\in[15;64]} \tau_{h,age,t}(EIR_{h,t}) * POP_{h,gen,age,t}$) corrected for female labour participation rates ($PRate_{,female'}$) and (female) labour factor skill shares ($SklShr_{h,flab|mapgflab(,female',flab)}$), and multiplied by the average absenteeism measured by the amount of reduced worktime (years/episode) per uncomplicated episode ($\nu_{h,t}$,)[10]:

(5.5) $QFH^{H,morb,child}_{h,flab,t} = \nu_{h,t} * SklShr_{h,flab} * PRate_{,female'} * \sum_{age,gen|age\in[15;64]} \tau_{h,age,t}(EIR_{h,t}) *$
$POP_{h,gen,age,t}, \forall h \in H, flab \in FLAB|mapgflab('female', flab), t \in T.$

[10] Due to limited evidence of presenteeism (=workdays lost due to low productivity while at work; see Table 6.4 for available evidence), this dimension was not included in the current model specification.

5.3. Parametrization

The full system of effective labour factor supply and labour factor ownership equations (5.1)-(5.5) is determined by population demographics, $POP_{h,gen,age,t}$ (implicitly accounting for malaria-related mortality, see section 4), and the two malaria-related morbidity relationships (eqs. (5.4)-(5.5)) which refer to reduced adult labour supplies associated with respectively adult illness ($QFH_{h,flab,t}^{morb,child}$) and female adults caring for sick children ($QFH_{h,flab,t|mapgflab('female',flab)}^{morb,child}$). Morbidity effects are calculated as the affected gender-specific working age population group ($POP_{h,gen,age,t|age\in[15;64]}$) multiplied by four sets of parameters: gender-specific participation rates ($PRate_{gen}$) multiplied by (gender-specific) labour factor skill shares ($SklShr_{h,flab}$) multiplied by the %-share reduction in annual labour supply per malaria episode ($MRate$) multiplied by the average number of malaria episodes per person per year ($\tau_{h,age,t}$).

The parametrization of the equation for the determination of uncomplicated malaria episodes ($\tau_{h,age,t}$) has already been discussed above (see section 3) and so has the initialization of population demographics, $POP_{h,gen,age,t}$ (see section 4). Labour force data from the 2005/06 GLSS household survey (GSS 2008) was employed to calibrate the $SklShr_{h,flab}$ skill share parameter values (see table F.1, annex F) while other external information was used to parameterize the two remaining sets of parameters: (1) Gender-specific labour market participation rates ($PRate_{'female'}$ = 73.8%; $PRate_{'male'}$ = 75.2%) were obtained from the World Development Indicators database (WB 2012) (Table 5.1), while (2) a central point estimate for the rate of malaria-related absenteeism ($\nu_{h,t}$) (see equation (6.20) and section 6.3 for details of the initialization).

Table 5.1. Labour force participation rates

	2009
Female participation rate (% of female population ages 15+)	73.8%
Male participation rate (% of male population ages 15+)	75.2%

Source: World Development Indicators (WB 2012).

Together with the parametrization of uncomplicated malaria episodes ($\tau_{h,age,t}$) and malaria-related mortality rates ($\mu_{h,age,t}$), discussed above (see section 3), the parameters, discussed here, provide the core information for calculating the labour market impact of the economy-wide malaria disease burden. It should specifically be noted that both skill shares and labour market participation rates, derived for the general labour force (to parametrize equation (5.3)), are assumed to apply equally to (1) sub-population groups of adults suffering from malaria illness, and (2) sub-population groups of female caregivers caring for sick children (equations (5.4)-(5.5)).

6. Health intervention equations and parametrization

In this section, we outline a full set of endogenous malaria intervention coverage rate specifications, based on extended household and public demand systems, which allows for measuring public and private household costs of composite intervention commodities including intervention and administrative and laboratory service input costs (section 6.1), and for measuring prevention intervention impacts on epidemiological parameters including mosquito mortality rates and female mosquito populations, and treatment intervention impacts on morbidity outcomes (section 6.2). All health intervention-related specifications are parameterized in section 6.3, while endogenous variables and exogenous parameters are listed and defined in Annex E.

6.1. Malaria intervention equations

The health intervention module keeps track of malaria-related composite intervention commodities, and their component parts including (1) malaria interventions and (2) administrative and laboratory services, according to three index dimensions: (1) regional household type ($h \in H$), (2) malaria interventions ($int \in INT$), and (3) time period ($t \in T$). Total malaria intervention coverage rates ($COVER_{int,h,t}$), defined as #ITNs per household member/#ACT doses per malaria case, are modelled, in equation (6.1), as the sum of private and public malaria intervention coverage rates ($COVER^{prv}_{int,h,t}$, $COVER^{pub}_{int,h,t}$):

(6.1) $$COVER_{int,h,t} = COVER^{prv}_{int,h,t} + COVER^{pub}_{int,h,t}, \forall int \in INT, h \in H, t \in T,$$

where the malaria intervention coverage rates ($COVER_{int,h,t}$) are stratified over two Insecticide Treated Nets (ITN) and Artemisinin-based Combination Therapy (ACT) malaria interventions ($INT = \{ITN, ACT\}$), 19 household categories ($H = [h01; h19]$), and 20 time periods ($T = [2015; 2034]$).

The aggregate coverage rates ($COVER_{int,h,t}$) are multiplied by household-specific uptake rates ($UPTAKE_{int,h,tt}$), defined as #household members sleeping under each ITN/share of ACT doses administered correctly, to calculate effective coverage rates ($COVER^{eff}_{int,h,t}$) in equation (6.2):

(6.2) $$COVER^{eff}_{int,h,t} = COVER_{int,h,t} * UPTAKE_{int,h,t}, \forall int \in INT, h \in H, t \in T,$$

and uptake rates ($UPTAKE_{int,h,t}$) are modelled, in equation (6.3), as functions of household-specific average human malaria prevalence rates:

(6.3) $$ln\left(UPTAKE_{int,h,t}\right) = \bar{\alpha}^{upt}_{int} + \bar{\beta}^{upt}_{int} * \ln\left(p^{H}_{h,t}\right), \forall int \in INT, h \in H, t \in T,$$

where parameters include scale parameters ($\bar{\alpha}_{int}^{upt}$) and malaria prevalence elasticities of intervention uptake ($\bar{\beta}_{int}^{upt}$).

Private and public coverage rates are determined by private and public malaria-related composite intervention commodities ($QH_{int,h,t}$, $\overline{QG}_{int,h,t}$), and by underlying regional population levels ($POP_{h,t}^{H}$) in the case of prevention interventions ($int \in INT\_P$) and by numbers of uncomplicated episode cases ($\sum_{age,gen} \tau_{h,age,t}(EIR_{h,t}) * POP_{h,gen,age,t}$) in the case of treatment interventions ($int \in INT\_T$):

(6.4_P) $COVER_{int,h,t}^{prv} = \frac{QH_{int,h,t}}{POP_{h,t}^{H}}$, $\forall int \in INT\_P, h \in H, t \in T$,

(6.4_T) $COVER_{int,h,t}^{prv} = \frac{QH_{int,h,t}}{\sum_{age,gen} \tau_{h,age,t}(EIR_{h,t}) * POP_{h,gen,age,t}}$, $\forall int \in INT\_T, h \in H, t \in T$,

(6.5_P) $COVER_{int,h,t}^{pub} = \frac{\overline{QG}_{int,h}}{POP_{h,t}^{H}}$, $\forall int \in INT\_P, h \in H, t \in T$,

(6.5_T) $COVER_{int,h,t}^{pub} = \frac{\overline{QG}_{int,h}}{\sum_{age,gen} \tau_{h,age,t}(EIR_{h,t}) * POP_{h,gen,age,t}}$, $\forall int \in INT\_T, h \in H, t \in T$,

where INT_P and INT_T are sets containing all elements along the prevention intervention ($INT\_P = \{ITN\}$) and treatment intervention ($INT\_T = \{ACT\}$) dimensions.[11]

Public demand for composite intervention commodities ($\overline{QG}_{int,h}$) is assumed to be exogenous, while private demand for composite intervention commodities ($QH_{int,h,t}$) is determined as part of an expanded household LES demand system in equations (6.6')-(6.6'') in the CGE model:

(6.6') $PQ_{c,t} * QH_{c,h,t} = PQ_{c,t} * \bar{\gamma}_{c,h} + \bar{\beta}_{c,h} * \left(EH_{h,t} - \sum_{c1} PQ_{c1,t} * \bar{\gamma}_{c1,h} - \sum_{int1} PQ_{int,t} * \bar{\gamma}_{int1,h}\right)$,

$\forall c \in C, h \in H, t \in T$

(6.6'') $PQ_{int,t} * QH_{int,h,t} = PQ_{int,t} * \bar{\gamma}_{int,h} + \bar{\beta}_{int,h} * \left(EH_{h,t} - \sum_{c1} PQ_{c1,t} * \bar{\gamma}_{c1,h} - \sum_{int1} PQ_{int,t} * \bar{\gamma}_{int1,h}\right)$,

$\forall int \in INT, h \in H, t \in T$

[11] Our current model specification excludes the use of ACTs for medical prevention purposes. Furthermore, while our model framework is set up to analyse public-funded In-door Residual Spraying (IRS) prevention interventions, these interventions are also excluded from current analyses, since regional coverage data are not immediately available (see discussion in section 6.3).

where consumption demand ($QH_{c,h,t}, QH_{int,h,t}$) is stratified over 10 commodities ($c \in C$) and two malaria interventions ($int \in INT$), 19 household categories ($H = [h01; h19]$), and 20 time periods ($T = [2015; 2034]$), and where parameters include autonomous consumption levels ($\bar{\gamma}_{c,h}, \bar{\gamma}_{int,h}$) and marginal consumption shares ($\bar{\beta}_{c,h}, \bar{\beta}_{int,h}$).

The total intervention-specific demands for malaria interventions ($\sum_h(QH_{int,h,t} + \overline{QG}_{int,h})$) are subsequently set equal, in equation (6.7), to the total supply of individual health interventions ($QQ_{int,t}$):

(6.7) $QQ_{int,t} = \sum_h(QH_{int,h,t} + \overline{QG}_{int,h})$, $\forall c \in C, h \in H, t \in T$

and the total supply of individual malaria-related composite intervention commodities ($QQ_{int,t}$) are, in turn, assumed to be determined by Leontief production specifications with inputs covering (1) malaria interventions ($QQ^{INT}_{int,t}$), and (2) administrative and laboratory services ($QQ^{ADM}_{'c10',int,t}$), leading to the following first order conditions, provided in equations (6.8')-(6.8''), to be included in the CGE model:

(6.8') $QQ^{INT}_{int,t} = \beta^{INT}_{int} * QQ_{int,t}$, $\forall int \in INT, t \in T$

(6.8'') $QQ^{ADM}_{'c10',int,t} = \beta^{ADM}_{int} * QQ_{int,t}$, $\forall int \in INT, t \in T$

The supply of administrative and laboratory services is assumed to form part of the overall supply of health services (commodity 'c10'), while the supplies of malaria interventions are assumed to consist, entirely, of imports from the Rest of the World. Total demand for administrative and laboratory services ($\sum_{int} QQ^{ADM}_{'c10',int,t}$) is therefore included, in equation (6.9), in an extended version of the commodity market equilibrium constraint from the CGE model:

(6.9) $QQ_{c,t} = \sum_a QINT_{c,a,t} + \sum_h QH_{c,h,t} + \overline{QG}_c + \sum_{int} QQ^{ADM}_{'c10',int,t} + QINV_{c,t} + \overline{qdst}_c + QT_{c,t}$, $\forall c \in C, t \in T$

while total (import) demand for malaria-interventions is included, in equation (6.10), in a new equilibrium constraint ($QQ^{INT}_{int,t} = QM^{INT}_{int,t}$) as well as, in equation (6.11), in an updated version of the balance-of-payments equilibrium constraint from the CGE model:

(6.10) $QQ^{INT}_{int,t} = QM^{INT}_{int,t}$, $\forall int \in INT, t \in T$

(6.11) $\sum_c \overline{pwm}_c * QM_{c,t} + \sum_f \overline{trnsfr}_{'ROW',f,t} + \sum_{int} \overline{pwm}^{INT}_{int,t} * QM^{INT}_{int,t} = \sum_c \overline{pwe}_c * QE_{c,t} + \sum_{insd} \overline{trnsfr}_{insd,'ROW'} + \overline{FSAV}$, $\forall t \in T$

The costs of private interventions are included in individual household budgets (implicit in the LES demand system equations (6.6')-(6.6'')), while the costs of public sector interventions are included, in equation (6.12), in an updated version of the public sector expenditure equation from the CGE model:

(6.12) $EG_t = \sum_c PQ_{c,t} * \overline{QG}_{c,t} + \sum_{int,h} PQ_{int,t} * \overline{QG}_{int,h,t} + \sum_{insdng} \overline{trnsfr}_{insdng,'GOV'}, \forall t \in T$

Imports of malaria interventions are also assumed to incur import-related trade and transportation margin costs as well as import tariffs and sales taxes, in line with other imports. The added demand for import-related trade and transportation margin services from malaria interventions ($\sum_{int} \overline{\imath cm}^{INT}_{int,t} * QM^{INT}_{int,t}$ was included, in equation (6.13), in an updated version of the trade and transport margin demand equation from the CGE model:

(6.13) $QT_{ct,t} = \sum_c \left(\overline{\imath cm}_{ct,c} * QM_{c,t} + \overline{\imath ce}_{ct,c} * QE_{c,t} + \overline{\imath cd}_{ct,c} * QD_{c,t}\right) + \sum_{int} \overline{\imath cm}^{INT}_{ct,int} * QM^{INT}_{int,t}, \forall t \in T$

while additional malaria intervention-related import tariffs ($\sum_c \overline{tm}^{INT}_{int} * \overline{pwm}^{INT}_{int,t} * QM^{INT}_{int,t} * EXR_t$) and sales taxes ($\sum_c \overline{tq}^{INT}_{int} * PQ^{INT}_{int,t} * QQ^{INT}_{int,t}$) were included, in equation (6.14), in an updated version of the public sector income equation from the CGE model:

(6.14) $YG_t = \sum_{insdng} TINS_{insdng,t} * YI_{insdng,t} + \sum_t \overline{tf}_f * \sum_h WF_{f,t} * QFH_{h,f,t}$

$+ \sum_a \overline{tva}_a * PVA_{a,t} * QVA_{a,t}$

$+ \sum_a \overline{ta}_a * PA_{a,t} * QA_{a,t}$

$+ \sum_c \overline{tm}_c * \overline{pwm}_c * QM_{c,t} * EXR_t$

$+ \sum_c \overline{te}_c * \overline{pwe}_c * QE_{c,t} * EXR_t$

$+ \sum_c \overline{tq}_c * PQ_{c,t} * QQ_{c,t}$

$+ \sum_f YIF_{'GOV',f,t} + \overline{trnsfr}_{'GOV','ROW'}$

$+ \sum_{int} \overline{tm}^{INT}_{int} * \overline{pwm}^{INT}_{int,t} * QM^{INT}_{int,t} * EXR_t$

$+ \sum_c \overline{tq}^{INT}_{int} * PQ^{INT}_{int,t} * QQ^{INT}_{int,t}, \forall t \in T$

The import and market prices of malaria-interventions ($PM^{INT}_{int,t}, PQ^{INT}_{int,t}$) are specified, in equations (6.15)-(6.16) as functions of exogenous world market import prices ($\overline{pwm}_{int}$) and trade and transport service rates ($\overline{\imath cm}^{INT}_{ct,int}$), as well as import tariff rates ($\overline{tm}^{INT}_{int}$) and sales tax rates ($\overline{tq}^{INT}_{int}$):

(6.15) $PM_{int,t}^{INT} = \left(1 + \overline{tm}_{int}^{INT}\right) * \overline{pwm}_{int,t}^{INT} * EXR_t + \sum_{ct} PQ_{ct,t} * \overline{icm}_{ct,int}^{INT}, \forall int \in INT, t \in T$

(6.16) $PQ_{int,t}^{INT} = \frac{PM_{int,t}^{INT}}{1-\overline{tq}_{int}^{INT}}, \forall int \in INT, t \in T$

The aggregate price of malaria-related composite intervention commodities ($PQ_{int,t}$) is, finally, defined, in equation (6.17), from the Leontief production specification, as a weighted average of the market prices of malaria interventions ($PQ_{int,t}^{INT}$) and administrative and laboratory services ($PQ_{'c10',t'}$):

(6.17) $PQ_{int,t} = \frac{PQ_{int,t}^{INT} * QQ_{int,t}^{INT} + PQ_{'c10',t} * QQ_{'c10',int,t}^{ADM}}{QQ_{int,t}}, \forall int \in INT, t \in T$

6.2. Epidemiological impact equations

The modelling of the epidemiological impact of preventive malaria interventions is focussed on the impact on epidemiological model parameters (see section 3). In particular, we initially specify and calibrate the epidemiological model to measure the impact of two types of preventive interventions: (1) Indoor Residual Spraying (IRS) and (2) Insecticide Treated Nets (ITN).[12] These interventions are assumed to affect the following two epidemiological model parameters: (1) The mosquito mortality rate (μ^M), and (2) the mosquito population/number of female mosquitoes per person (m).

We follow the established approach in the literature and assume that coverage rates affect epidemiological model parameters linearly (Smith, Chitnis, Brie & Tanner 2011). However, since we are working with multiple prevention interventions, potentially affecting the same model parameters, we introduce a new specification which (1) retains the linearity when only one intervention is applied, but (2) allows for multiplicative effects when multiple interventions are applied simultaneously (see equations (6.17)-(6.18)).

Based on measures of %-point reductions in parameter values associated with 100% coverage rates of preventive interventions ($\Delta\bar{\mu}_{int,h}^{M,max}, \Delta\bar{m}_{int,h}^{max}$; see section 6.3, below, for calibration discussion) and effective coverage rates ($COVER_{int,h,t}^{eff}$; see equation 6.2), the following generalized multiple-intervention specifications of intervention impacts are specified:

(6.18) $\mu_{h,t}^{M} = \bar{\mu}_h^M * \prod_{int}\left(\left(1 - COVER_{int,h,t}^{eff}\right) + \left(1 - \Delta\bar{\mu}_{int,h}^{M,max}\right) * COVER_{int,h,t}^{eff}\right), \forall h \in H, t \in T$

(6.19) $m_{h,t} = \bar{m}_h * \prod_{int}\left(\left(1 - COVER_{int,h,t}^{eff}\right) + \left(1 - \Delta\bar{m}_{int,h}^{max}\right) * COVER_{int,h,t}^{eff}\right), \forall h \in H, t \in T$

[12] While our model framework is set up to analyse public-funded In-door Residual Spraying (IRS) prevention interventions, these interventions are excluded from current analyses, since regional coverage data are not immediately available (see section 6.3).

While our epidemiological model framework is set up to analyse public-funded In-door Residual Spraying (IRS) prevention interventions through multiple-intervention specifications of intervention impacts on epidemiological model parameters (see equations (6.18)-(6.19) above and calibration discussion below), we exclude IRS interventions from our model applications since regional IRS coverage data are not immediately available. This implies that our multiple-intervention specifications in equations (6.18)-(6.19) are reduced to standard single-intervention specifications (Smith, Chitnis, Brie & Tanner 2011):

(6.18') $\mu_{h,t}^{M} = \bar{\mu}_{h}^{M} * \left(\left(1 - COVER_{'ITN',h,t}^{eff}\right) + \left(1 - \Delta\bar{\mu}_{'ITN',h}^{M,max}\right) * COVER_{'ITN',h,t}^{eff}\right), \forall h \in H, t \in T$

(6.19') $m_{h,t} = \bar{m}_{h} * \left(\left(1 - COVER_{'ITN',h,t}^{eff}\right) + \left(1 - \Delta\bar{m}_{'ITN',h}^{max}\right) * COVER_{'ITN',h,t}^{eff}\right), \forall h \in H, t \in T$

Finally, the modelling of treatment-focussed malaria interventions is focussed on one intervention type: Artemisinin-based Combination Therapy (ACT). We choose to focus on modelling of ACT treatment impact on the absenteeism morbidity rate, which measures the reduced worktime associated with uncomplicated malaria episodes.[13] Based on a measure of worktime lost following proper ACT treatment and the assumption that average population-wide morbidity rates will decline linearly with effective ACT coverage rates, we specify the following relation for computation of population-wide household-specific average morbidity rates ($\nu_{h,t}$) as a function of ACT effective coverage rates ($COVER_{'ACT',h,t}^{eff}$) and fixed morbidity rates associated with and without effective ACT treatment ($\bar{\nu}^{ACT}, \bar{\nu}^{NoACT}$):

(6.20) $\nu_{h,t} = COVER_{'ACT',h,t}^{eff} * \bar{\nu}^{ACT} + \left(1 - COVER_{'ACT',h,t}^{eff}\right) * \bar{\nu}^{NoACT}, \forall h \in H, t \in T$

6.3. Parametrization

The parametrization of the malaria-related intervention equations from section 6.1 (equations (6.1)-(6.17)) relied on the coverage and uptake data and intervention cost data presented in Table 6.1 and the income and malaria prevalence elasticities of intervention demand and uptake presented in Table 6.2, while the parametrization of the epidemiological impact equations from section 6.2 (equations (6.18)-(6.19)) relied on basic data on mosquito population mean catch (MC) reductions and mosquito sporozoite rate (SR) reductions. Details are provided below.

While our epidemiological model framework is, in principle, set up to analyse public-funded In-door Residual Spraying (IRS) prevention interventions through multiple-intervention specifications of intervention impacts on epidemiological model parameters (see equations (6.18)-(6.19)), we exclude, as

[13] Due to limited evidence of presenteeism (=workdays lost due to low productivity while at work; see Table 6.4 for available evidence), this dimension was not included in the current model specification.

discuss above, IRS interventions from our model applications since regional IRS coverage data are not immediately available. The following discussion of model equation parametrization therefore focus on one prevention intervention (ITN) and one treatment intervention (ACT), except for the parameterization of the epidemiological impact equations (equations (6.18)-(6.19)) where parametrization for both ITN and IRS prevention interventions are discussed for completeness.

The household-specific estimates of coverage and uptake rates and intervention costs (Table 6.1) were computed from a range of data sources. For Insecticide-Treated Nets (ITNs), household-specific coverage and uptake rates were derived from the 2014 Ghana Demographic and Health Survey (GHS 2015), while household-specific splits between private and public coverage rates were based on 'public sector' and 'public campaign' coverage estimates from the fourth round 2012 MICS4 Multiple Indicator Cluster Survey (GSS 2012a). A single average cost estimate for ITNs, computed as the weighted average of 'Public', 'Private' and 'Other' median costs from the 2012 MICS4 Survey (ibid.), was attributed to each of our 19 household types since no household-specific information was available.

For Artemisinin-based Combination Therapy (ACT), region-specific ACT coverage rates, obtained from the 2007 Ghana Health Services annual report (GHS 2007), were distributed across the 2005 Ghana district classification (110 districts) and used to compute weighted average ACT coverage rates for each of our 19 household types based on population shares from the 2005/06 GLSS survey (GSS 2008). No household-level data were available concerning public/free supplies of ACT treatment. Instead, regional NHIS active membership rates, available from the 2012 National Health Insurance Agency annual report (NHIA 2012) were used to calculate public/free ACT coverage rates. Due to the nature of ACT treatment, uptake rates were assumed to be 100% (i.e. publicly free supplies/private purchases of medication are always assumed to be administered/applied appropriately by malaria patients).

Finally, an estimate of the (non-AMFm) medical cost of standard AS/AQ-type medical treatment[14] was obtained from a study of interventions under the Affordable Medicines Facility-malaria (AMFm) initiative (Bate et al. 2012), while an estimate of total drug, administrative and laboratory costs was obtained from a case study of the KNUST hospital (Dontwi, Dedu & Aboagye 2013). These estimates were applied,

[14] Two standard treatments are widely available for Ghana including (1) Artemether-lumefantrine fixed-dose combination (AL 20/120mg tablets; pack size 6x4), and (2) Artesunate-amodiaquine fixed-dose combination and co-blister (AS/AQ 100/270mg tablets; pack size 3x2) (Bate et al. 2012). While AL-type drugs has been reported to be most popular among Ghanaians in both rural and urban areas (Davis et al. 2013), AS/AQ remains the standard treatment recommended by Ministry of Health (MoH 2009). In either case, reported medical costs are virtually the same. Average 2011 AL-treatment costs (US$4.4/GHC6.5) are reported to be slightly higher than average 2011 AS/AQ-treatment costs (US$4.3/GHC6.7) (Bate et al. 2012), implying that our medical cost estimates are likely to be conservative.

uniformly, across our 19 household types, since no regional or household-specific cost estimates were available.

Table 6.1. Malaria-related composite intervention commodities variable initialization and parameter values

		Coverage rates (percent)		Uptake rates (percent)	Unit costs (2011 GHC per unit/dose)	
Intervention	Household	private	public/free		Medical/Physical	Administration
ITN	H01	16.3%	17.0%	49%	5.7	-
ITN	H02	14.2%	22.9%	75%	5.7	-
ITN	H03	14.2%	22.9%	75%	5.7	-
ITN	H04	14.2%	22.9%	75%	5.7	-
ITN	H05	18.5%	24.1%	70%	5.7	-
ITN	H06	18.5%	24.1%	70%	5.7	-
ITN	H07	18.5%	24.1%	70%	5.7	-
ITN	H08	10.9%	20.1%	78%	5.7	-
ITN	H09	10.9%	20.1%	78%	5.7	-
ITN	H10	10.9%	20.1%	78%	5.7	-
ITN	H11	13.2%	30.0%	128%	5.7	-
ITN	H12	13.2%	30.0%	128%	5.7	-
ITN	H13	13.2%	30.0%	128%	5.7	-
ITN	H14	18.1%	31.4%	120%	5.7	-
ITN	H15	18.1%	31.4%	120%	5.7	-
ITN	H16	18.1%	31.4%	120%	5.7	-
ITN	H17	9.7%	26.2%	134%	5.7	-
ITN	H18	9.7%	26.2%	134%	5.7	-
ITN	H19	9.7%	26.2%	134%	5.7	-
ACT	H01	50.1%	19.6%	100%	6.7	16.3
ACT	H02	37.8%	16.9%	100%	6.7	16.3
ACT	H03	25.3%	13.6%	100%	6.7	16.3
ACT	H04	15.8%	9.0%	100%	6.7	16.3
ACT	H05	46.6%	23.1%	100%	6.7	16.3
ACT	H06	40.4%	22.7%	100%	6.7	16.3
ACT	H07	19.2%	12.2%	100%	6.7	16.3
ACT	H08	36.6%	22.2%	100%	6.7	16.3
ACT	H09	26.1%	21.5%	100%	6.7	16.3
ACT	H10	42.9%	23.3%	100%	6.7	16.3
ACT	H11	40.2%	18.3%	100%	6.7	16.3
ACT	H12	39.8%	19.5%	100%	6.7	16.3
ACT	H13	15.8%	9.0%	100%	6.7	16.3
ACT	H14	43.2%	22.9%	100%	6.7	16.3
ACT	H15	43.8%	21.2%	100%	6.7	16.3
ACT	H16	18.4%	11.4%	100%	6.7	16.3
ACT	H17	36.6%	22.2%	100%	6.7	16.3
ACT	H18	27.8%	21.8%	100%	6.7	16.3
ACT	H19	40.0%	25.7%	100%	6.7	16.3

Sources: own calculations based on (1) ACT coverage rates from GHS 2007 Annual Report (GHS 2007), (2) Public/free ACT coverage rates based on National Health Insurance Authority 2012 annual report (NHIA 2012), (3) non-AMFm ACT prices and laboratory costs from AMFm evaluation study (Bate et al. 2012) and from a KNUST hospital study (Dontwi, Dedu & Aboagye 2013), (4) ITN coverage and uptake rates from the 2014 Ghana Demographic and Health Survey (GHS 2015), and (5) unit costs and Public/free ITN coverage rates from the 2011 MICS4 Multiple Indicator Cluster Survey (GSS 2012a).

Based on the ITN and ACT intervention coverage and uptake rates, and the unit cost estimates for individual interventions and associated administrative and laboratory services (Table 6.1) combined with income and malaria prevalence elasticities of intervention demand and intervention uptake derived from a literature survey (Dzator & Asafu-Ajaye 2004; Gingrich, Hanson et al. 2011; Picone, Kibler & Apouey 2013)

(Table 6.2), and combined with assumptions that (1) all interventions are imported (with trade margins and indirect tax structures similar to the manufactured goods sector, commodity 'c02'; see Annex A) and that (2) administrative and laboratory services are supplied domestically by the health services sector (commodity 'c10'), it was possible to parametrize and initialize the malaria-related composite intervention commodities equations, including equations for (1) malaria interventions and (2) administrative and laboratory services, from section 6.1 (equations (6.1)-(6.16)), including the (re-)specification of our Linear Expenditure System (LES) of private demand to include demand for composite health services related to prevention (ITN) and treatment (ACT) interventions (see section 2 for additional discussion of the calibration methodology for our extended LES demand system as part of the calibration of our broader CGE model framework).

Table 6.2. Income and prevalence elasticities of intervention demand and uptake

	Income elasticity[a,b]	Malaria prevalence elasticity[c]
Intervention demand		
- ACT	0.21	-
- ITN	0.459	-
Intervention uptake		
- ACT	-	-
- ITN	-	0.0043

Sources: [a] Income elasticity of ACT demand is proxied by minimum inverse price-income ratio elasticity for public (0.21) and private (0.22) providers (Dzator & Asafu-Ajaye 2004); [b] Income elasticity of ITN demand is proxied by maximum elasticity for socioeconomic groups SES 2-3 (0.459), SES4 (0.140), SES5 (0.067) (Gingrich, Hanson et al. 2011); [c] Malaria prevalence elasticity of ITN uptake is proxied by the minimum male adult (0.0044) and female adult (0.0043) elasticities (Picone, Kibler & Apouey 2013).

The parametrization of the epidemiological impact equations (6.18)-(6.19) relied on basic data on mosquito population mean catch (MC) reductions and mosquito sporozoite rate (SR) reductions associated with (100%) coverage of respectively ITN and IRS (Curtis, Maxwell, Finch & Njunwa 1998). The basic data are presented in Table 6.3 along with the derived household-specific 'maximum impact/100% coverage' parameters on epidemiological model parameters including maximum reduction in (female) mosquito population per person ($\underline{\Delta}\bar{m}_{int,h}^{max}$) and maximum increase in mosquito mortality rate ($\Delta\bar{\mu}_{int,h}^{M,max}$). For the former 'mosquito population, maximum impact' parameter, we could simply apply the observed MC reduction (equivalent to the population reduction impact) from 100% intervention coverage. We applied this reduction, uniformly, across our 19 household types, since no regional or household-specific estimates were available.

In contrast, for the latter 'mosquito mortality rate, maximum impact' parameter (for which we had no direct observations), we used our household-specific epidemiological models (see section 3) to simulate measures of the 'mosquito mortality rate, maximum impact' parameter as the (1) model-consistent increases in mosquito mortality rates ($\Delta\mu^{M}_{int,h}$) following from (2) household- and intervention-specific

reductions in mosquito malaria prevalence rates ($p^{M}_{h,t}$) associated with the SR-reduction following from 100% intervention coverage. Due to differences in our regional household-specific epidemiological model specifications, the 'mosquito mortality rate, maximum impact' parameters ($\Delta\mu^{M,max}_{int,h}$) vary (quite strongly) across our household types (Table 6.3). While parameterization data are presented for both IRS and ITN prevention interventions in Table 6.3, we exclude, as discussed above, IRS interventions from our model applications since regional IRS coverage data are not immediately available. Actual model applications are therefore based on standard single-intervention specifications (Smith, Chitnis, Brie & Tanner 2011) as presented in equations (6.18')-(6.19').

Finally, the household-specific morbidity rates ($\nu_{h,t}$), defined in equation (6.20), were initialized based on household-specific effective ACT coverage rates (see section 5.3) and parametrization of fixed morbidity rates associated with and without effective ACT treatment ($\bar{\nu}^{ACT}, \bar{\nu}^{NoACT}$): (1) a central point estimate for malaria-related absenteeism without ACT treatment (%-share reduction in annual labour supply per malaria episode; $\bar{\nu}^{NoACT}$=4/260≈1.54%) was based on the assumption of 4 workdays lost per malaria episode due to incapacitation, where the latter choice was informed by a literature survey of available (African) statistical evidence (Table 6.4); (2) a point estimate for malaria-related absenteeism with ACT treatment ($\bar{\nu}^{ACT}$=2/260≈0.77%) was based on the assumption of 2 workdays lost per malaria episode since ACT treatment reduces the febrile period to approx. 1 day (Mayxay et al. 2012) and another day of incapacitation was added for diagnosis and seeking treatment.

Table 6.3. Epidemiological impacts of malaria interventions

			Coverage = 100%	
			ITN	IRS
Baseline data[1]				
ΔMean Catch (mosquito population)			-59.1%	-71.7%
ΔSporozoite Rate (mosquito malaria prevalence)			-74.7%	-74.0%
Household-specific epidemiological parameter impacts[2]				
1. Mosquito population reduction ($\Delta m_{int,h}$)				
	H01	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H02	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H03	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H04	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H05	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H06	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H07	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H08	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H09	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H10	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H11	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H12	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H13	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H14	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H15	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H16	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H17	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H18	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
	H19	$\Delta m^{max}_{int,h}$	-71.7%	-59.1%
2. Mosquito mortality rate increase ($\Delta\mu^{M}_{int,h}$)				
	H01	$\Delta\mu^{M,max}_{int,h}$	8.7%	8.8%
	H02	$\Delta\mu^{M,max}_{int,h}$	17.7%	17.9%
	H03	$\Delta\mu^{M,max}_{int,h}$	31.5%	31.9%
	H04	$\Delta\mu^{M,max}_{int,h}$	73.4%	74.5%
	H05	$\Delta\mu^{M,max}_{int,h}$	19.6%	19.8%
	H06	$\Delta\mu^{M,max}_{int,h}$	28.0%	28.3%
	H07	$\Delta\mu^{M,max}_{int,h}$	54.3%	55.1%
	H08	$\Delta\mu^{M,max}_{int,h}$	18.0%	18.2%
	H09	$\Delta\mu^{M,max}_{int,h}$	34.8%	35.2%
	H10	$\Delta\mu^{M,max}_{int,h}$	78.5%	79.8%
	H11	$\Delta\mu^{M,max}_{int,h}$	21.9%	22.1%
	H12	$\Delta\mu^{M,max}_{int,h}$	35.4%	35.8%
	H13	$\Delta\mu^{M,max}_{int,h}$	91.8%	93.4%
	H14	$\Delta\mu^{M,max}_{int,h}$	25.0%	25.3%
	H15	$\Delta\mu^{M,max}_{int,h}$	40.2%	40.7%
	H16	$\Delta\mu^{M,max}_{int,h}$	75.4%	76.6%
	H17	$\Delta\mu^{M,max}_{int,h}$	24.8%	25.1%
	H18	$\Delta\mu^{M,max}_{int,h}$	44.0%	44.6%
	H19	$\Delta\mu^{M,max}_{int,h}$	107.5%	109.5%

Sources: [1]Curtis, Maxwell, Finch & Njunwa (1998); [2]own calculations

Table 6.4. Morbidity effects associated with uncomplicated malaria episodes in Sub-Saharan African countries

Country/region	Year	days ill	workdays lost due to incapacitation (absenteeism)	workdays lost due to low productivity (presenteeism)	Reference
Tanzania	1941[a]	1.5-4 days/episode			Brinkmann & Brinkmann (1991)
East Africa	1944[a]	4-9 days/episode			Brinkmann & Brinkmann (1991)
Malawi	1950[a]	3.4 days/episode			Brinkmann & Brinkmann (1991)
Nigeria	1954-56	2.6 days/episode			Bruce-Chwatt (1963)
Ghana	1955	4.5 days/episode[b]			Bruce-Chwatt (1963)
Liberia	1958[a]	4.2 days/episode			Brinkmann & Brinkmann (1991)
Ethiopia	1961[a]	3-4 days/episode			Brinkmann & Brinkmann (1991)
Nigeria	1963[a]	2.6 days/episode			Brinkmann & Brinkmann (1991)
Uganda	1964-66		1.16 days per year		Hall & Wilks (1967)
Ghana	1965[a]	2-6 days/episode[b]			Brinkmann & Brinkmann (1991)
Rwanda	1965[a]	5.2 days/episode[b]			Brinkmann & Brinkmann (1991)
Ghana	1981[a]	7 days/episode			Brinkmann & Brinkmann (1991)
Togo	1984[a]	5.3 days/episode[b]			Brinkmann & Brinkmann (1991)
Ivory Coast	1987[a]	3.4 days/episode[b]			Brinkmann & Brinkmann (1991)
Burkina Faso	1987		3.5 days/episode		Gazin et al. (1988)
Malawi	1992		2.41 days/episode[iii]	1.21 days/episode[iii]	Ettling, McFarland, Schultz & Chitsulo (1994)
Burkina Faso	1992		3.5 days/episode		Sauerborn et al. (1995)
Burkina Faso	1992		4 days/episode		Guiguemdé et al. (1997)
Kenya	1993	3-7 days/episode	2-4 days/episode	2 days/episode[g]	Leighton & Foster (1993)
Nigeria	1993	2-7 days/episode	1-3 days/episode	3 days/episode[h]	Leighton & Foster (1993)
Ghana	1993		2895 days lost among 1614 cases[c]		Asenso-Okyere & Dzator (1997)
Sudan	1993		6.2 days/episode	2.6 days/episode	Nur (1993)
Nigeria	1998		4-9 days/episode		Onwujekwe, Chima & Okonkwo (2000)
Ethiopia[i]	2000	19 days/episode	14 days/episode		Cropper et al. (2000)
Ethiopia[ii]	2000	22 days/episode	18 days/episode		Cropper et al. (2000)
Mozambique	2001/02		3.4 days/episode[d]		Castillo-Riquelme, McIntyre & Barnes (2008)
South Africa	2001/02		2.4/3.2 days/episode[e]		Castillo-Riquelme, McIntyre & Barnes (2008)
Ethiopia	2003		6.82 days/episode (for 66.3% of cases)		Deressa, Hailemariam & Ali (2007)
Ghana	2003	10.79 days/episode	9.03 days/episode[f]		Asante, Asenso-Okyere & Kusi (2005)
Ghana	2007-2010	3.5 days/episode			Hanlon (2011)
Kenya	2010		3.9-7.8 days/episode		Chuma, Okungu & Molyneux (2010)

Notes: i 'Malaria test' sub-sample; ii full sample; iii estimates derived from information in Ettling et al. (1994), but not consistent with reported overall estimate (2.66 days/ episode); a study year of original study, referenced in Brinkmann & Brinkmann (1991) survey; b child morbidity; c majority of child cases, and number of cases not corrected for labor force participation; d days taken off school or work; e days taken off school or work for SA1/SA2 regions; f aggregate unweighted measure of absenteeism and presenteeism; g presenteeism estimates varied between agriculture (-50% to -75%), industry (-25% to -50%), and services (0% to -25%) ; h presenteeism estimates varied between agriculture (-50%), industry (-25%), and services (-25%);

7. Conclusion

In this paper, we developed a novel and fully integrated epidemiological-demographic-macroeconomic EDM-malaria simulation model framework for modelling of *P. falciparum* malaria transmission in Ghana. The macroeconomic malaria-focussed CGE model (see section 2) was calibrated on the basis of a 2004 malaria-focussed Ghana SAM, where regional households were stratified according to (1) rural-urban location, (2) coastal-forest-savannah eco-region location, and (3) low-medium-high malaria prevalence district location. Based on the regional household stratification, we constructed 19 consistently stratified sets of epidemiological models (see section 3), and demographic models (see section 4). Our regional epidemiological models are MacDonald-Ross compartment models of malaria transmission which have been extended to account for human super-infections, and they are meant to replicate the reputable Swiss Tropical Institute model. They were calibrated on the basis of data from the Malaria Atlas Project and clinical health outcomes are modelled through endogenous application of closed-form piece-wise linear specifications based on seasonal transmission-corrected lookup tables derived from the STI model. Our regional demographic models are specified as annual models with 1-year age groups and extended to include wage-driven interregional and international migration specifications based on Harris-Todaro migration specifications. In order to integrate the region-specific epidemiological and demographic models, the former were transformed from continuous time to bi-weekly discrete time models, where the final 26th time period solution is used as the annual equilibrium solution. Repeated inspection suggests that our epidemiological models achieve rapid convergence. In order to fully integrate the epidemiological and demographic models with our macroeconomic CGE model, we finally specified how effective labour supplies and labour factor ownerships are affected by malaria-related clinical health outcomes (section 5), and how malaria intervention coverage rates, derived from extended household and public demand systems, affect epidemiological model parameters and morbidity health outcomes through generalized multiple-intervention specifications, and determine public and private household costs of malaria-related composite interventions including costs of interventions and administrative and laboratory services (section 6).

Our model framework represents a milestone, as the first fully integrated EDM model framework for any type of infectious disease. The complex specification and integration of regional epidemiological-demographic models within a national macroeconomic model was undertaken with the twin purposes of (1) providing a methodologically novel approach to macroeconomic modelling of malaria transmission in a high transmission intensity setting which captures both perennial and (savannah region) seasonal

transmission, and (2) to provide a tool for policy analysis and consistent assessment of the twin macroeconomic and clinical health disease burdens. Going forward, we aim to use the EDM-malaria framework, described in this paper, to undertake studies of future malaria transmission in Ghana over the coming 20 years (2015-34). The macroeconomic growth assumptions underlying our future baseline projections are outlined in Section 2, and, together with our EDM-malaria model framework, they form the basis for on-going work, where we aim to investigate the magnitudes and interplay of future macroeconomic and health disease burdens, to measure the health and economic impacts of future economic growth and scaling-up of malaria interventions, and to study the importance (or lack thereof) of the general omission of proper epidemiological underpinnings and integration of economic incentive feedback effects in the existing literature on macroeconomic assessment of infectious disease.

Annex A. 2004 Ghana MalSAM accounts

Table A.1. 2004 Ghana MalSAM accounts (excl. factors & households)

Account Identifier	Account description
1. Activities	
A01	Agriculture
A02	Industry
A03	Utilities
A04	Housing and infrastructure
A05	Transport, fuel, motor vehicles and repairs
A06	Trade
A07	Services
A08	Public administration and defense
A09	Education
A10	Health
2. Commodities	
C01	Agriculture
C02	Industry
C03	Utilities
C04	Housing and infrastructure
C05	Transport, fuel, motor vehicles and repairs
C06	Trade
C07	Services
C08	Public administration and defense
C09	Education
C10	Health
3. Other accounts	
TRD	Trade and transportation margins
E	Enterprise
G	Government
T01	Activity tax
T02	Sales tax
T03	Import tariff
T04	Export duty
T05	Direct enterprise tax
T06	Direct household tax
CAP	Savings-investment account
DSTK	Change in stocks
R	Rest of the world

Source: own definitions based on Jensen, Keogh-Brown et al. (2012)

Table A.1 (cont.) 2004 Ghana MalSAM accounts (factors & households)

Account identifier	Household /Factor type	rural-urban location	eco-region location	Gender type	Malaria Prev./ Labour skill	Account description
4. Factors						
F01					Low Skill	GAMA Male low skill labour
F02				Male	Med Skill	GAMA Male med skill labour
F03					High Skill	GAMA Male high skill labour
F04		GAMA			Low Skill	GAMA Female low skill labour
F05				Female	Med Skill	GAMA Female med skill labour
F06					High Skill	GAMA Female high skill labour
F07					Low Skill	Urban Coastal Male low skill labour
F08				Male	Med Skill	Urban Coastal Male med skill labour
F09					High Skill	Urban Coastal Male high skill labour
F10			Coastal		Low Skill	Urban Coastal Female low skill labour
F11				Female	Med Skill	Urban Coastal Female med skill labour
F12					High Skill	Urban Coastal Female high skill labour
F13					Low Skill	Urban Forest Male low skill labour
F14				Male	Med Skill	Urban Forest Male med skill labour
F15					High Skill	Urban Forest Male high skill labour
F16		Urban	Forest		Low Skill	Urban Forest Female low skill labour
F17				Female	Med Skill	Urban Forest Female med skill labour
F18					High Skill	Urban Forest Female high skill labour
F19					Low Skill	Urban Savannah Male low skill labour
F20				Male	Med Skill	Urban Savannah Male med skill labour
F21					High Skill	Urban Savannah Male high skill labour
F22	Labour		Savannah		Low Skill	Urban Savannah Female low skill labour
F23				Female	Med Skill	Urban Savannah Female med skill labour
F24					High Skill	Urban Savannah Female high skill labour
F25					Low Skill	Rural Coastal Male low skill labour
F26				Male	Med Skill	Rural Coastal Male med skill labour
F27					High Skill	Rural Coastal Male high skill labour
F28			Coastal		Low Skill	Rural Coastal Female low skill labour
F29				Female	Med Skill	Rural Coastal Female med skill labour
F30					High Skill	Rural Coastal Female high skill labour
F31					Low Skill	Rural Forest Male low skill labour
F32				Male	Med Skill	Rural Forest Male med skill labour
F33					High Skill	Rural Forest Male high skill labour
F34		Rural	Forest		Low Skill	Rural Forest Female low skill labour
F35				Female	Med Skill	Rural Forest Female med skill labour
F36					High Skill	Rural Forest Female high skill labour
F37					Low Skill	Rural Savannah Male low skill labour
F38				Male	Med Skill	Rural Savannah Male med skill labour
F39					High Skill	Rural Savannah Male high skill labour
F40			Savannah		Low Skill	Rural Savannah Female low skill labour
F41				Female	Med Skill	Rural Savannah Female med skill labour
F42					High Skill	Rural Savannah Female high skill labour
F43	Capital					Capital
5. Households						
H01		GAMA	-		Low Mal. Prev.	Low prevalence GAMA
H02					Low Mal. Prev.	Low prevalence Urban Coastal
H03			Coastal		Med Mal. Prev.	Med prevalence Urban Coastal
H04					High Mal. Prev.	HIgh prevalence Urban Coastal
H05					Low Mal. Prev.	Low prevalence Urban Forest
H06		Urban	Forest		Med Mal. Prev.	Med prevalence Urban Forest
H07					High Mal. Prev.	HIgh prevalence Urban Forest
H08					Low Mal. Prev.	Low prevalence Urban Savannah
H09			Savannah		Med Mal. Prev.	Med prevalence Urban Savannah
H10	Household				High Mal. Prev.	HIgh prevalence Urban Savannah
H11					Low Mal. Prev.	Low prevalence Rural Coastal
H12			Coastal		Med Mal. Prev.	Med prevalence Rural Coastal
H13					High Mal. Prev.	HIgh prevalence Rural Coastal
H14					Low Mal. Prev.	Low prevalence Rural Forest
H15		Rural	Forest		Med Mal. Prev.	Med prevalence Rural Forest
H16					High Mal. Prev.	HIgh prevalence Rural Forest
H17					Low Mal. Prev.	Low prevalence Rural Savannah
H18			Savannah		Med Mal. Prev.	Med prevalence Rural Savannah
H19					High Mal. Prev.	HIgh prevalence Rural Savannah

Source: own definitions based on Jensen, Keogh-Brown et al. (2012)

Annex B. Epidemiological model variable and parameter definitions

Variable definitions

$p^H_{h,tbw}$	true human malaria prevalence rate
$p^M_{h,tbw}$	mosquito malaria prevalence rate
$N_{h,tbw}$	multiplicity of malaria infections in humans
$\lambda^{FOI}_{h,tbw}$	force of infection
$EIR_{h,tbw}$	entomological inoculation rate
$\mu^M_{h,t}$	mortality rate for mosquitoes
$m_{h,t}$	number of female mosquitoes per person
$\tau_{h,age,t}$	number of uncomplicated malaria episodes per person per year
$\mu_{h,age,t}$	malaria mortality rate per person per year
$sp^H_{h,tbw}$	expected human malaria prevalence rate detectable by microscopy; slide prevalence
$q_{h,t}$	correction factor for detectability of malaria infection by microscopy

Parameter definitions

$\bar{\lambda}^S_h$	arrival rate of superinfections
$\bar{\mu}^{\mathrm{S}}$	clearance rate of superinfections
a	human feeding rate of female mosquitoes
b	infectiousness of infective mosquito bites to humans
c	infectiousness of humans to mosquitoes
$\bar{\tau}^{\mathrm{incub}}$	incubation period for mosquitoes
$\bar{\alpha}^{\tau}$	Scaling parameter for morbidity (malaria episodes) clinical outcome specifications

$\bar{\alpha}^{\mu}$ Scaling parameter for excess mortality clinical outcome specifications

$\bar{\beta}_{age}^{\tau,s}$ parameters for morbidity (malaria episodes) clinical outcome polynomial approx. specifications

$\bar{\beta}_{age}^{\mu,s}$ parameters for excess mortality clinical outcome polynomial approx. specifications

$\bar{b}^{min}$ proportion of successful inoculations as the EIR approaches infinity (EIR→∞)

$\overline{eir}^{b}$ EIR at which half the reduction in the FOI (and mosquito infectiousness b) is achieved

$\bar{q}^{min}$ minimum detection rate for malaria prevalence by microscopy (EIR→0)

$\bar{q}^{max}$ maximum detection rate for malaria prevalence by microscopy (EIR→∞)

$\overline{eir}^{q}$ EIR at which half the possible increase in detectability ($\bar{q}^{max} - \bar{q}^{min}$) is achieved

Annex C. Demographic variable and parameter definitions

Variable definitions

$POP^{H}_{h,t}$	Population (by all household types)
$POP_{h,gen,age,t}$	Population (by all household, gender and age groups)
$Deaths_{h,gen,age,t}$	Deaths (by all household, gender and age groups, except infants)
$Births_{h,gen,t}$	Gender-specific infant births (by all household and gender types)
$InfDeaths_{h,gen,t}$	Gender-specific infant deaths (by all household and gender types)
$POP^{migr,net}_{h,gen,age,t}$	Net total immigrant population stocks (by all household, gender, and age groups)
$POP^{imigr,net}_{h,gen,age,t} =$	Net international immigrant population stocks (by all household, gender, and age groups)
$POP^{dmigr,net}_{h,gen,age,t}$	Net domestic immigrant population stocks (by all household, gender, and age groups)
$POPshr^{imigr,LOC2}_{loc2,t}$	Int'l immigrant share of regional population (by rural/urban household types)
$POPshr^{emigr,LOC2}_{loc2,t}$	Int'l emigrant share of regional population (by rural/urban household types)
$POPshr^{dmigr,LOC2}_{loc2,t}$	Domestic immigrant share of regional population (by rural/urban household types)
$POP^{imigr,H}_{h,t}$	Gross int'l immigrant population stocks (by all household types)
$POP^{imigr}_{h,gen,age,t}$	Gross int'l immigrant population stocks (by all household, gender, and age groups)
$POP^{emigr,H}_{h,t}$	Gross int'l emigrant population stocks (by all household types)
$POP^{emigr}_{h,gen,age,t}$	Gross int'l emigrant population stocks (by all household, gender, and age groups)
$POP^{dmigr,LOC2}_{loc2,gen,age,t}$	Gross domestic immigrant population stocks (by rural/urban household types and all gender and age groups)

$POP^{dmigr,net,LOC2}_{loc2,gen,age,t}$	Net domestic immigrant population stocks (by rural/urban household types and all gender and age groups)
$POP^{dmigr,net}_{h,gen,age,t}$	Net domestic immigrant population stocks (by all household, gender, and age groups)
$QFH_{h,flab,t}$	Labour factor ownership (by all household and labour factor types)
$WF_{flab,t}$	Average labour factor wages (by all labour factor types)
$WF^{LOC2}_{loc2,t}$	Average regional wage levels (by rural/urban household types)
WF^{ROW}_{t}	Int'l wage level for workers from the Rest of the World (domestic currency)
$\mu_{h,age,t}$	Malaria excess mortality rates (by household and age groups)

Parameter definitions

$\overline{wf}^{ROW}$	Int'l wage level for workers from the Rest of the World (foreign currency)
$\bar{\mu}^{all\ cause}_{h,gen,age,t}$	Baseline all-cause mortality rates (by household, gender and age groups)
$\bar{\mu}_{h,age,t}$	Baseline malaria excess mortality rates (by household and age groups)

Annex D. Effective labour supply variable and parameter definitions

Variable definitions

$QFS^{eff}_{flab,t}$	Effective labour supply (by labour factor type)
$QFH^{H,eff}_{h,flab,t}$	Effective labour supply (by household and labour factor type)
$QFH^{H}_{h,flab,t}$	Labour force participation (by household and labour factor type)
$QFH^{H,morb,adult}_{h,flab,t}$	Morbidity-related labour supply impact due to adult illness (by household and labour factor type)
$QFH^{H,morb,child}_{h,'female',t}$	Morbidity-related labour supply impact due to child illness (by household and 'female' labour factor type)
$\tau_{h,age,t}$	Age-specific uncomplicated malaria episodes per person per year (by household type)
$MRate_{h,t}$	Malaria morbidity rate = work-years lost per uncomplicated episode

Parameter definitions

$PRate_{gen}$	Labour market participation rates (by gender type)
$SklShr_{h,flab}$	Labour factor skill shares (by household and labour factor types)

Annex E. Health intervention equation variable and parameter definitions

Variable definitions

$COVER_{int,h,t}$	malaria intervention coverage rates (by intervention and household types)
$COVER^{prv}_{int,h,t}$	private malaria intervention coverage rates (by intervention and household types)
$COVER^{pub}_{int,h,t}$	public malaria intervention coverage rates (by intervention and household types)
$COVER^{eff}_{int,h,t}$	effective malaria intervention coverage rates (by intervention and household types)
EG_t	government expenditures
$EH_{h,t}$	household consumption spending (by household types)
EXR_t	exchange rate in local currency per unit of foreign currency
$p^H_{h,t}$	true human malaria prevalence rate (by household types)
$POP^H_{h,t}$	Population (by household types)
$POP_{h,gen,age,t}$	Population (by all household, gender and age groups)
$PM^{INT}_{int,t}$	price of intervention imports in domestic currency (by intervention types)
$PQ_{c,t}/PQ_{int,t}$	composite commodity price (by commodity/intervention types)
$PQ^{INT}_{int,t}$	composite commodity price for interventions (by intervention types)
$PVA_{a,t}$	value-added price or factor income per unit of output (by activity types)
$QD_{c,t}$	quantity sold domestically of domestic output (by commodity types)
$QE_{c,t}$	quantity of commodity exports (by commodity types)
$QFH_{h,f,t}$	household factor ownership (by household and factor types)
$QH_{c,h,t}/QH_{int,h,t}$	quantity of household consumption (by commodity/intervention and household types)

$QINT_{c,a,t}$	quantity of intermediate input (by commodity and activity types)
$QINV_{c,t}$	quantity of investment demand (by commodity types)
$QM_{int,t}^{INT}$	quantity of intervention imports (by intervention types)
$QM_{c,t}$	quantity of commodity imports (by commodity types)
$QQ_{c,t}/QQ_{int,t}$	quantity of goods/composite malaria-related composite intervention commodities supplied to domestic market (by commodity/intervention types)
$QQ_{'c10',int,t}^{ADM}$	quantity of malaria-related administrative and laboratory services supplied to domestic market (by intervention types)
$QQ_{int,t}^{INT}$	quantity of malaria interventions supplied to domestic market (by intervention types)
$QT_{c,t}$	quantity of commodity demanded as trade input (by commodity types)
$QVA_{a,t}$	quantity of value-added (by activity types)
$TINS_{insdng,t}$	direct tax rate (by domestic non-government institutions)
$UPTAKE_{int,h,t}$	intervention uptake rates (by intervention and household types)
$WF_{f,t}$	average factor price (by factor types)
YG_t	government revenue
$YI_{insdng,t}$	income of domestic non-government institutions (by domestic non-government institutions)
$YIF_{'GOV',f,t}$	factor income to government (by factor types)
$\mu_{h,t}^{M}$	mortality rate for mosquitoes (by household types)
$\tau_{h,age,t}$	number of uncomplicated malaria episodes per person per year (by household and age groups)
$m_{h,t}$	number of female mosquitoes per person (by household types)

$MRate_{h,t}$ average morbidity rates, measured by the fraction of a work-year lost per uncomplicated malaria episode (by household types)

Parameter definitions

$\bar{\alpha}_{int}^{upt}$ scale parameters of intervention uptake (by intervention types)

$\bar{\beta}_{c,h}/\bar{\beta}_{int,h}$ marginal share of consumption spending (by commodity/intervention and household types)

$\bar{\beta}_{int}^{ADM}$ Leontief quantity of intermediate input of administrative and laboratory per unit of malaria-related composite intervention commodity output (by intervention types)

$\bar{\beta}_{int}^{INT}$ Leontief quantity of intermediate input of malaria intervention per unit of malaria-related composite intervention commodity output (by intervention types)

$\bar{\beta}_{int}^{upt}$ malaria prevalence elasticities of intervention uptake (by intervention types)

$\bar{\gamma}_{c,h}/\bar{\gamma}_{int,h}$ autonomous consumption (by commodity/intervention and household types)

$\bar{\mu}_{h}^{M}$ baseline mortality rate for mosquitoes (by household types)

$\Delta\bar{\mu}_{int,h}^{M,max}$ change in mortality rate for mosquitoes associated with 100% coverage rates of preventive interventions (by intervention and household types)

$\overline{FSAV}$ foreign savings in foreign currency

$\overline{\imath cd}_{ct,c}$ quantity of trade input commodity per unit of commodity produced and sold domestically (by trade input commodity and traded commodity types)

$\overline{\imath ce}_{ct,c}$ quantity of trade input commodity per unit of exported commodity (by trade input commodity and traded commodity types)

$\overline{\imath cm}_{ct,c}$ quantity of trade input commodity per unit of imported commodity (by trade input commodity and traded commodity types)

$\overline{\imath cm}_{ct,int}^{INT}$ quantity of trade input commodity per unit of imported malaria intervention (by trade input commodity and traded intervention types)

$\bar{m}_{h}$ baseline number of female mosquitoes per person (by household types)

$\Delta\bar{m}_{int,h}^{max}$	change in number of female mosquitoes per person associated with 100% coverage rates of preventive interventions (by intervention and household types)
$\overline{MRate}^{ACT}$	morbidity rates with ACT treatment, measured by the fraction of a work-year lost per uncomplicated malaria episode
$\overline{MRate}^{NoACT}$	morbidity rates without ACT treatment, measured by the fraction of a work-year lost per uncomplicated malaria episode
$\overline{pwm}_c$	price of commodity imports in foreign currency (by commodity types)
$\overline{pwm}_{int,t}^{INT}$	price of intervention imports in foreign currency (by intervention types)
$\overline{pwe}_c$	price of commodity exports in foreign currency (by commodity types)
$\overline{qdst}_c$	quantity of stock change (by commodity types)
$\overline{QG}_c$	quantity of government consumption (by commodity types)
$\overline{QG}_{int,h}^{INT}$	quantity of government consumption (by intervention and household types)
$\overline{trnsfr}_{insdng,'GOV'}$	transfer from Government to domestic non-government institutions (by domestic non-government institutions)
$\overline{trnsfr}_{insd,'ROW'}$	transfer from Rest of the World to domestic institutions (by institution types)
$\overline{trnsfr}_{'GOV','ROW'}$	transfer from Rest of the World to Government
$\overline{trnsfr}_{'ROW',f,t}$	transfer from factor f to institution Rest of the World (by factor types)
$\bar{te}_c$	export tax rate (by commodity types)
$\bar{tf}_f$	direct factor tax rate (by factor types)
$\overline{tm}_c$	import tariff rate for commodities (by commodity types)
$\overline{tm}_{int}^{INT}$	import tariff rate for malaria interventions (by intervention types)
$\bar{tq}_c$	sales tax rate for commodities (by commodity types)
$\bar{tq}_{int}^{INT}$	sales tax rate for malaria interventions (by intervention types)

$\overline{tva}_a$	value-added tax rate (by activity types)

Annex F. Household-specific labour force skill shares (by gender)

Table F.1. Household-specific labour force skill shares (by gender)

Household type	rural/urban region	Ecological region	Malaria prev.-level	Gender	Factor type	Labour factor skill level	Gender-specific skill share
H01	GAMA		Low	Male	F01	Low Skill	18.3%
					F02	Med Skill	41.3%
					F03	High Skill	40.3%
				Female	F04	Low Skill	32.6%
					F05	Med Skill	41.0%
					F06	High Skill	26.4%
H02	Urban	Coastal	Low	Male	F07	Low Skill	28.7%
					F08	Med Skill	46.1%
					F09	High Skill	25.2%
				Female	F10	Low Skill	46.5%
					F11	Med Skill	33.7%
					F12	High Skill	19.9%
H03	Urban	Coastal	Medium	Male	F07	Low Skill	25.0%
					F08	Med Skill	40.4%
					F09	High Skill	34.7%
				Female	F10	Low Skill	42.2%
					F11	Med Skill	40.7%
					F12	High Skill	17.1%
H04	Urban	Coastal	High	Male	F07	Low Skill	33.4%
					F08	Med Skill	51.6%
					F09	High Skill	15.0%
				Female	F10	Low Skill	62.7%
					F11	Med Skill	24.7%
					F12	High Skill	12.6%
H05	Urban	Forest	Low	Male	F13	Low Skill	25.6%
					F14	Med Skill	47.0%
					F15	High Skill	27.4%
				Female	F16	Low Skill	43.9%
					F17	Med Skill	39.5%
					F18	High Skill	16.7%
H06	Urban	Forest	Medium	Male	F13	Low Skill	32.0%
					F14	Med Skill	45.0%
					F15	High Skill	23.1%
				Female	F16	Low Skill	51.4%
					F17	Med Skill	37.1%
					F18	High Skill	11.5%
H07	Urban	Forest	High	Male	F13	Low Skill	40.2%
					F14	Med Skill	48.4%
					F15	High Skill	11.4%
				Female	F16	Low Skill	51.0%
					F17	Med Skill	43.2%
					F18	High Skill	5.7%

Source: 2005/06 GLSS5 household survey (GSS 2008).

Table F.1 (cont.) Household-specific labour force skill shares (by gender)

Household type	rural/urban region	Ecological region	Malaria prev.-level	Gender	Factor type	Labour factor skill level	Gender-specific skill share
H08	Urban	Savannah	Low	Male	F19	Low Skill	32.2%
					F20	Med Skill	50.3%
					F21	High Skill	17.5%
				Female	F22	Low Skill	54.8%
					F23	Med Skill	31.0%
					F24	High Skill	14.2%
H09	Urban	Savannah	Medium	Male	F19	Low Skill	36.7%
					F20	Med Skill	35.4%
					F21	High Skill	28.0%
				Female	F22	Low Skill	56.4%
					F23	Med Skill	33.3%
					F24	High Skill	10.3%
H10	Urban	Savannah	High	Male	F19	Low Skill	61.4%
					F20	Med Skill	16.5%
					F21	High Skill	22.1%
				Female	F22	Low Skill	77.7%
					F23	Med Skill	12.1%
					F24	High Skill	10.2%
H11	Rural	Coastal	Low	Male	F25	Low Skill	52.0%
					F26	Med Skill	39.5%
					F27	High Skill	8.5%
				Female	F28	Low Skill	78.2%
					F29	Med Skill	18.8%
					F30	High Skill	3.0%
H12	Rural	Coastal	Medium	Male	F25	Low Skill	51.0%
					F26	Med Skill	36.3%
					F27	High Skill	12.7%
				Female	F28	Low Skill	78.5%
					F29	Med Skill	19.1%
					F30	High Skill	2.3%
H13	Rural	Coastal	High	Male	F25	Low Skill	57.4%
					F26	Med Skill	29.7%
					F27	High Skill	12.9%
				Female	F28	Low Skill	78.5%
					F29	Med Skill	15.9%
					F30	High Skill	5.6%

Source: 2005/06 GLSS5 household survey (GSS 2008).

Table F.1 (cont.) Household-specific labour force skill shares (by gender)

Household type	rural/urban region	Ecological region	Malaria prev. level	Gender	Factor type	Labour factor skill level	Gender-specific skill share
H14	Rural	Forest	Low	Male	F31	Low Skill	45.9%
					F32	Med Skill	45.4%
					F33	High Skill	8.7%
				Female	F34	Low Skill	71.5%
					F35	Med Skill	26.0%
					F36	High Skill	2.5%
H15	Rural	Forest	Medium	Male	F31	Low Skill	51.4%
					F32	Med Skill	39.9%
					F33	High Skill	8.7%
				Female	F34	Low Skill	71.4%
					F35	Med Skill	25.6%
					F36	High Skill	3.1%
H16	Rural	Forest	High	Male	F31	Low Skill	47.4%
					F32	Med Skill	42.5%
					F33	High Skill	10.1%
				Female	F34	Low Skill	72.7%
					F35	Med Skill	24.3%
					F36	High Skill	3.0%
H17	Rural	Savannah	Low	Male	F37	Low Skill	46.0%
					F38	Med Skill	41.9%
					F39	High Skill	12.1%
				Female	F40	Low Skill	65.7%
					F41	Med Skill	28.1%
					F42	High Skill	6.2%
H18	Rural	Savannah	Medium	Male	F37	Low Skill	69.5%
					F38	Med Skill	24.1%
					F39	High Skill	6.4%
				Female	F40	Low Skill	83.3%
					F41	Med Skill	15.1%
					F42	High Skill	1.6%
H19	Rural	Savannah	High	Male	F37	Low Skill	86.1%
					F38	Med Skill	9.0%
					F39	High Skill	4.9%
				Female	F40	Low Skill	93.7%
					F41	Med Skill	4.4%
					F42	High Skill	1.9%

Source: 2005/06 GLSS5 household survey (GSS 2008).

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