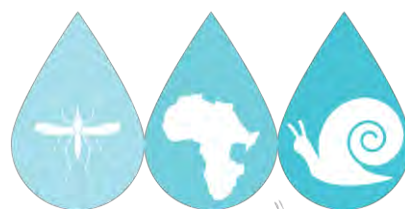


HEALTHY FUTURES



HEALTHY FUTURES

Health, environmental change and adaptive capacity; mapping, examining & anticipating future risks of water-related vector-borne diseases in eastern Africa

Collaborative Project
Seventh Framework Programme
Cooperation

Deliverable D4.6

Future disease risk and vulnerability maps

Grant Agreement no. 266327

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List of acronyms

ACLED	Armed Conflict Location & Event Data Project
C2A	Capacity to anticipate
C2C	Capacity to cope
C2R	Capacity to recover
CGIAR-CSI	Consultative Group on International Agricultural Research - Consortium for Spatial Information
DHS	Demographic and Health Survey
DSF	Decision Support Framework
EAC	East African Community
EIR	Entomological Inoculation Rate
ESA	European Space Agency
FAO	Food and Agriculture Organization of the United Nations
GPWv3	Gridded Population of the World – Version 3
HF	HEALTHY FUTURES
IPCC	Intergovernmental Panel on Climate Change
JRC	Joint Research Center
LMM	Liverpool Malaria Model
OSM	Open Street Map
RCP	Representative Concentration Pathway
RVF	Rift Valley fever
SES	Socio-ecological system
SRTMv4	Shuttle Radar Topography Mission – version 4
VBD	Vector Borne Disease
VECTRI	Vector borne disease community model of ICTP, Trieste
UNEP	United Nations Environment Program
USAID	United States Agency for International Development

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Annex 1 Present and Future Malaria Risk (VECTRI model)
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A. Introduction

The primary aim of task 4.6 and its associated deliverable (D4.6) is to develop spatial assessments of risks of, and vulnerability to, current and future disease burden. To achieve this, the disease-adapted risk and vulnerability framework of deliverable 4.1 (Notenbaert *et al.* 2012), the results of the social vulnerability assessments for the three target diseases of HEALTHY FUTURES (HF) (malaria, schistosomiasis, and Rift Valley fever) (Task 4.1) and output from the disease-specific models of WP3 (Leedale 2014) were integrated under a common risk modelling framework. A spatial assessment of risk of, and vulnerability to, was conducted for the HF target region which comprises the five member states of the East African Community (EAC).

This report starts with the presentation of the spatial characteristics of hazards for the 3 vector borne diseases using the approach adopted in the framework of HF (Notenbaert *et al.* 2012; Leedale 2014). The hazard is quantified for both the present and future taking into account the entomological inoculation rate and specific models projected within specified time frames. This approach enables comparative analysis of spatial variation for each vector borne disease within East African region. In addition the spatial variation of the 3 VBDs in different countries is investigated using existing literature. In this regards, most of published documents are countries, regional or site specific and depict local situation but do not allow comparative analysis regionally. The comparison is furthermore complicated by the heterogeneity of spatial data used in those secondary sources due to different time scale as well as varying environmental and anthropogenic factors. Due to that the harmony in the mapping exercises was not possible.

The final and adapted version of the conceptual risk and vulnerability framework was initially developed in D4.1, the final hazard (i.e., disease) and vulnerability indicators, as well as the methods for modelling risk of current and future disease burden for the three target diseases on the regional scale. Next to the present day assessments, future estimations of risk are built on the two emission scenarios RCP4.5 and RCP8.5 with a medium (~2050) and long-term (~2100) time range as provided from D3.4 (Leedale 2014). RCP8.5 (rising radiative forcing pathway leading to 8.5 W/m² in 2100) represents a high-level change scenario, whereas RCP4.5 reflects a mid-level change of radiative forcing in the atmosphere (stabilisation without overshoot pathway to 4.5 W/m² at stabilisation after 2100). As such it reflects a worst

case scenario (RCP8.5) compared with a more realistic scenario of RCP4.5 (compared to RCP2.6) if mitigation efforts would be fully addressed. The social vulnerability component of risk is kept constant to present-day conditions, due to the lack of spatially disaggregated and future estimations of socioeconomic indicators. Results of the risk modelling are provided in the HF Atlas¹ for interactive exploration. Additionally, we integrate future estimations of key socioeconomic indicators on the national level - based on SSP2 assumptions - in the vulnerability section of the Atlas (Kienberger *et al.* 2013), to allow the estimations of future trends of key socioeconomic factors. Finally, the risk modelling outputs were presented at a stakeholder workshop, held in Nairobi in November 2014. The workshop was part of an iterative process of developing a common decision support framework (DSF) for HF (see WP5).

B. Methodology

The spatial assessment of risk for the three target VBDs was based on a specific approach designed to model spatial multi-dimensional, thus latent, and complex phenomena. The methodology used for the modelling of risk builds on the concept of *integrated geons* (Lang *et al.* 2014), which are defined as integrative (due to the integration of multiple indicators), homogenous spatial objects, in terms of their underlying policy-relevant indicators (Lang *et al.* 2014). An enhanced generic workflow to model integrated geons, following a four-stage procedure (see below), is provided in more detail in Kienberger and Hagenlocher (2014).

The advantage of using geons as reporting units compared with administrative boundaries or a continuous grid is the process-oriented presentation of results as well as its spatial-explicitness in conjunction with unique characteristics for each region (e.g. see Hagenlocher *et al.*, 2014a). The use of geons allows homogenous regions to be identified, for which tailored and spatially explicit (as well as trans-border) interventions can be developed. Geons can also be characterised through specific metrics and measures, which allow the multi-dimensionality of the spatial phenomenon of interest to be integrated, while simultaneously highlighting the separate components of risk, vulnerability domains and single indicators. Thus geons do not mask out spatial variations within administrative units, but much rather help to identify policy-relevant, workable spatial units for intervention development and implementation as well as the monitoring of temporal trends.

¹ Accessible through the project website <http://www.healthyfutures.eu/>

As the spatial assessment of risk consider as point of departure the spatial distribution of the hazards, it is necessary here to explain the methodology used to quantify the hazards for the 3 vector borne diseases. To improve the understanding of the relationship between climate drivers and disease, HF project has developed a number of dynamical modelling for the three target diseases. The modelling platforms enable disease transmission to be modelled in time and space in response to changing climate and in some cases environmental factors.

- The modelling of malaria used both temperature and precipitation. While precipitation provides the temporary breeding sites necessary for the key Anopheles vectors to breed, temperature affects the lifecycles of both the adult and immature vector as well as the plasmodium parasite development rate in the adult vector after infection (Craig *et al.* 1999). Two diseases models are used to model malaria: The Liverpool Malaria Model (LMM) (Hoshen & Morse 2004) and the vector borne disease community model of ICTP Trieste (VECTRI) (Tompkins & Ermert 2013). The approach used in the modelling consists in the representation of delay between the rainfall and malaria transmission. The Liverpool model employs a simple linear relationship between rainfall and female egg-laying, while VECTRI uses a simple surface hydrology model changing fractional coverable of the small temporary pools.

- A mathematical modelling approach has been used to generate hazard maps highlighting areas where temperatures may become suitable for increases schistosome transmission, and where there is a risk of new endemic areas developing. The modelling approach of schistosomiasis is explained by McCreesh & Booth (2014) : the schistosomiasis: a snail egg was introduced into the model each hour with a probability of 0.00012 (which gives an average rate of one snail egg per model year). As non-temperature-dependent egg mortality is simulated using reduced egg production rates in the model, this is equivalent to a “real life” egg introduction rate of 10 eggs/year. To reduce model stochasticity, a rate of miracidium introduction of 30/hour was simulated.

- In the case of RVF, the LMM is used to model the dynamics of RVF transmission and its dependence on climatic factors. The model distinguishes between two different types of vector that transmit RVF: *Aedes* (reservoir vector) and *Culex* (amplifying vector). The model also sorts the host component of the model by treating mature and immature livestock as separate dynamic variables due to significantly different transmission characteristics. The LRVF is a dynamic, process-based model that employs mean-field

assumptions, and follows a deterministic compartmental approach to the epidemiology. The model is driven by climate input data and provides an output that indicates which areas are vulnerable to RVF epizootics as a result of the state of the climate and predicted livestock immunity at the time.

The methodology of the spatial risk assessment for HF and its three target diseases follows a generic four-stage procedure (Kienberger & Hagenlocher 2014) which is applied for present-day and future risk estimations:

1. The first relevant step is the definition of the conceptual risk and vulnerability framework (see Figure 1), which includes defining the risk concept and identifying relevant indicators of the vulnerability and hazard (disease) components of risk. This is a crucial step for any modelling exercise, following the maxim that *what is badly defined is likely to be badly modelled*. A thorough desk study of recent, thematically-relevant literature is indispensable at this stage (see also D4.1), as are expert consultations, which took place on multiple occasions within the HF project (e.g. at the stakeholder workshop in Nairobi, Kenya in spring 2014). Since the deductive approach used for this assessment relies on expert-based knowledge and review of scientific expertise, whether indirect through secondary sources, or direct through consultations, the indicator development can be seen as a iterative and ongoing process, requiring frequent adjusting and feedbacks, due to limitations such as the saliency, credibility, and legitimacy of the proposed initial indicator set and particularly data quality and availability. The selection process resulted in a first set (or wish list) of indicators.
2. The second step includes the finalisation of the indicator set for each of the three diseases to render a dataset that can be subsequently used for modelling. During this stage, the data is pre-processed. This includes the interpolation of point data using kernel density estimators for survey data, resampling the data to 10 x 10 km² grids, the analysis and imputing of missing data, the detection and treatment of extreme values/outliers, as well as an assessment and ultimately the reduction of existing multicollinearities in the data. The original indicator values are then normalised through a linear min-max function to render them comparable.
3. The third step includes the spatial modelling of risk and its components, i.e. hazard (disease) and social vulnerability. Prior to the modelling, the indicators were weighted by local and regional domain experts (seven experts for malaria, seven experts for

schistosomiasis, and 24 for RVF) using budget allocation, i.e. experts were asked to allocate 100 points to the final set of indicators for each disease. By taking the mean value of the expert ratings, and standardising them to sum up to one, a weighting for each of the indicators was produced. In this way, indicators that are considered to be more/less important than others are attributed with greater/lower weightings. Homogeneous regions of disease risk were then delineated using a multi-resolution segmentation method (Baatz & Schäpe 2000) and automatically parameterised through a statistical analysis of the input layers (Drăgut *et al.* 2014), which takes into account the expert weights identified in the previous step. The modelling is built on a step-wise spatial delineation of the different components of risk (Figure 2). It is assumed that the hazard indicator reflects the suitable environmental conditions for disease occurrence. Based on the delineation of environmentally based hazard regions, subsequently the different domains of vulnerability are integrated towards a common measure of risk integrating all relevant indicators from the hazard and vulnerability domain. The risk indices itself (present and future) for the three target diseases are calculated using the geometric mean of the hazard indicator combined with the respective vulnerability index (for an overview of aggregation methods (OECD 2008). The geometric mean involves the multiplication of the equally weighted hazard and vulnerability values to arrive at a final risk index. As it is assumed that, where non-suitable environmental/hazard conditions exists (hazard value=0; and vice versa non-suitable vulnerability conditions; vulnerability index value=0), the overall risk value is zero, the geometric mean is the most suitable among the different aggregation schemes. This ‘compensation’ is best reflected by the multiplicative nature of the geometric mean (OECD 2008; Fritzsche *et al.* 2014). The vulnerability index itself was calculated as the vector magnitude combining the different vulnerability indicators reflecting its distance within an n -dimensional indicator (feature) space, where n reflects the number of indicators (see also Kienberger and Hagenlocher, 2014). For each disease, the analysis was carried out for the present (status quo) as well as for the future by using two different emission scenarios, RCP4.5 and RCP 8.5, in 10 year intervals.

4. Lastly, in the fourth stage of the methodology, the **results are visualised** using pie/bar charts in order to explore the relative contributions of the indicators to the final scores.

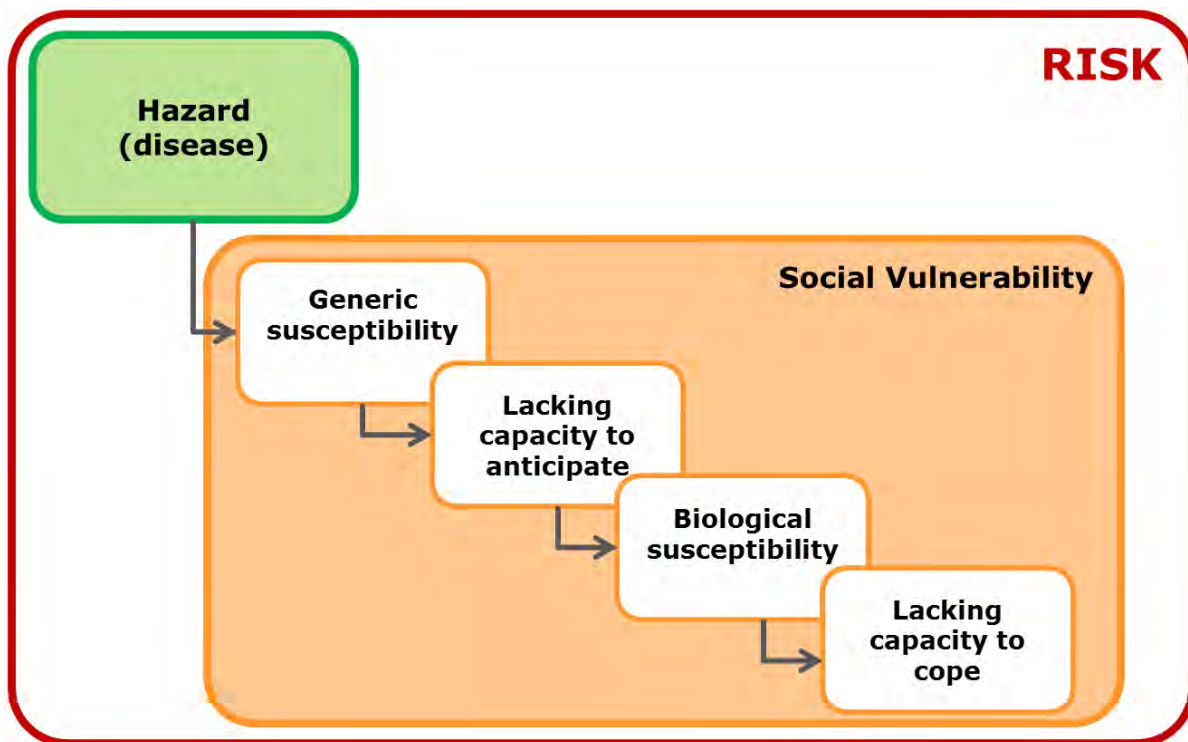


Figure 1: Modelling framework for delineating homogeneous regions of disease risk while taking into account both hazard (disease) and social vulnerability of the population

C. Defining hazard, risk and vulnerability to Vector Borne diseases

The concept of risk and vulnerability is promising for linking disease prevention and control strategies with development agendas and climate change adaptation, as it helps to identify potential intervention options for reducing overall disease risk and strengthening resilience to VBDs in both a pro- and re-active fashion.

Concepts and terminologies of risk, vulnerability and related terms such as resilience or adaptive capacity are manifold and vary between different schools of thought. Within the climate change research arena, the previous IPCC approach (IPCC 2001, 2007) conceptualised vulnerability as a function of exposure, sensitivity, and adaptive capacity (Füssel 2007). By comparison, the disaster risk reduction community defined risk as an integrative concept defined by vulnerability, exposure and hazard. Studies in the context of public health either use (the previous) IPCC-based concepts, or understand risk simply as the likelihood of disease occurrence (Kienberger & Hagenlocher 2014).

With the fifth IPCC assessment reports (AR5; IPCC, 2012, 2014) a significant change in the understanding of risk and vulnerability in the context of climate change adaptation has

occurred. The result is a risk framework that aims to unify the different perspectives and provide a common terminology across the different disciplines (Figure 2). As such, risk is defined by three components: the (social) vulnerability, hazard and exposure. In line with that, the IPCC reports stress that risk management, adaptation and action on climate change should be placed in the context of a planning and analysis framework that considers societal issues along with environmental factors.

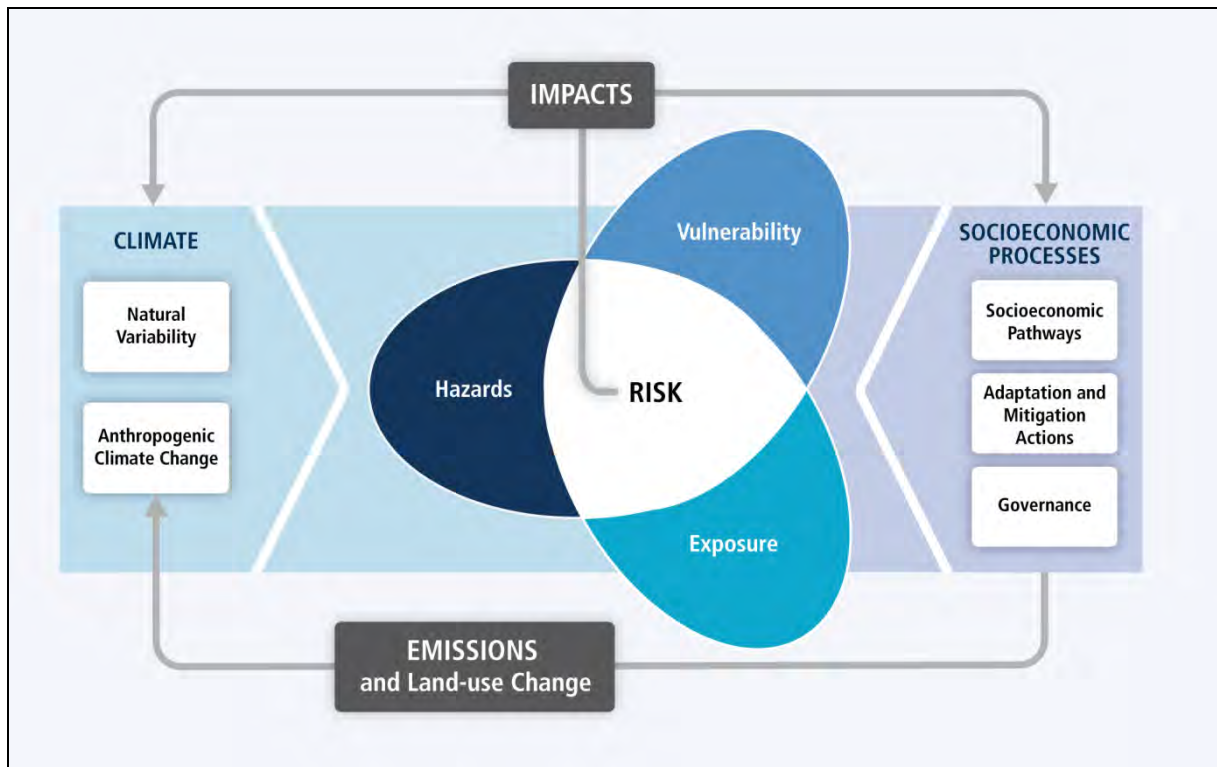


Figure 2: IPCC risk and vulnerability framework
Source: IPCC, 2014.

Understanding disease risk management as a social process allows for a shift in focus from responding to disease prevalence alone, towards a holistic understanding of disease risk. This requires knowledge about how human interactions with the natural environment lead to the spread and prevalence of diseases, and how society is vulnerable to the potential burden of these diseases. Such an approach requires an understanding of the vulnerability of the population, including the allocation and distribution of social and economic resources that can work for, or against, the achievement of reduced diseases impacts (IPCC 2012).

Against this background a holistic conceptual risk and vulnerability framework was developed and further adapted throughout HF (Notenbaert *et al.* 2012) that (i) considers the notion of multiple inter-related factors contributing to disease risk, (ii) provides a clear framing of risk and vulnerability in-line with current IPCC recommendations, (iii) establishes

a clear link to risk governance, climate change adaptation and related intervention measures, (iv) allows the identification of possible development pathways, and finally, (v) provides a holistic view of disease risk considering spatial and temporal scales.

In the HF disease risk framework (Figure 3), risk is defined as the potential occurrence of harmful consequences or losses (i.e., the potential burden of diseases) resulting from interactions between VBDs and vulnerable conditions of different population groups. The proposed framework reflects the multi-faceted nature of vulnerability, accounting for key causal factors such as (generic and biological) susceptibility (both generic and biological) and lack of resilience.

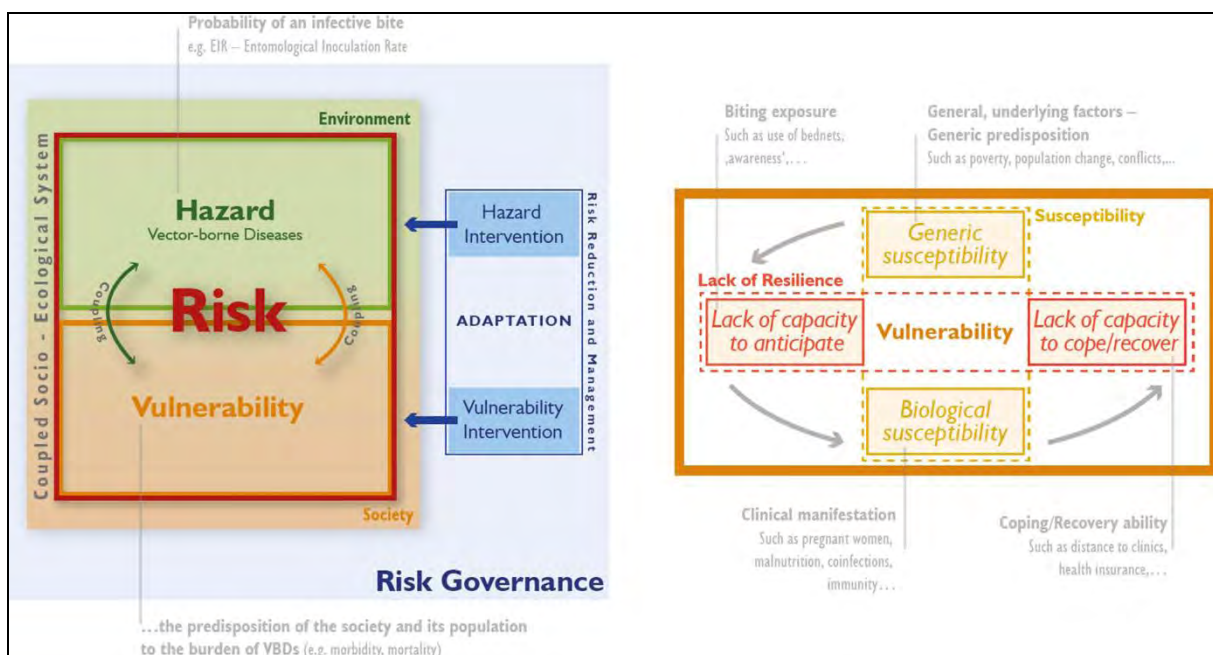


Figure 3: HEALTHY FUTURES conceptual risk and vulnerability framework
Kienberger and Hagenlocher, 2014

The framework was initially developed and proposed in D4.1 (Notenbaert *et al.* 2012) but was further enhanced through feedback gained from the scientific and policy communities, and including selected decision makers in the fields of human and animal health in eastern Africa. The risk framework itself and its application through the modelling of social vulnerability to malaria is presented in more detail in Kienberger and Hagenlocher (2014). The following paragraphs provide an overview and definition of the core elements of the framework (Notenbaert *et al.* 2012; Hagenlocher *et al.* 2013; Hagenlocher *et al.* 2014; Kienberger & Hagenlocher 2014):

- A **hazard** in the context of water-related VBDs is defined as the potentiality of disease occurrence, which may have a negative impact on social assets in a given area and over a given period of time. Hazards include latent conditions that can represent future

threats and are characterised by their location, magnitude, frequency and probability. An example for malaria is the probability of an infective bite, which can be represented through the Entomological Inoculation Rate (EIR). In the framework of this report hazards for the 3 VBDs was estimated, first, using climate and environmental drivers in the modelling exercise that gave outputs in terms of current hazards distribution as well as future projections. Input data for precipitation and temperature were used in the models. Hazard distribution was also identified through literature review, the ultimate aim being the comparison with spatial distribution of vulnerability.

- **Vulnerability** is defined as the predisposition of the society and its population to the burden of water-related VBDs, considering spatial and temporal differences in their susceptibility and lack of resilience. Vulnerability largely rests within the condition and dynamics of the coupled socio-ecological system (SES) exposed to VBDs. Due to its multi-faceted nature it is however mainly linked to societal conditions and processes. In the HF risk framework, vulnerability is seen as a dynamic process that represents the conditions set by the environment and the characteristics and actions of the vulnerable populations themselves, whereby dynamic is understood as the change of factors of vulnerability (and risk) over time.

The framework was designed to be holistic and integrative in a sense that it can be applied heuristically to guide the assessment of risk and vulnerability to several water-related VBDs (here: malaria, schistosomiasis, and RVF) at different spatial or temporal scales. Depending on the disease that is addressed, different indicators (and indicator weights) for modelling disease risk and/or vulnerability were considered to be relevant. In the framework, vulnerability rests largely within the social dimension, which encompasses various socioeconomic and demographic factors and could be extended to institutional, ecological, cultural (or if relevant physical) dimensions. Thereby vulnerability is defined by susceptibility and lack of resilience (Figure 3):

- **Susceptibility** represents the propensity of societies and their population to be negatively affected by a VBD. Thereby a distinction is made between generic susceptibility and biological susceptibility:
 - **Generic susceptibility** encompasses general underlying factors and the general predisposition of societies to malaria (e.g. poverty, population change, conflicts, etc.).

- **Biological susceptibility** relates to the clinical manifestation of malaria, which depends for instance on malnutrition, disease co-infection or immunity and provides an important link to the hazard component of the risk framework.
- **Lack of resilience** refers to limitations in the capacity of societies and their population to respond and absorb negative impacts, through, for example, limited capacity to anticipate, respond to and recover from diseases.
 - The **capacity to anticipate** (C2A) itself entails a coherent set of strategies or programs and social capital available before the disease hazard arises and deals mainly with the reduction of biting exposure (e.g. use of bed nets, awareness, early warning systems etc.).
 - The **capacity to cope** (C2C) refers to the ability of people, organisations, systems and/or communities, using available skills and resources to face and manage adverse conditions arising from endemic and epidemic diseases (such as distance to clinics).
 - The **capacity to recover** (C2R) refers to the capacity to restore adequate and sustainable living conditions, as well having the capacities to overcome or manage the disease in a way that allows living in a physically healthy way (e.g. the availability of adequate treatment and health insurance).
- Compared to **adaptation** processes and adaptive capacities, these capacities focus mainly on the ability to maintain the system's functionality in the light of VBDs impacting the society or system. Adaptation deals with the ability of a community or a system to learn from present and past disease outbreaks and to change existing practices for potential future changes in environmental as well as societal conditions.

Through the integrative and decomposable nature of the framework, it serves as a forward-looking guidance tool for the identification and development of systems of indicators of risk, hazard and vulnerability relevant for assessments at different spatial and temporal scales. In addition, the framework helps to identify targeted intervention measures – at the hazard and vulnerability level – with the ultimate aim to reduce risk to – and finally the impact of - VBDs (Kienberger & Hagenlocher 2014).

D. Results and discussion

1. Current disease hazards

1.2. Malaria

1.2.1. Regional level

When it comes to understand the hazard characteristics the analysis can take into account different factors and make use of certain methods. The malaria transmission could be better understood if regional scale dynamical models account for the role of climate variability and population dynamics. According to McKenzie (2000), models of malaria transmission are useful tools for understanding the disease dynamic and have long been applied to assess the potential for intervention. As weather parameters such as temperature and precipitation are important in determining the disease niche, there is also potential, given accurate forecasts/projections of these parameters, to use malaria models that account for climate in early warning systems in epidemic regions or to assess potential shifts in niche regions under climate change scenarios (Lafferty 2009).

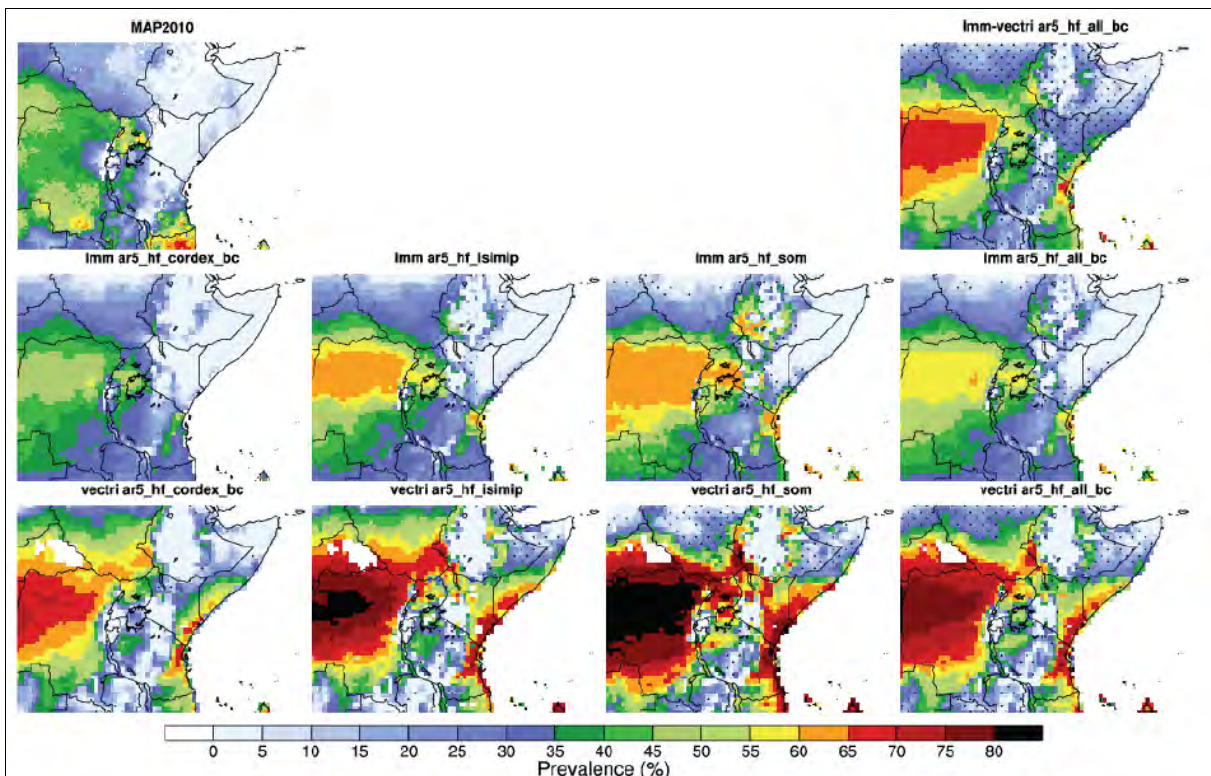


Figure 4: Observed MAP₂₀₁₀ and simulated mean malaria prevalence over East Africa
Source: Leedale, 2014.

Figure 4 compares Malaria Atlas project (Map₂₀₁₀) statistical analysis developed (Gething *et al.* 2011) with the model output of VECTRI and LMM. It comes out from the comparison that malaria prevalence is overestimated by both VECTRI & LMM. The overestimation is higher

for VECTRI compared to LMM. The main cause is attributed to the established immunity that is not taken into account.

1.2.2. Country level

a. Malaria prevalence in Rwanda

The analysis of malaria prevalence conducted in 1962 divided Rwanda into four malaria ecozones. The subdivision is based on elevation, climate plasmodium parasite infection and disease vectors (Meyus *et al.* 1962). This stratification was confirmed 20 years later (Ivorra Cano 1983): the first layer extends from Kivu Lake to the Nile-Congo Crest between 1461 m and 1800 m of altitude; where the malaria prevalence rates ranged between 5% and 30%. The second stratum is Nile-Congo Crest, long of 160 km and 20 to 50 km wide, located in the East of the first layer between 1,800 meters and 3000 meters altitude. The prevalence rate in this zone was less than 2%. The third stratum is located at Central Plateau level where altitude varies between 1,000 m and 2,000 m (Government of Rwanda 2006). The prevalence rate of malaria varies between 10 % and 50 % and many epidemics have been recorded in this stratum. The fourth layer covers the eastern lowlands of the central plateau with altitudes of 1000-1500 meters, where malaria is endemic and appears to be stable (Meyus *et al.* 1962; Ivorra Cano 1967; Vermeylen 1967; Ivorra Cano 1983, 1994; PNILP 2005; Government of Rwanda 2006).

The modelling results of malaria incidences show that the spatial distribution of malaria reflects some similarities with the main strata previously identified based on environmental factors like elevation and climate. Within these major strata, a micro-stratification is possible due to the topography as well as the farming activities in bottom valleys. Indeed, a transversal study among children of Bungwe (between 2,000 m and 3,000 meters of elevation) and in Nyarurema zone (1,500 meters of elevation) showed a difference of malaria prevalence in these two zones that are primarily agricultural areas (Rusanganwa 1999). In the mountain area of Bungwe, it was noted a malaria prevalence of 2.2% at hilltops against 16.4% in the valleys. In the lower altitudes of Nyarurema zone, malaria prevalence rates were not significantly different (39.7% at the hilltop against 41.4% in the bottom valley) (Rusanganwa, 1999). This indicates that malaria is now present in areas and at altitudes where the disease was not previously a major public health concern (Government of Rwanda 2006).

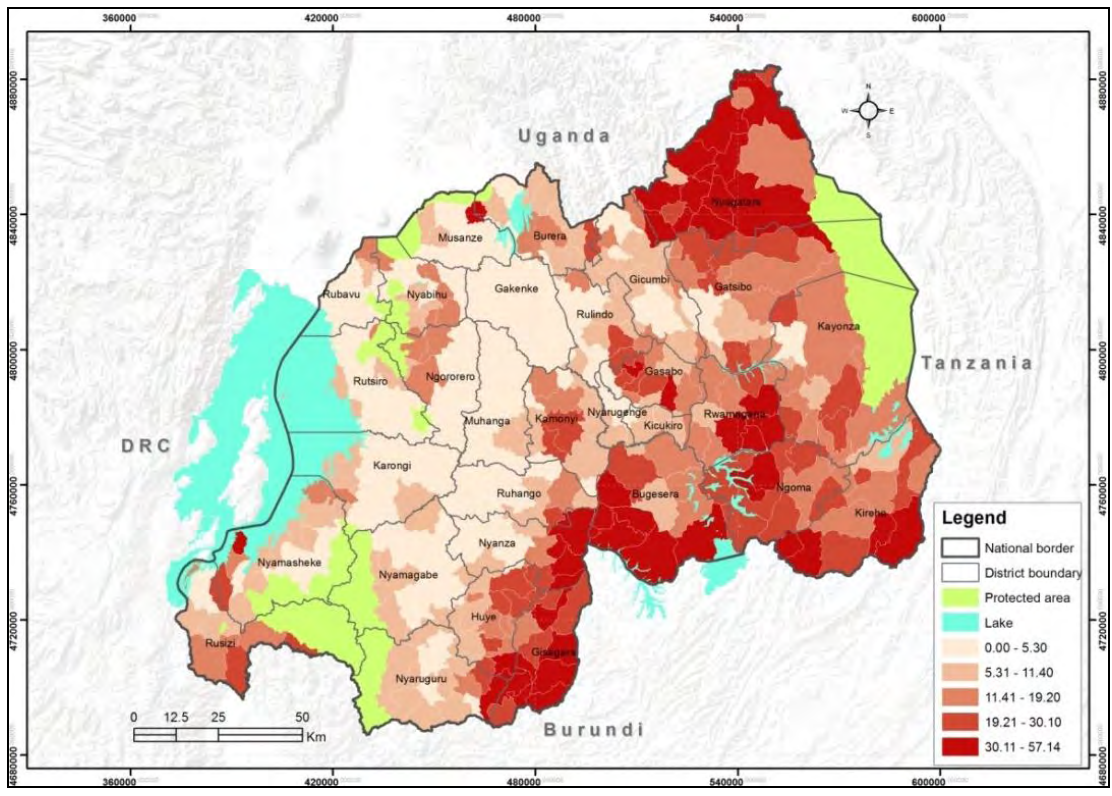


Figure 5: Malaria infection rate in Rwanda in 2012
 Source: MOPDD/Rwanda Biomedical Center

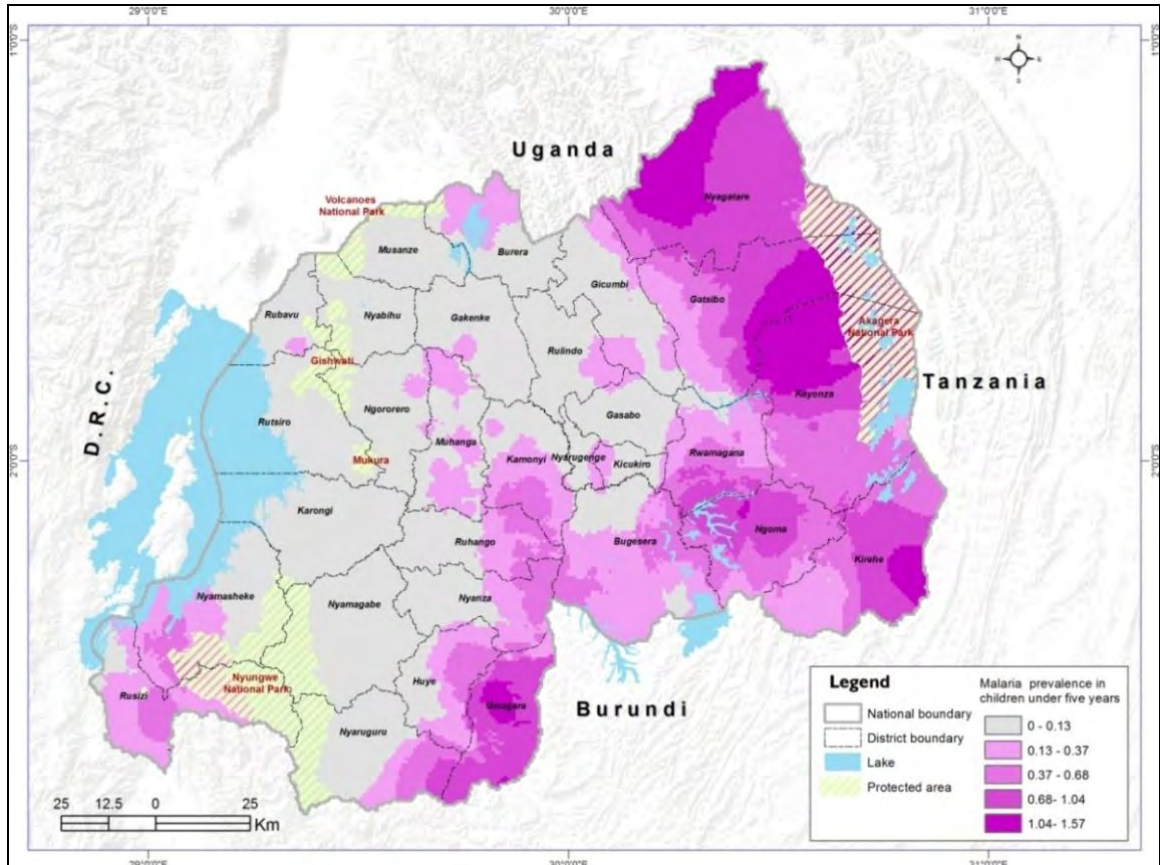


Figure 6: Spatial distribution of malaria prevalence in children less than five years of age in Rwanda
 Source: Demographic and Health Survey 2010

Figure 6 indicates that malaria prevalence in children under five years of age is high in the lowland Eastern Province, including Bugesera District as well as the part of Ruhango and Kamonyi districts dominated by wetland; the region bordering Burundi in the south; in the extreme southwest in the locality of Rusizi. High prevalence is also found in the high altitude of northern part of Rwanda around Butaro.

b. Malaria prevalence in Uganda

Malaria continues to be a major public health problem and the most frequently reported disease at both public and private health facilities in Uganda. Clinically-diagnosed malaria is the leading cause of morbidity and mortality, accounting for 25-40% of outpatient visits at health facilities, 15-20% of hospital admissions, and 9-14% of all hospital deaths (Yawe 2014). Data from highland and lowland areas in western Uganda showed steadily increasing numbers of malaria cases and deaths in district hospitals from 1991 to 2000, with a two-fold to four-fold overall increase in the number of children admitted to hospital with the disease (O'Meara *et al.* 2010). In an area of moderate transmission, a slight decline in the proportion of positive blood films was observed in Uganda in 2007 (O'Meara *et al.* 2010). Uganda presents among the highest reported malaria transmission rates in the world and has the third highest number of annual deaths from malaria in Africa, with approximately 16 million cases reported in 2013 and over 23,000 deaths annually (Government of Uganda 2014b).

In Uganda, malaria prevalence is associated with socio-economic and demographic variables. At household level malaria prevalence is associated with crop choices, wealth, education, gender and bed-nets use with malaria risk differing across age groups. The cultivation of maize, rice, cotton, sweet potatoes/yams, beans, millets and a variety of tree crops have been identified as potentially impacting rural malaria transmission (Wielgosz *et al.* 2013; Wielgosz *et al.* 2014).

In the fight against malaria, households in Uganda are spending a part of the income on treatment when the disease is diagnosed or suspected. The socio-economic impact of malaria includes out-of-pocket expenditure for consultation fees, drugs, transport and subsistence at a distant health facility. These costs are estimated to be between USD 0.41 and USD 3.88 per person per month (equivalent to USD 1.88 and USD 26 per household). A single episode of malaria costs a family on average 9 US dollars, or 3% of annual income. Workers suffering from malaria may be unable to work for an estimated 5-20 days per episode. Given that many people are infected multiple times a year, this has substantial financial consequences to families. Moreover, a poor family in a malaria endemic area may spend up to 25% of the household income on malaria prevention and treatment (Government of Uganda 2014a).

A decline in malaria prevalence would therefore imply a decline in spending of household budgets. Variation in malaria prevalence exists between urban and rural areas with urban households being associated with less malaria compared to rural households (Wielgosz *et al.* 2014). Research has also shown that male headed households are associated with lower malaria prevalence than female-headed households as the former possess more economic capability to access treatment (WHO 2007).

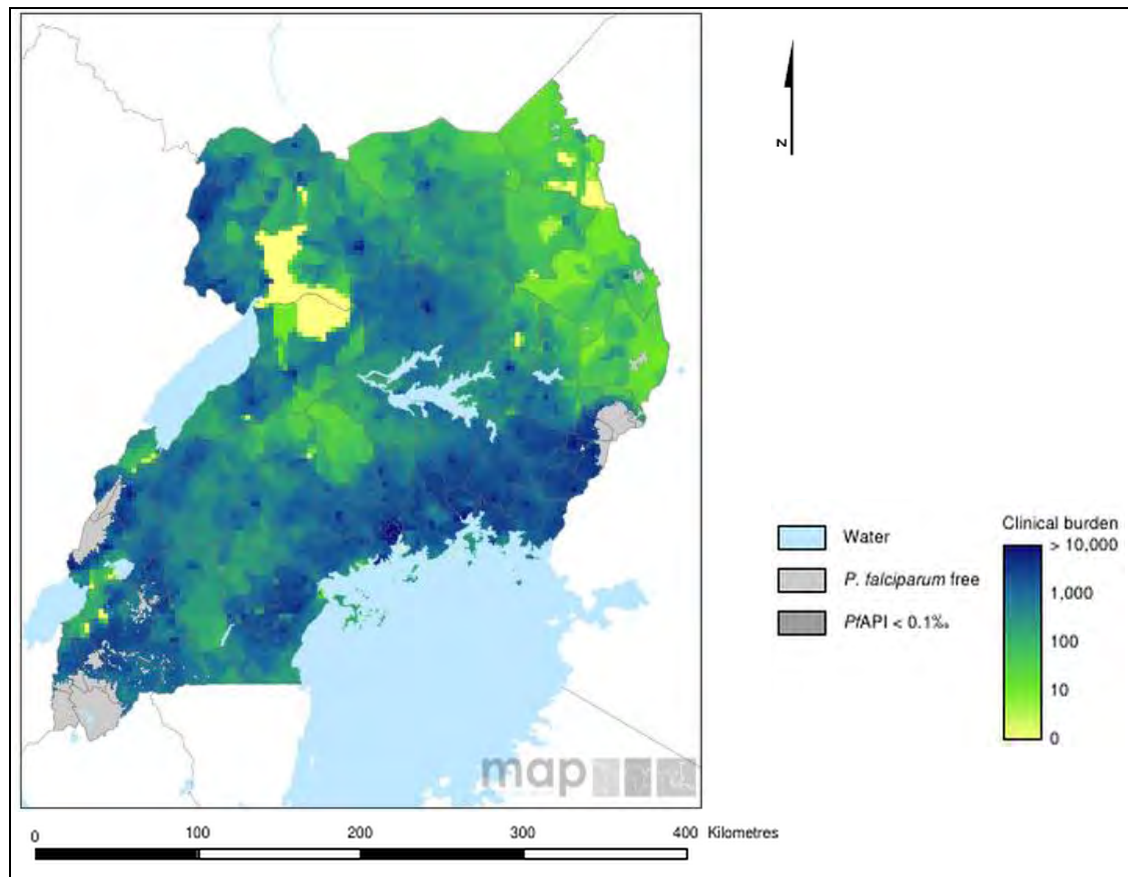


Figure 7: The clinical burden of *Plasmodium falciparum* map in Uganda in 2007
Source: WHO, Malaria Atlas Project.

Figure 7 indicate that malaria infection is highest in the region bordering Lake Victoria. But the infection is also present in the Centre, South and South West of the country.

To explain the increase of malaria transmission in Uganda there have been several hypothesis proposed including land-use/land cover changes, global climate changes, increased drug resistance, cessation of malaria control activities, demographic changes (Imbahale *et al.* 2012), and topography.

c. Malaria prevalence in Kenya

Kenya has four malaria epidemiological zones, largely determined by altitude, rainfall patterns and temperature (Ojaka *et al.* 2014). Those zones are:

- **Endemic areas** as areas of stable malaria (western, Nyanza and coastal provinces fall in this category). They have altitudes ranging from 0 to 1300 m around Lake Victoria in western Kenya and in the coastal regions. In those areas, malaria transmission is intense throughout the year, with annual entomological inoculation rates of 30–100 (Ojaka *et al.* 2014).

- **Seasonal transmission areas** comprising arid and semi-arid areas of northern and south-eastern parts of the country. Temperatures are usually high and water pools created during the rainy season provide breeding sites for the malaria vectors. El Niño induced floods in these areas also result in epidemic outbreaks with high morbidity rates. Eastern and North Eastern Provinces and parts of Central Province fall in this seasonal transmission zone (Ojaka *et al.* 2014).
- **Epidemic-prone areas** of the Western highlands of Kenya. In those areas Malaria transmission is seasonal, with considerable year-to-year variation. Epidemics are associated with minimum temperatures around 18°C. This increase in minimum temperatures during the long rains favours and sustains vector breeding, resulting in increased intensity of malaria transmission. Rift Valley Province and some parts of Nyanza Province fall in this zone (Tonui *et al.* 2013; Ojaka *et al.* 2014).
- **Low risk malaria areas:** This zone covers the central highlands of Kenya, including Nairobi, where temperatures are too low to allow completion of the development cycle of the malaria parasite in the mosquito vector (Ernst *et al.* 2006; Ojaka *et al.* 2014).
- **Pediatric** malaria admissions in Kenya declined by 75 % between 2003 and 2007 in the coastal area while in central Kenya, the proportion of malaria outpatient visits reduced from 40 % to 0 % between 2000 and 2006 with the largest decline between 2003 and 2005. The lowland areas around Lake Victoria in Western Kenya have historically been characterised by very high transmission with entomological inoculation rates estimated to be as high as 250 infectious bites per person). Following the widespread introduction of long-lasting insecticide-treated nets, the burden of malaria has significantly reduced in many parts of the country, but transmission in western Kenya remains high for over 30 years. In this area, the community prevalence of the malaria parasite parasitemia (the number of people with parasites in their blood) among children below five years declined from 70 per cent in 1997 to around 40 per cent in 2008. While since 2008, transmission intensity and malaria prevalence has stagnated (WHO 2013).

In Western Kenya, malaria transmission in the lowland areas around Lake Victoria has historically been very high, with entomological inoculation rates estimated to be as high as 250 infectious bites per person per year. Between 2003 and 2007, a 16% decline in malaria specific mortality data compiled from pediatric inpatient records of 17 hospitals across Kenya (including hospitals on the coast and in western Kenya) showed different temporal trends in malaria admissions between 1999 and 2008 (Ochomo *et al.* 2013). Western Kenya has a high number of people who are asymptomatic (carrying the malaria parasite but having no

symptoms) and up to 50 per cent of parasitemic individuals in community-based cross-sectional surveys report not having fever anytime during the previous two weeks (O'Meara *et al.* 2010).

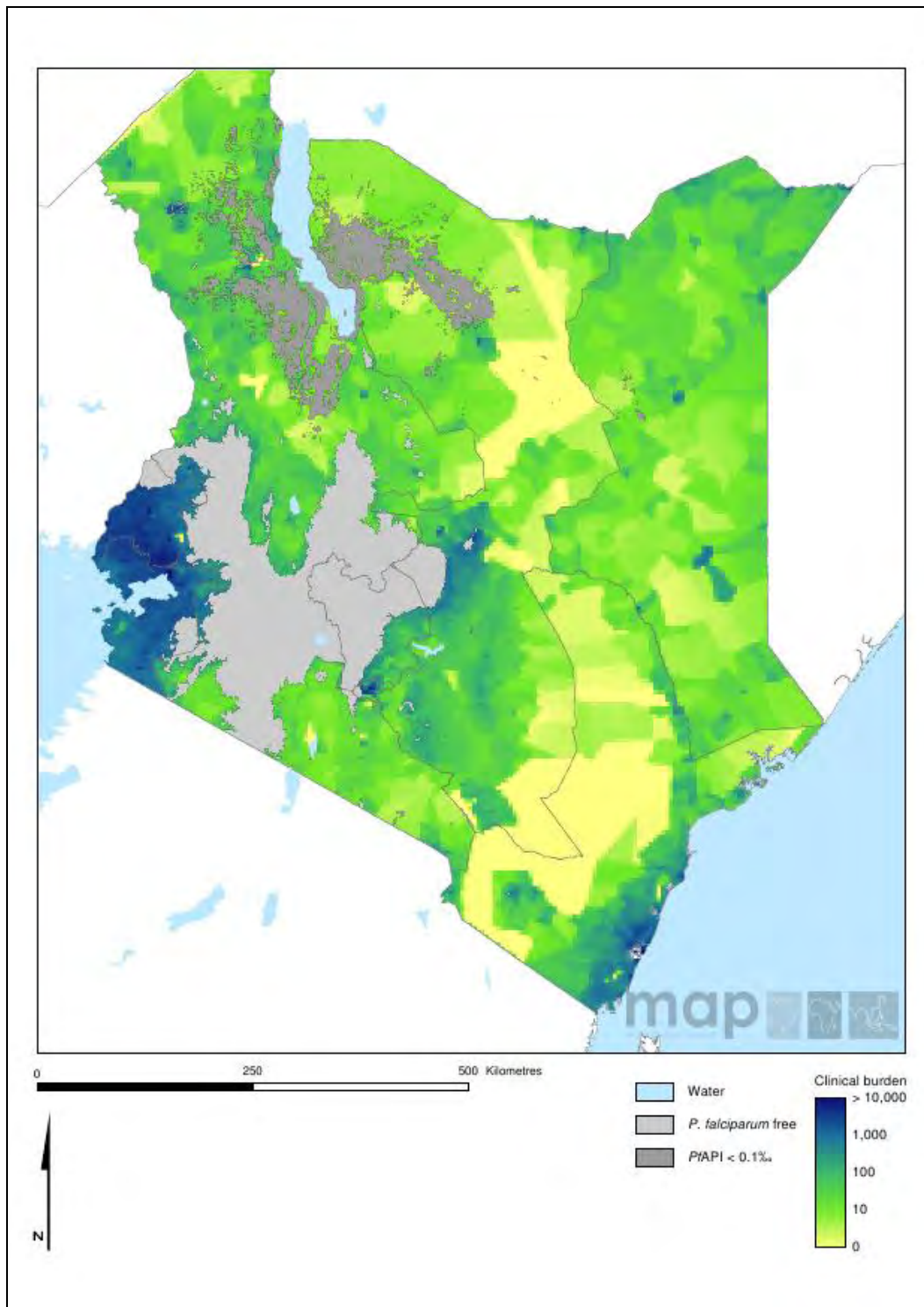


Figure 8: The spatial distribution of *Plasmodium falciparum* malaria endemicity in 2010 in Kenya
Source: Malaria Atlas Project, 2010.

d. Malaria prevalence in Tanzania

Malaria is endemic in most parts of Tanzania, and remains a major cause of morbidity and mortality both in rural and urban areas. Four plasmodia species, namely *Plasmodium*

falciparum, *P. vivax*, *P. malaria*, and *P. ovale* are prevalent in the country (Mboera *et al.* 2010). Over 93% of the Tanzania mainland population lives in areas where malaria is transmitted. The level of transmission is high in Lake Zone regions, coastal regions and southern lowlands (Chamwali 2013).

In Muheza district, the number of malaria cases increased between 1994 and 2002, with prevalence among children remaining consistently above 80% (Ishengoma *et al.* 2013). However, after 2002, the incidence of malaria begun to decline substantially in some parts of North Eastern Tanzania (USAID 2012) In the same district, the incidence of malaria in children fell rapidly during the early 2000s, reaching such a low level that a trial of intermittent preventive treatment in infants had to be stopped in 2005 as there were not enough cases. In the neighbouring Korogwe district, the prevalence of malaria parasitaemia among febrile patients fell substantially between 2003 and 2006 from 78% to 24% in lowland areas and from 25% to 7% in highland areas. Improved access to effective treatment through the community Programme probably contributed to the decline (Ishengoma *et al.* 2013). At least four of eight hospitals included in a review of admission data in Southwest Tanzania showed higher numbers of malaria admissions between 1995 and 2000 than in the preceding 10 years; entomological data from the Kilombero Valley in Tanzania, an area with one of the highest rates of transmission in the world, reported a 60–70% lower entomological inoculation rate than previously recorded (Ishengoma *et al.* 2013).

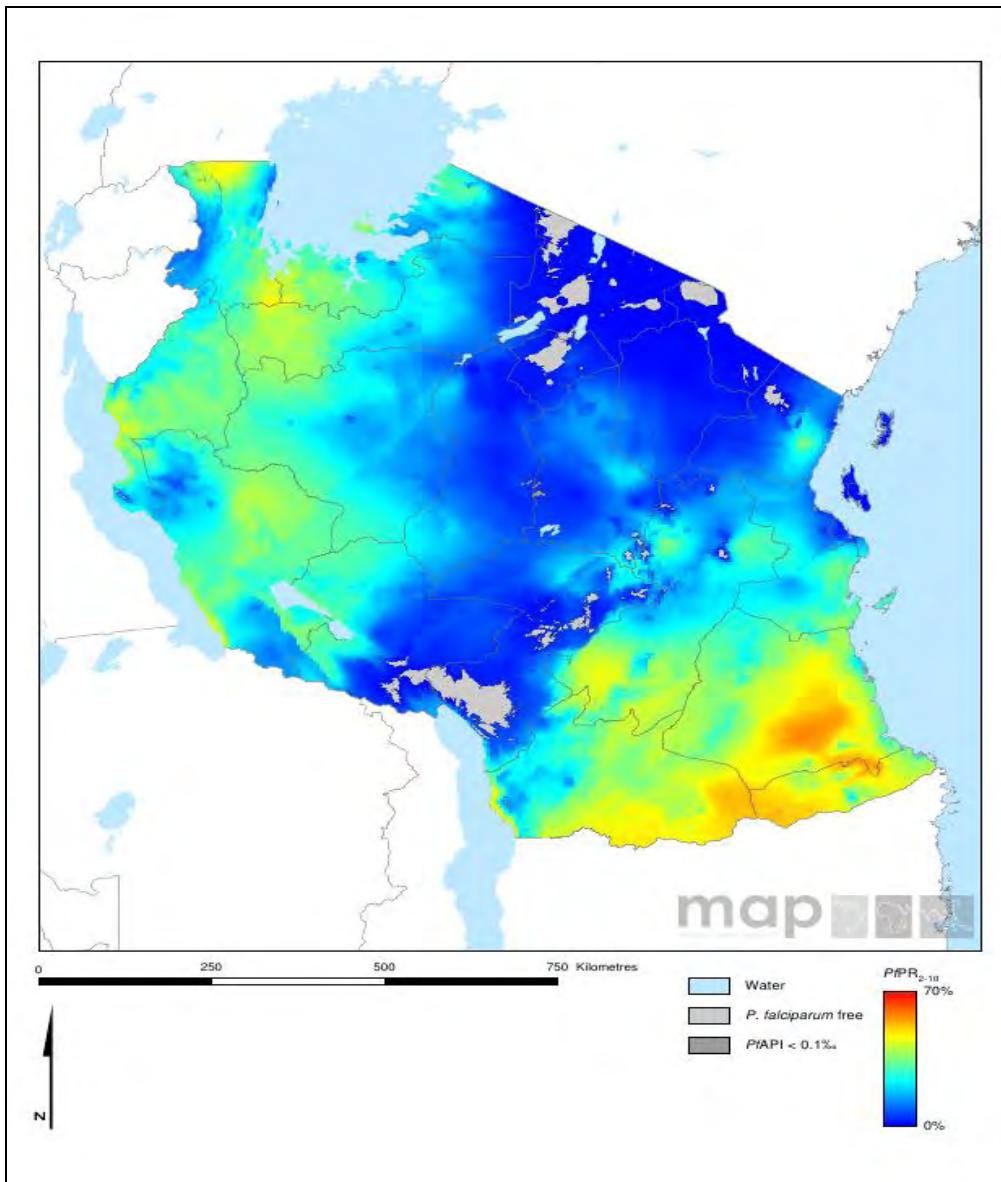


Figure 9: The spatial distribution of *Plasmodium falciparum* malaria endemicity in 2010 in Tanzania.
Source: Malaria Atlas Project, 2010

e. Malaria prevalence in Burundi

Malaria is a major public health issue in Burundi with high morbidity and mortality, as around 2.5 million clinical cases and more than 15,000 deaths are registered each year. It is still the single main cause of mortality in pregnant women and children below five years of age (Nkurunziza *et al.* 2011). In Burundi, three *Plasmodium* species are present and *Plasmodium falciparum* is the most dangerous species because it is responsible for severe and fatal cases accounting for 90 % of infections encountered in Burundi. The other two species (*Pmalariae* and *ovale*) represent only 8% and 2 % respectively. Epidemiological data show that all of Burundi's population is at risk of contracting malaria, although at varying degrees. The epidemiological stratification of 1998 had identified 8 of the 17 provinces as being at risk of an epidemic in the country: Gitega, Karusi, Kayanza, Muramvya, Muyinga, Mwaro, Ngozi

and Cankuzo. Based on data from the General Census of Population and Housing of 2008 (République du Burundi 2008), these provinces account for 48% of the population in Burundi. Statistical Yearbook data from the 2011 National Health Information System (SNIS) show that, compared with 2010, there has been 22 % reduction in terms of morbidity and 17% for mortality in hospitals (République du Burundi 2013).

With the highest rates of malaria in the region, the social and economic toll of malaria on Burundi's primarily rural, subsistence-farming society is enormous. Cankuzo province is among the most hit by malaria (Groen & Jacobs 2012). As a rural province with high levels of poverty and poor shelter conditions, vulnerability to malarial mosquitoes remains elevated. The lack of education and high illiteracy rates inhibit knowledge of prevention, recognition and proper case management (Kajangwa *et al.* 2013).

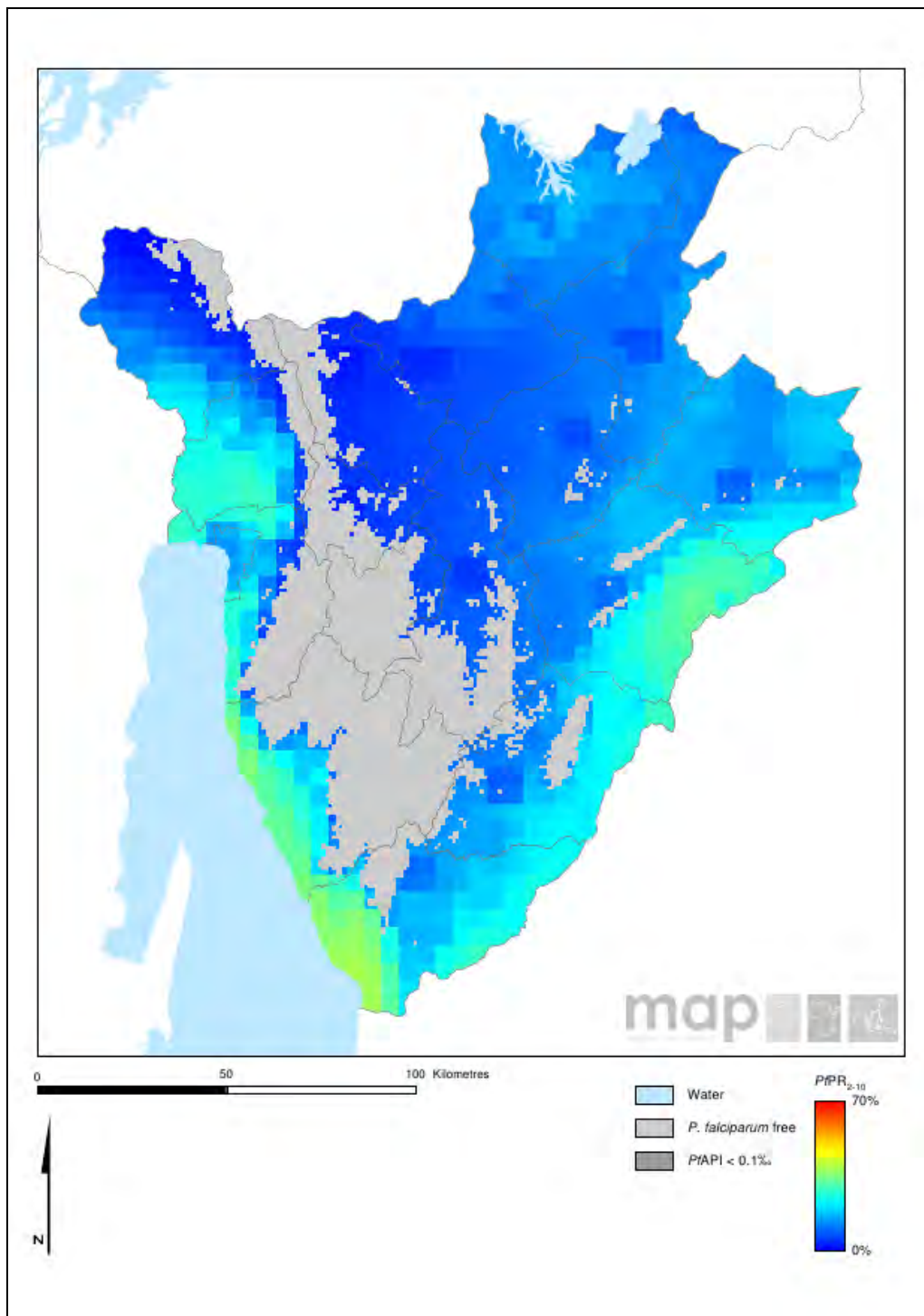


Figure 10: The spatial distribution of *Plasmodium falciparum* malaria endemicity in 2010 in Burundi
 Source: Malaria Atlas Project, 2010.

1.3. Schistosomiasis

1.3.1. Regional level

Schistosomiasis is a water-based disease which is considered the second most important parasitic infection after malaria in terms of public health and economic impact. The infected snails produce other larvae called “cercariae,” which infect humans by entering the body through the skin during water contact (Boelee & Madsen 2006). Both the schistosome parasite

and its intermediate snail hosts are very sensitive to water temperature (McCreesh & Booth 2014). Increasing temperatures in fresh water bodies in sub-tropical and tropical areas may therefore alter the geographic distribution of schistosomiasis. There is some empirical evidence that this may be occurring already in Uganda, with transmission occurring at altitudes previously considered too cold (Kabatereine *et al.* 2004; Rubaihayo *et al.* 2008).

Projections of Climate illustrate that there will be increasing temperatures across Africa (Stocker *et al.* 2013), including areas where the majority of people infected with schistosome parasites are located (World Health Organization 2012). It remains unclear how this phenomenon might affect the transmission potential of schistosomiasis in different locations, as the relationship between water temperature and schistosomiasis is non linear (McCreesh & Booth 2014). The results of the model developed in the framework of HF indicates the comparisons of model output at baseline with prevalence data showing that suitable temperatures are necessary but not sufficient for both schistosome transmission, and for high prevalence. Eight model scenarios were simulated, making a range of different assumptions about the relationship between air temperature and water temperature, and about snail mortality rates. For each scenario, the model was run using three sets of climate projections for eastern Africa over the next 50 years, based on three Representative Concentration Pathway (RCP) scenarios - RCP2.6, RCP4.5 and RCP8.5 (which represent low, moderate and high levels of warming respectively). In this study, maps of eastern Africa were produced highlighting areas where temperatures may become suitable for increased or decreased transmission, and where schistosomiasis may spread to new areas.

Figure 11 gives an indication of the level of agreement between scenarios in the overall direction of change (increased risk or decreased risk), and the number of scenarios that disagree with the overall direction. There is widespread agreement between scenarios and climate projections that infection risk may increase in Rwanda, Burundi, and eastern Zambia and over most of Uganda, Tanzania and south-west Kenya over the next 20 years, and that infection risk may decrease in north-east Kenya. A similar picture is found in 50 years' time, with the exception of the high warming scenario where risk is predicted to decrease over larger areas, and where there is disagreement between scenarios in the direction of change in risk over larger areas. In the majority of areas, the median predicted increase in infection risk is less than 20%. In parts of Rwanda, Burundi, south-west Kenya and eastern Zambia however, the median increase in risk is higher.

There is widespread agreement between scenarios that infection risk may decrease by more than 50% over the next 20 and 50 years in parts of north and east Kenya, southern South Sudan, and eastern Democratic Republic of Congo. The size of the area over which reductions may occur is larger with higher levels of warming, and in 50 years' time.

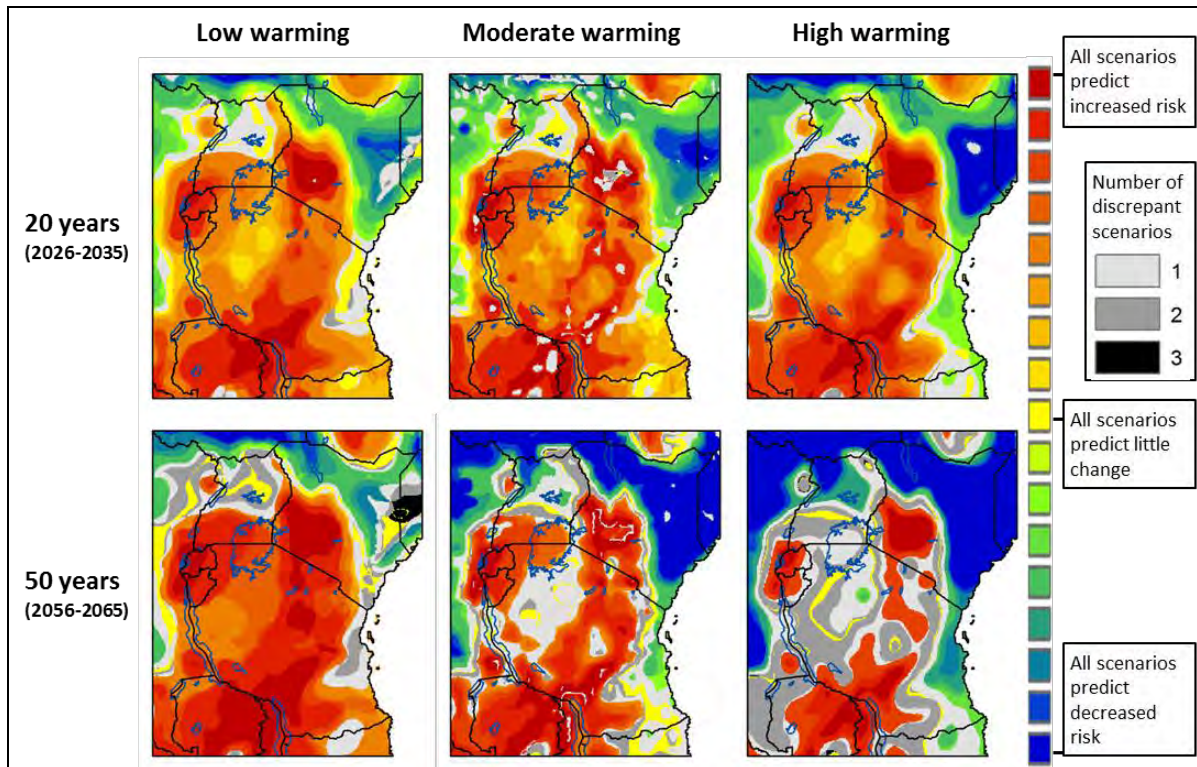


Figure 11: Change in *S. mansoni* risk in eastern Africa over the next 20 and 50 years.
Source: Leedale, 2014

Results are for 2026-35 compared to 2006-2025 (top) and 2056-2065 compared to 2006-2015 (bottom). Areas are shown in yellow if all scenarios agree that increasing temperatures will have little effect on schistosomiasis transmission. Areas are shown in red and blue respectively if there is widespread agreement between scenarios that temperatures will become suitable for increased or decreased schistosomiasis transmission over the next 20 years. Areas are shown in grey if the majority of scenarios predict increasing risk or little change, but one or more scenarios predict decreasing risk, or *vice versa*.

Figure 11 highlights areas at risk of new transmission foci developing. The left-hand maps show areas where the model predicts that cut-offs corresponding to 1-33% of maximum risk will be crossed over the next 20 and 50 years. These cut-offs correspond to temperatures

which are suitable for transmission, but not ideal. These cut-offs are therefore most likely to be crossed in areas where both levels of human risk behaviour are high, and where highly suitable snail habitats are found (for instance permanent habitats with a good supply of food and few predators). The right-hand maps show areas where the model predicts that cut-offs corresponding to 67-99% of maximum risk will be crossed. These cut-offs correspond to temperatures which are highly suitable for transmission. Schistosome transmission may therefore newly occur in areas and at potential transmission sites where levels of human risk behaviour are lower and/or snail habitats are more marginal.

1.3.2. Country level

a. Schistosomiasis prevalence in Kenya

The prevalence of schistosomiasis infection in Kenya is confined to fresh water lakes and rivers and inexistent in salt water. It poses a major public health concern in areas affected by widescale irrigation such as rice-growing schemes and dams following high level of water contamination. Clennon *et al* (2004) report that the coastal strip in Kenya is hyper endemic for urinary schistosomiasis, but with substantial spatial and temporal heterogeneities. The focal distribution of schistosomiasis is driven by local water use behaviour and the proximity of snail intermediate host breeding site (Clennon *et al.* 2006).

Factors influencing human infection patterns are varied including water contact patterns, immunity, the presence of competent intermediate snail hosts and the availability of suitable aquatic habitats for water use (Bayne & Loker 1987). Along the southern coast of Kenya, human exposure to *S. haematobium* takes place at snail habitats (ponds and streams) infested with the intermediate host snail *Bulinus nasutus*. In coastal Kenya effective targeting of control measures is complicated by the use of variety of different ponds for bathing, swimming and washing laundry by residents (Mutuku *et al.* 2011). It has been shown that the frequency and intensity with which people use a diversity of contaminated and uncontaminated water sources affects transmission patterns (Mutuku *et al.* 2011).

A study conducted in Msambweni area, Kwale District of Coast Province since 1984 on *Schistosoma haematobium* revealed that the prevalence has remained high (> 50%) over the years, despite introduction of alternative water sources (boreholes) and chemotherapy programs targeting school children (Chandiwana & Woolhouse 1991). The intermediate host in coastal Kenya is *Bulinus nasutus* snails that are commonly found breeding in rain-fed ponds. As most of the important transmission foci are rain-fed, rainfall is the key abiotic factor (Chandiwana & Woolhouse 1991).

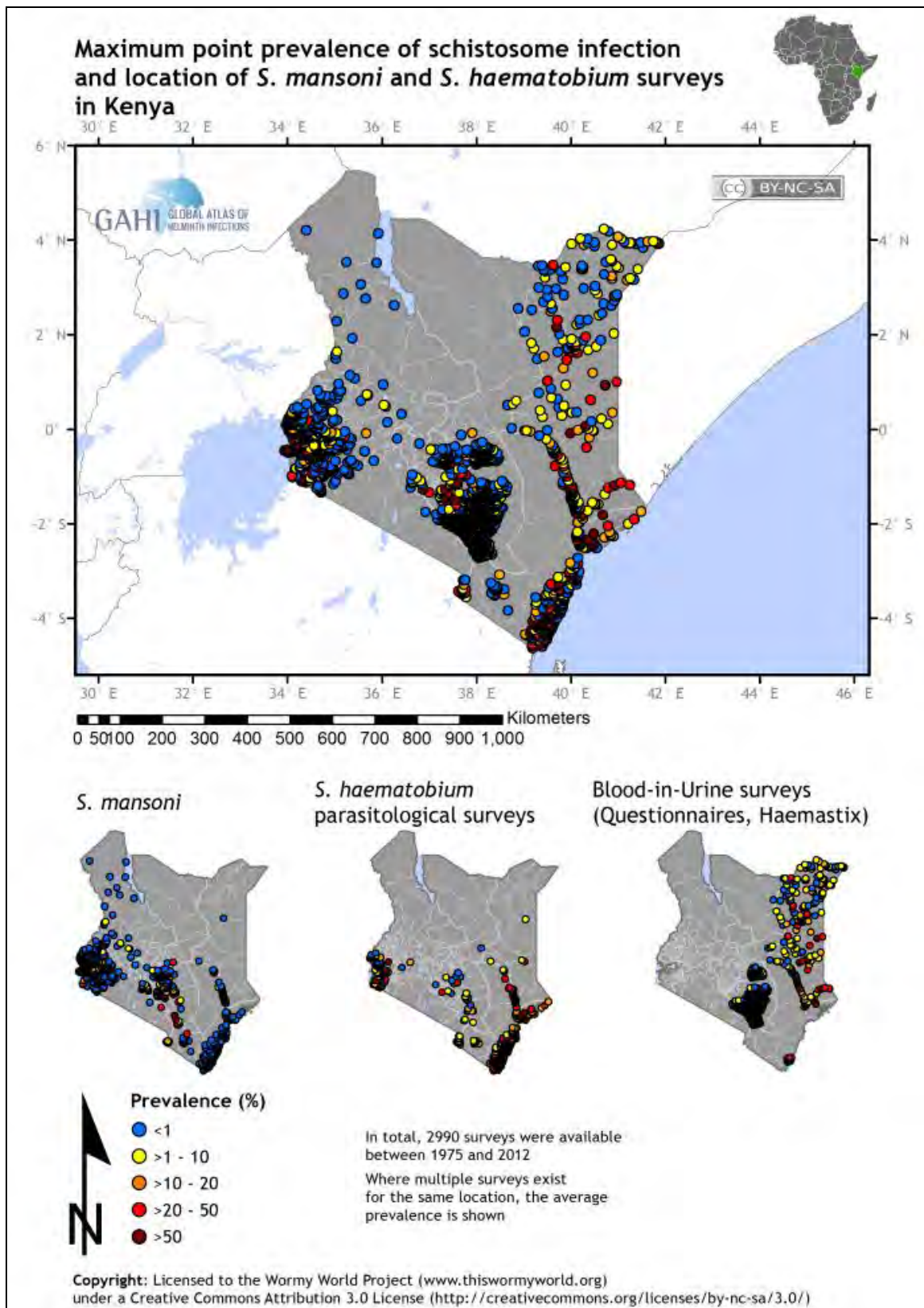


Figure 12: Schistosomiasis prevalence in Kenya

Source: <http://www.thiswormyworld.org/maps/2013/distribution-of-schistosomiasis-survey-data-in-kenya>

Figure 12 illustrates that the eastern part of Kenya is the most affected as well as the south and western area.

b. Schistosomiasis prevalence in Tanzania

In SSA, Tanzania occupies the second position (after Nigeria), in terms of highest burden of schistosoma (Mazigo *et al.* 2012). Both *Schistosoma haematobium* and *Schistosoma mansoni* have been endemic for a long time in Tanzania according to historical studies (Kinoti 1964; Doumenge *et al.* 1987; Mwambungu 1988; Malenganisho *et al.* 2008). As reported by Doumenge *et al.* (1987), the first scientific report of schistosomiasis in Tanzania was traced in 1895 when Manson Bahr published the first recorded case of intestinal bilharziasis. The early studies in the Lake Victoria province by Cook in 1905 at Kwimba identified and described the distribution of *S. mansoni* and *S. haematobium* in the region (Doumenge *et al.* 1987). It was found that over 50 % of individuals examined had urogenital schistomiasis while *S. mansoni* was noted to be widespread on the southern and eastern shores of Lake Victora (Cook 1909). Large and small scale epidemiological surveys conducted since 1920s to date indicated the distribution, intermediate hosts, prevalence and intensity of both urogenital and intestinal schistosomiasis (Maclean *et al.* 1958; Magendantz 1972). The review of the distribution of *S. mansoni* and *S. haematobium* by McCullough (1972) and Doumenge *et al.* (1987) revealed the main extensive zones were found in the south eastern and south western sides of Lake Victoria and its island. *Schistosoma haematobium* was widely distributed and two extensive zones were noted to have high transmission, namely the inland on the eastern and south-eastern hinterland of Lake Victoria and low land zones on the eastern coast of the country (McCullough 1972). Regarding Zanzibar islands, the distribution of *S. haematobium* was restricted to the north western and central areas of Unguja Island while Pemba Island was endemic for *S. Haematobium* on the western, southern central and northwest of the island. The regions that are highly endemic to *S. mansoni* cover the north-west surrounding lake Victoria, the northern, central, southern and south east of the country. On the other hand, the hinterland areas of the country were identified to be highly endemic for *S. haematobium* (Webbe 1959; Magendantz 1972).

Today, both urogenital and intestinal schistosomiasis remains a major public health problem in Tanzania. Both categories are endemic at varying levels of transmission in different administrative regions. *Schistosoma haematobium* is widely distributed (Doumenge *et al.* 1987; Jordan *et al.* 1993).

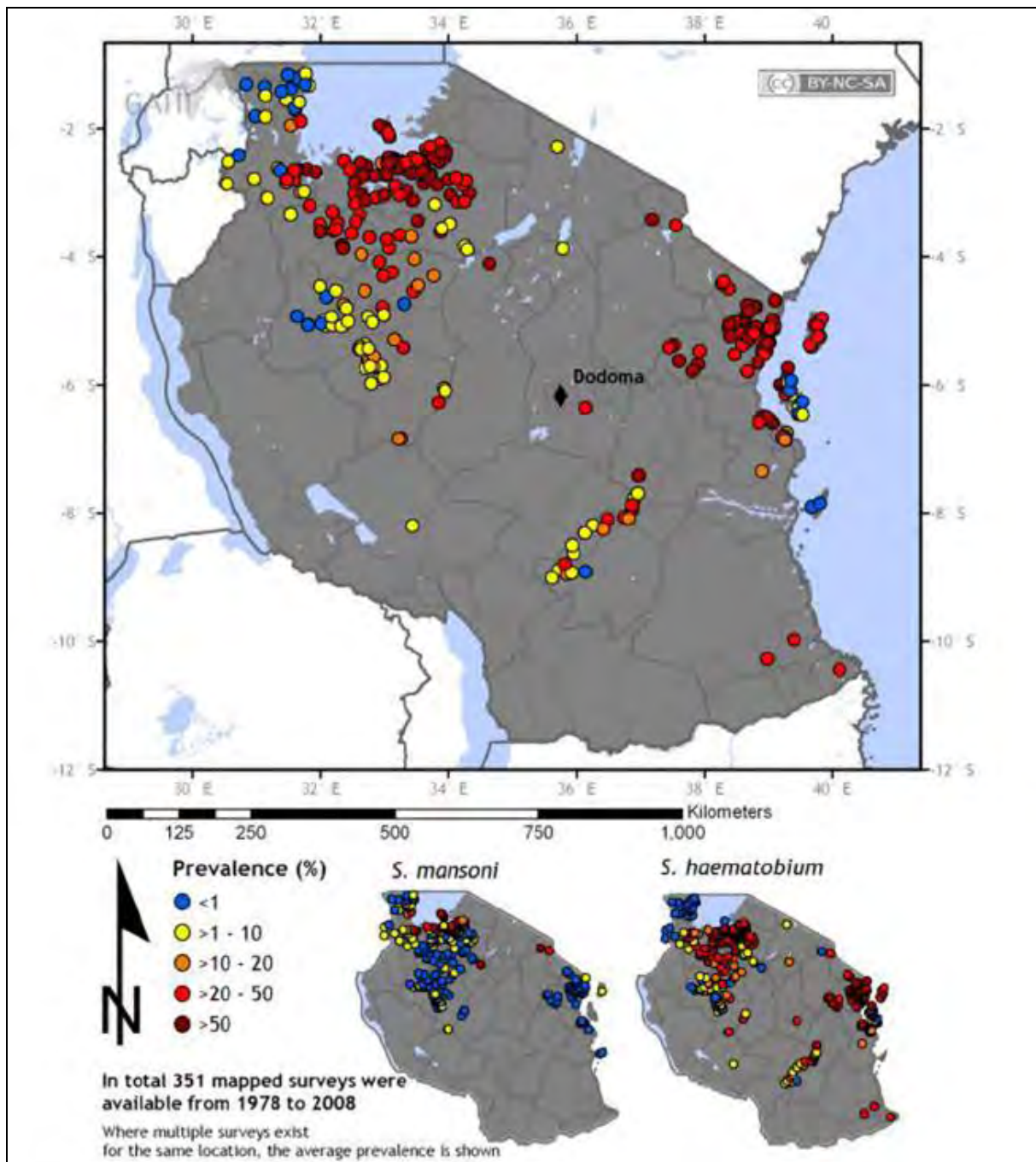


Figure 13: Distribution of schistosomiasis (both *S. mansoni* and *S. haematobium*) in Tanzania
 Source: Mazigo et al, 2012

Figure 13, illustrates prevalence of schistosomes infection and location of *S. mansoni* and *S. haematobium* surveys in the United Republic of Tanzania. The observed geographical distribution indicated that *S. haematobium* is highly endemic along the eastern and south-eastern coasts, the islands of Unguja and Pemba (Zanzibar) and the interland areas of the north-western zones of the country (Brooker & Clements 2009). These have been identified as potential areas for the intermediate-host snail species responsible for transmission of *S. haematobium* (Brooker & Clements 2009). *Schistosoma mansoni* is absent on the coastal

area due to the absence of its intermediate host snails and thermal exclusion (Brooker *et al.* 2009) but is dominant along the shores and islands of Lake Victoria.

c. Schistosomiasis prevalence in Uganda

In Uganda, schistosomiasis is mainly caused by *Schistosoma mansoni* and affects more than 10% of the population (Tukahebwa *et al.* 2013). A study was conducted in Musoli village along the shore of Lake Victoria, Mayuge District in Southeast Uganda in a district located at an altitude of 1161 m above sea level, with temperatures ranging from 19–27 °C and receiving annual rainfall in the range of 600– 1100 mm. Like in other areas surrounding lakes in Uganda, the transmission of schistosomiasis around Lake Victoria is stable and intense throughout the year. The findings indicated that Schistosomiasis is highly prevalent in (88.6%) of the population with high intensity of infection in this Victoria Lake shore community and infection is highly related to water contact. The high infection level is typical for endemic areas around Lake Victoria (Kardoff R *et al.* 1997). Intensity of infection was higher in males than females and this could be due to occupational exposure such as fishing, which prolongs the duration of contact with schistosome-infested water (Tukahebwa *et al.* 2013). The peak *S. mansoni* infection intensity in the shore of lake Victoria, Mayuge District in Southeast Uganda occurred in the 15–19 year age group (Malenganisho *et al.* 2008).

However, exposure alone may not explain this age difference in infection. A study on a fishing community along Lake Albert, where adults were more exposed to infested water than children recorded a similar age infection pattern (Tukahebwa *et al.* 2013). This pattern could be explained by slow development of acquired immunity to schistosomiasis infection. In endemic areas, people acquire immunity in response to parasite antigens and this immunity is influenced by age or duration of exposure (Tukahebwa *et al.* 2013). Another explanation for infection peaking in age group of 15-19 years could be due to physiological changes at puberty. Hormonal changes during puberty, such as increase in skin thickness or deposition of fat, is reported to increase resistance to *S. mansoni* infection by reducing cercarial penetration (Gryseels 1994).

Concerning the spatial distribution of schistosomiasis in the whole country, data aggregated at the school or community level were analysed to investigate the geographical distribution of *S. mansoni* (Kabaterine *et al.* 2004). Figure 14 highlights an uneven distribution of infection prevalence. The prevalence appears to be highest close to the shores of Lake Albert, the Albert Nile, Lake Kyoga and the eastern shores of Lake Victoria. In the northern areas of the

country, prevalence were lower (<50%), while prevalence was generally <20% in the south-west of the country away from Lake Victoria, and >50% close to Lake Victoria. Areas of zero or low prevalence are found in the north-east of the country. Prevalence is also low in much of eastern Uganda; but paddy rice cultivation here has become popular in recent years and is associated with the emergence of *S. mansoni* (Bukonya *et al.* 1994). For Lira District, north of Lake Kyoga, because of civil unrest these data have not been included in the analyses, although survey results indicate prevalences of 15–75% (Vector Control Division, Unpublished data). In the capital, Kampala, previous surveys report a prevalence of 4% (Kabaterine *et al.* 1996).

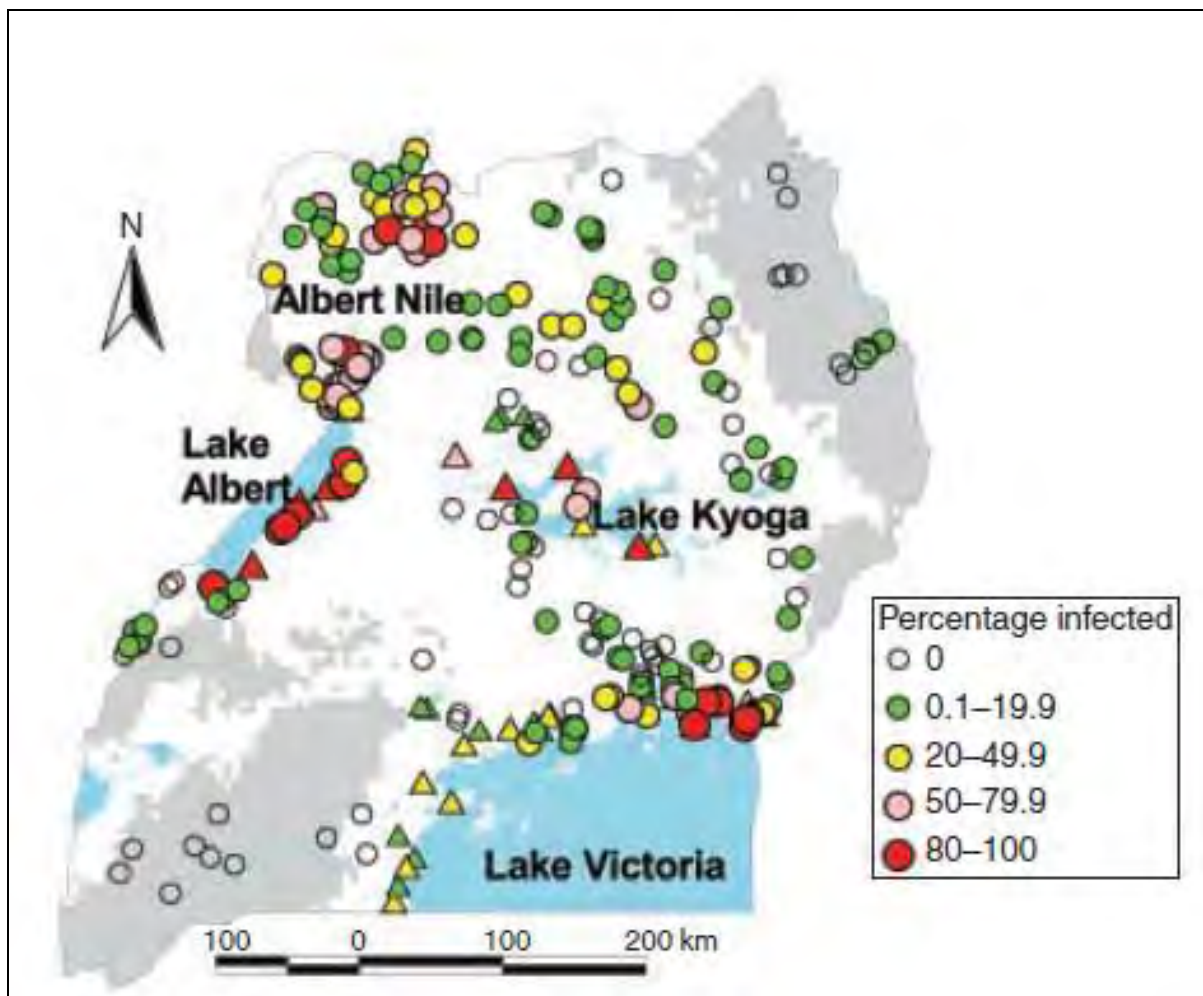


Figure 14: Distribution of *Schistosomamansoni* in Uganda.
Source: Kabaterine *et al.* 2004

In Figure 14, the circles indicate school survey prevalences and triangles represent community survey prevalences. Grey areas indicate areas where either altitude exceeds 1325 m or total annual rainfall is < 900 mm. Water bodies were defined on the basis of Landsat Thematic Mapper (ETM+).

d. Schistosomiasis prevalence in Burundi

In Burundi intestinal schistosomiasis caused by *Schistosoma mansoni* seems to be essentially an anthropogenic problem: land reclamation, agricultural development, and human resettlement have largely contributed to its spread since the 1950s. According to Engels *et al.* (1993), four distinct endemic areas can be considered: the Rusizi Plain, the suburban focus of Bujumbura, Imbo-Sud bordering Lake Tanganyika south of the capital and the Bugesera depression, where schistosomiasis is mainly concentrated around Lake Cohoha and Lake Rwihinda where the combined population at risk is estimated to be 400,000 people (Engels *et al.* 1993). The most recently published data from the Burundian side of Lake Rweru indicates this area as a highly endemic focus for *Schistosoma mansoni* infection.

Although little information is known about schistosomiasis evolution in Burundi over the past few years, early studies have shown that the prevalence and intensities of infection as well as the number of symptomatic cases detected in general health services have decreased considerably. In areas with good access to basic health services, most schistosomiasis cases have received treatment. Yearly selective chemotherapy in primary schools in suburban Bujumbura reduced the prevalence of schistosoma infection among pupils from 23% to 9% over the period 1984-90, and this programme has now been extended to highly endemic areas in Imbo-Sud. Focal snail control produced disappointing results, and emphasis has therefore shifted towards health education and environmental control of transmission (Engels *et al.* 1993).

According to Ndayishimiye *et al.* (2014) schistosomiasis prevalence and intensities have been observed at the subregional, local and even sublocal level where children and adolescents appear to be to most affected category, with relatively high rates of infections recorded in adults in many areas. According to the same author, in children as well as in adults, schistosomiasis-related morbidity such as (bloody) diarrhoea, hepatomealy and splenomegaly was apparent mainly in areas with prevalences over 30-40%. The intermediate hosts were *Biomphalaria pfeifferi* (Imbo), *B. sudanica* (Tanganyika marshes) and *B. stanleyi* (Cohoha). Surveys in the eastern lowlands of Burundi, an area where rice cultivation is widely promoted, have shown that the progression of schistosomiasis in recent years has been slower than initially feared. However, further surveillance is necessary to fully eradicate the disease (Ndayishimiye *et al.* 2014).

The figure 15 (extracted from the Global Atlas of Helminth Infections) portrays the schistosomiasis prevalence in Burundi based on data obtained from 41 surveys conducted between years 1983 and 2007.

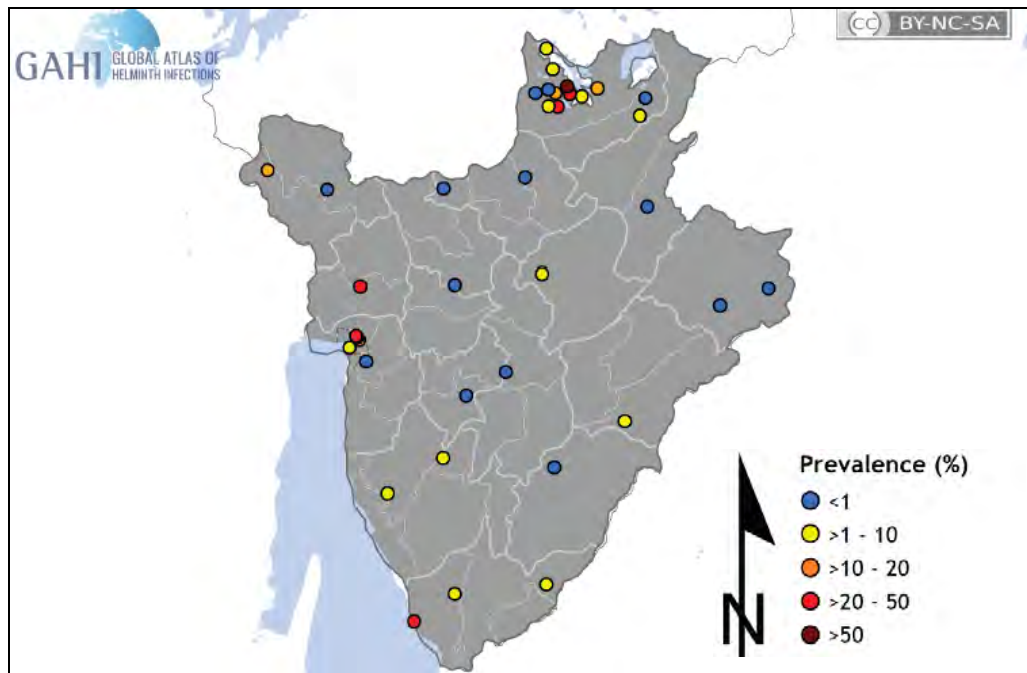


Figure 15: Maximum point prevalence of schistosome and location of *S. mansoni* and *S. haematobium* surveys in Burundi
 Source: Brooker *et al.*, 2009.

e. Schistosomiasis prevalence in Rwanda

In Rwanda Since 1975, intestinal schistosomiasis was locally endemic and was diagnosed in many areas (Isabwe *et al.* 2012). In 1980, a great number of infected children (5-10 years) were undoubtedly a sign of high potential of transmission in the country Infected snails of the genus *Biomphalaria* have been found around lakes mostly in May and June. The main transmission areas were found around Lakes Ruhondo, Burera, Kivu, Muhazi, Rweru, Mugesera and some swampy areas in Nyagatare district (Isabwe *et al.* 2012). Since 2008, the Rwanda Ministry of Health has conducted a de-worming campaign against schistosomiasis using praziquantel targeting school aged children and adults at high risk (Mupfasoni *et al.* 2009).

Schistosomiasis occurs in focal pockets and closely linked to the presence of water bodies that harbour susceptible species of snails (Meurs 2014). An investigation conducted on intestinal schistosomiasis around Lake Rweru in Rwanda has confirmed the presence of *Schistosomiasis mansoni* infection with a proportion of infected individuals of 21.1% which suggests high prevalence of the disease among the lake-shore inhabitants with great spatial

variation of infection (Ruberanziza *et al.* 2010). The highest proportion of infected individuals was observed among individuals living in close proximity to the lakes that are the only source of water for the community. Pre-school children harbour infection and are a source of transmission of schistosomiasis. In endemic Communities the infection of pre-school children early in life was due to exposures through bathing in the lake by their mothers, while the older children would visit the lake for washing, fetching of water, bathing and swimming. Further investigations are still needed in order to determine the true prevalence of this infection and to plan for disease control accordingly.

Figure 16 (extracted from the Global Atlas of Helminth Infections) portrays the schistosomiasis prevalence in Rwanda based on data obtained from 138 surveys conducted between years 1980 and 2008.

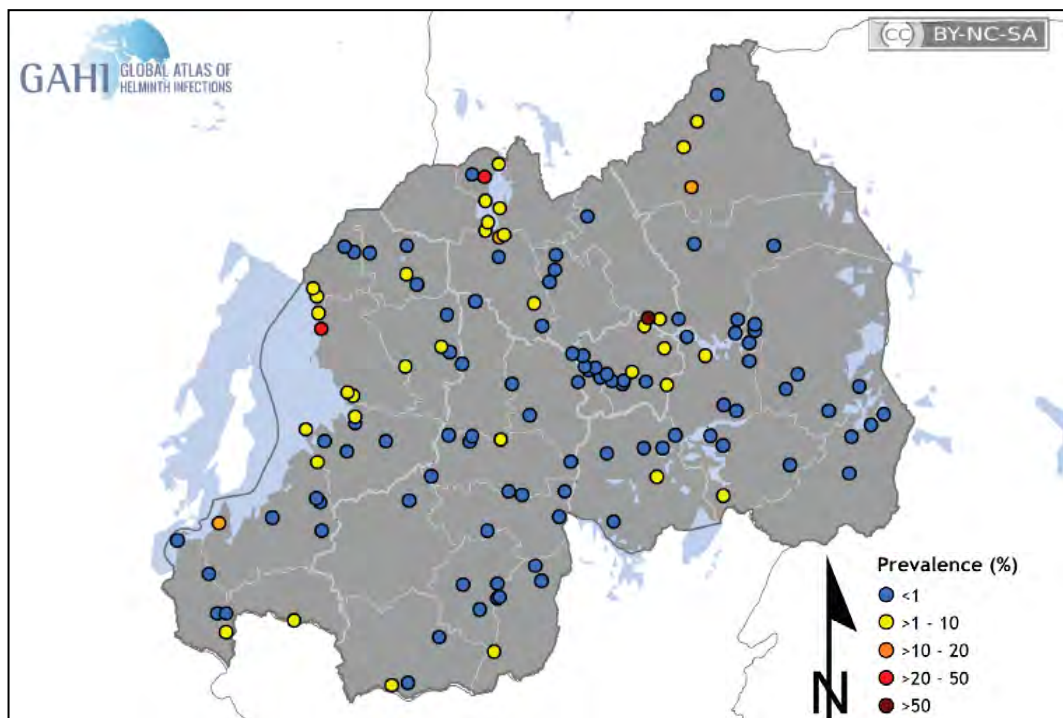


Figure 16: Maximum point prevalence of schistosome and location of *s. mansoni* and *s. haematobium* surveys in Rwanda

Source: Brooker, S., et al. 2009

Figure 16 illustrates that the schistosomiasis is more or less equally distributed apart from the North east where the prevalence is limited.

1.2. Rift Valley Fever

1.2.1. Regional level

Figure 17 illustrates how the peak year frequency in RVF incidence changes compared to historic period for both immature and mature livestock. There is significant increase in central/western Kenya & Rwanda, both considered as hotspots for vector abundance. In most of other areas there is decrease.

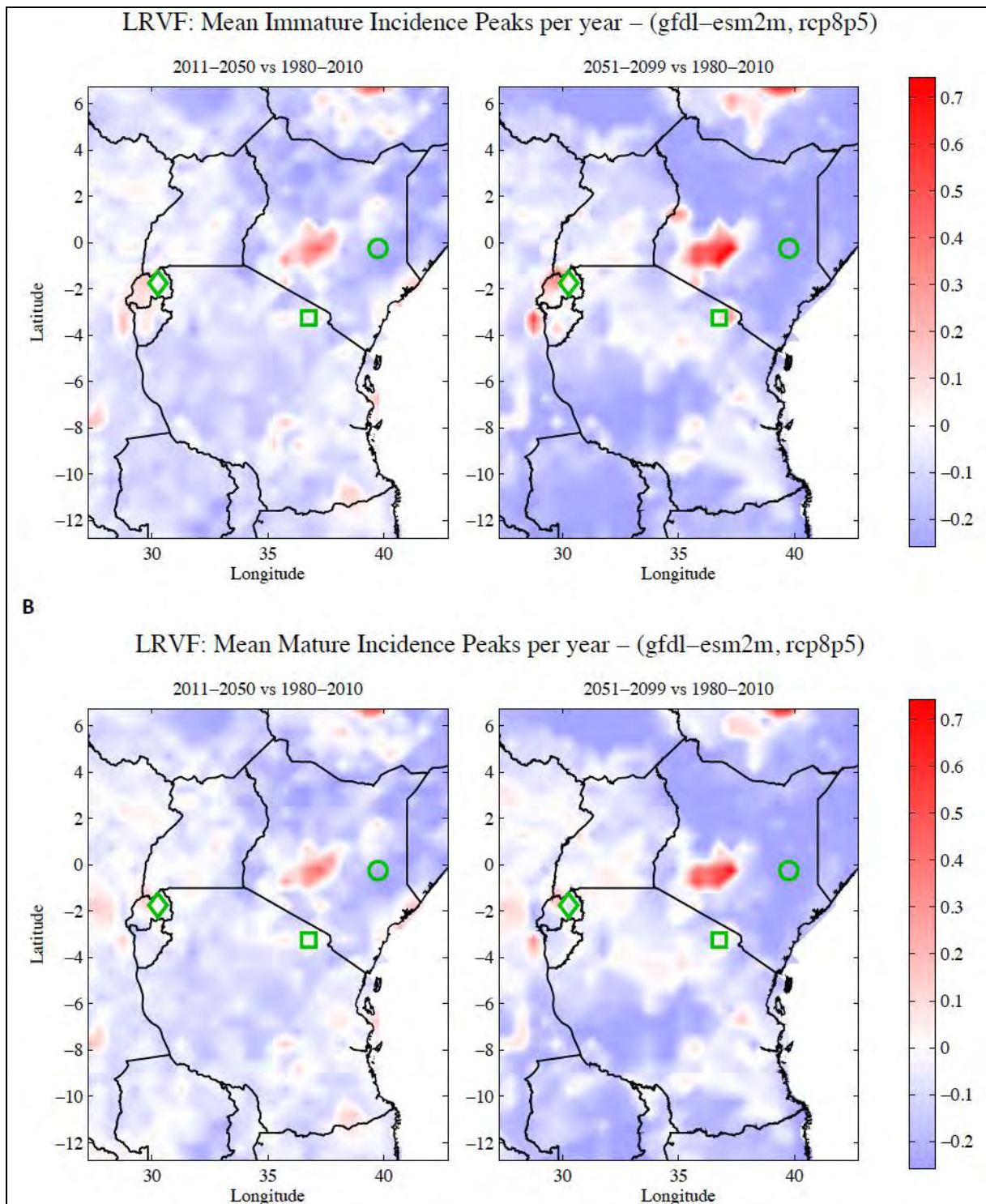


Figure 17: Difference in incidence peaks compared with the historical period for both immature (A) and mature (B) livestock for the early and late 21st Century using climate model gfdl-esm2m and emission scenario RCP 8.5 Source: Leedale, 2014.

1.2.2. Country level

RVF is a mosquito-borne viral zoonosis that mainly affects sheep, goats, cattle and camels. Humans become infected following a bite from an infected mosquito or after an intensive contact with acutely infected animals or infected tissues. In humans, the disease manifests

either as a mild influenza-like syndrome in a majority of cases (> 80 per cent) or as severe disease with haemorrhagic fever, encephalitis, or retinitis (Soumare *et al.* 2012). Its emergence and spread is influenced by climate change (Martin *et al.* 2008; Caminade *et al.* 2011) and land use patterns, specifically the development of dams (Martin *et al.* 2008). Livestock trade might also contribute to its transmission as genetic analyses of the viruses isolated in Saudi Arabia in the 2000-2001 outbreak was identical to the virus implicated in the 1997-1998 outbreak in eastern Africa (Shoemaker *et al.* 2002). The virus was first identified in Kenya in the 1930s (Daubney *et al.* 1931), and outbreaks have occurred in Kenya, Tanzania South Africa, Zambia and Zimbabwe following periods of exceptionally above normal rainfall (Anyamba *et al.* 2002). It recently spread to Madagascar, Saudi Arabia and Yemen.

RVF virus (RVFV) is transmitted by a wide diversity of arthropods; it has been isolated from more than 40 species of mosquitoes from eight genera (Meegan and Bailey 1988). At least six mosquito genera namely: *Aedes*, *Anopheles*, *Coquillettidia*, *Culex*, *Eretmapodites* and *Mansonia* have been proven to be capable of being infected with RVFV in the lab, or have been found to be infected in the wild (Chevalier *et al.* 2012). Based on initial studies done in eastern Africa by Linthicum *et al.* (1985) and Davies *et al.* (1985), RVFV vectors can be classified into primary and secondary vector species. Primary vectors (primarily *Aedes* spp) maintain the virus via trans-ovarial transmission through inter-epidemic periods and initiate transmission when infected adults emerge while secondary species (including *Culex* spp., etc) amplify RVFV transmission to epidemic proportions. To date, no alternative mechanism for RVFV maintenance in the environment between epidemics has been described.

Based on the observations made during the recent (1997-1998 and 2006-2007) RVF outbreaks published by Anyamba *et al.* (2009), it appears that Kenya and Tanzania are the only two countries in eastern Africa that continue to experience explosive outbreaks of the disease following periods of above-normal and persistent rainfall. The other countries (Uganda, Burundi and Rwanda) have not had RVF epidemics but it is believed that the virus is circulating in some permissive ecologies (Magona *et al.* 2013). Serological surveys that have been carried out in Uganda, for instance, demonstrate that up to 10% of goats have anti-RVF virus IgG in (Magona *et al.* 2013); this suggest that there is endemic transmission of the virus in a number of areas. Our review, however, focuses on epidemics that have been reported in Kenya and Tanzania. The detection of the RVF during inter-epidemic periods is difficult

(Lichoti *et al.* 2014) and the data that have been reported for these periods are based on active sero-epidemiological surveys.

a. Rift Valley fever in Kenya

In Kenya, RVF outbreaks have previously occurred in 1931, 1951/53, 1961/63, 1967/68, 1977/79, and most recently during 1997/98 and 2006/07 with unusually high human morbidity and mortality (Anyamba *et al.* 2009). The recent outbreak (2006/2007) had an unusually high human morbidity and mortality and was associated with considerable socio-economic impacts and disruption of livelihoods (Rich & Wanyoike 2010). Most of these impacts were felt in the north-eastern and central parts of Kenya which currently have the highest levels of hazards (Figure 18). In this figure, hazard is measured as the probability of an outbreak occurring and it has been estimated using an ecological niche model implemented in R using the Random Forest algorithm.

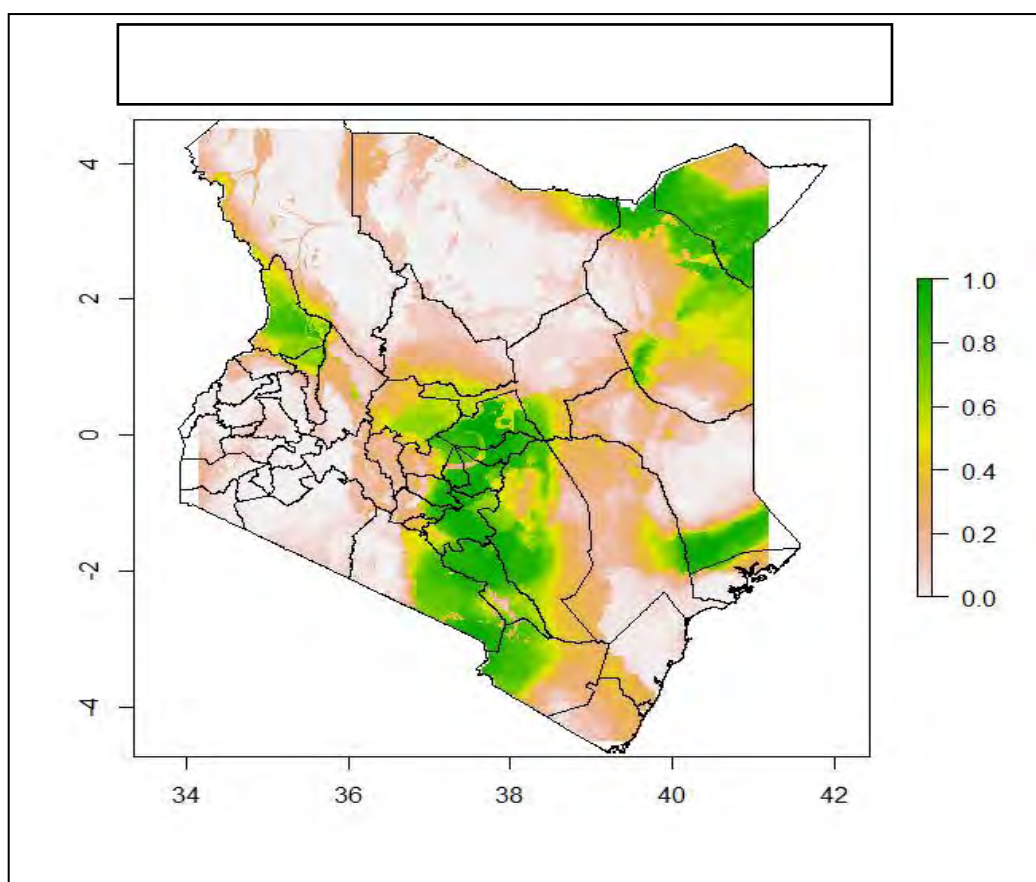


Figure 18: The current RVF hazard map of Kenya

Source: Unpublished and generated from ecological niche modelling conducted at ILRI

Bans on livestock movement and trade that were instituted following the 2006/2007 outbreak had substantial impacts on the country's economy. Rich and Wanyoike (2010) estimated the total losses as being in excess of USD \$ 32 million. Producers experienced livestock mortality losses, which in turn affected food security and incomes. The total economic losses from livestock mortality in the north-eastern region alone were estimated to be more than Ksh 610 million (over US\$9.3 million at an exchange rate of US\$1 = Ksh 65) (Rich & Wanyoike 2010). Livestock traders were also affected through losses from unsold animals that died under their possession, maintenance of unsold animals during the quarantine period or due to closure of slaughter houses or, generally, due to reduced number of animals slaughtered during the period. The traders who engaged in sale of livestock products such as meat, particularly the butchers, also faced negative impacts due to closure or reduced supply of their sale products (Rich & Wanyoike 2010).

b. Rift Valley fever in Tanzania

Tanzania has had a number of RVF outbreaks since RVF-like disease syndrome was reported in the 1930s. Successive outbreaks occurred in 1947, 1957, 1960, 1963, 1968, 1977/78, 1989, 1997/98, and 2006/2007 (Sindato *et al.* 2014). The estimated inter-epidemic interval is 7.9 with a range of 3 – 17 years. This compares favourably with the mean inter-epidemic period in Kenya estimated to be 5.4 years with a 95% confidence interval of 4.4 – 6.4 years (unpublished data). Between 1930 and 1979, RVF outbreaks were restricted to four districts in northern Tanzania –including Ngorongoro, Simanjiro, Monduli and Hai (Sindato *et al.* 2014). From the 1980s onwards, the spatial extent of the risk zone expanded progressively to east, central and southern parts of Tanzania. In the 2006-2007 outbreak, areas that had initial outbreaks before confirmatory diagnoses were carried out included Manyara, Kilimanjaro, Tanga, Dodoma, Iringa and Morogoro but by the time the outbreak ended in July 2007, the disease had affected 25 out of 126 districts in the country (Swai & Schoonman 2009). Areas with the greatest RVF hazard currently are illustrated in Figure 19; those represented in pink denote areas that reported the outbreak in 2007 while the sites in red represent areas predicted to have a hazard of at least 50% or higher based on a logistic regression model that used soil types, altitude, precipitation and land use as predictors. The outcome is the probability of an RVF outbreak.

There are scanty records on the impacts of past outbreaks; only the recent outbreak in 2006/2007 has reliable data on the numbers of livestock affected, estimated at 46,680 cattle, 56,990 goats, and 32,900 sheep. Of these, 15,726 cattle, 19,199 goats and 12,124 sheep aborted and 16,973 cattle, 20,913 goats and 12,124 sheep died from the disease (Sindato *et al.* 2011). The estimated cost of mortality was USD 4,243,250 and USD 2,202,467 for cattle and sheep and goats (combined), respectively (Sindato *et al.* 2011). Income of the livestock dependent communities dwindled as a result of reduction in the consumption of red meat (Sindato *et al.* 2011). These impacts further eroded rural people's food security and household nutrition. Actions taken to combat the epidemic (including restriction of animal movements and ban of the slaughter of cattle) also caused additional negative effects on peoples' livelihoods. A ban on the importation of livestock specifically by the countries in the Middle East led to a 54 per cent decline in exports, equivalent to loss of USD 352,750. This caused serious economic losses to populations who were totally dependent on this income. Internal market flows dropped by 37 per cent (Sindato *et al.* 2011).

The outbreak also caused psycho-social distress particularly for those households that lost family members. A total of 144 people out of 309 affected died from the disease – this represents a case fatality rate of 46.6 per cent (Sindato *et al.* 2011). Those who went through treatment were hospitalised within a period of 5 days from the onset of illness and they remained ill with RVF-associated symptoms for an average of 28 days (range of 2 to 120 days).

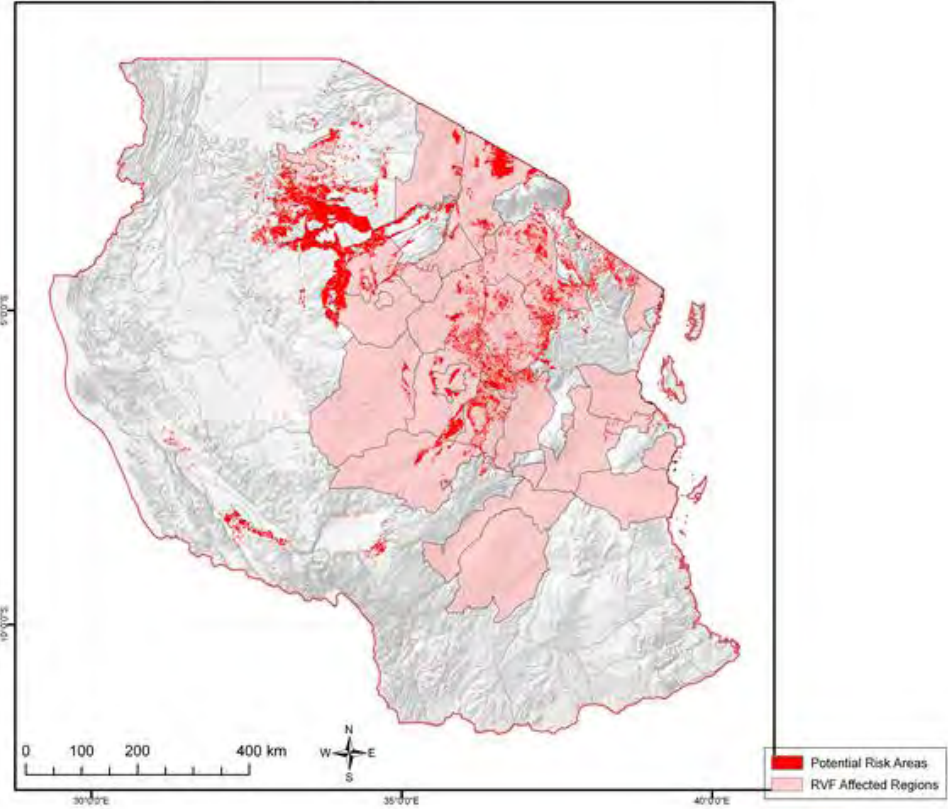


Figure 19: Regions with the greatest RVF hazard in Tanzania (unpublished data)

2. Integrated risk assessment: present day and future risk to malaria, schistosomiasis and Rift Valley fever

This section presents the results of the spatial risk assessment that was carried out for the three target VBDs. For each disease, the analysis was carried out for the present day as well as for the future based on two different emission scenarios: RCP4.5 and RCP 8.5. The following sections present the indicators and the risk, hazard and vulnerability maps for each of the three target diseases.

2.1. Malaria

As indicated earlier, the spatial modelling of malaria risk relies on a set of previously defined indicators that served as an input for the analysis. Table 1 provides an overview of the hazard (i.e. disease-related) and vulnerability indicators that were integrated into the assessment. The risk assessment results for malaria as shown in Figure 20 for the present days point out a significant hotspot area stretching from south-eastern Tanzania along the coast of east Kenya towards northern Kenya. Additional hotspots characterise north-western Uganda and can be found along Lake Tanganyika. The areas of central Tanzania, the east African highlands as well as Rwanda and Burundi are characterised by low risk values ('cold spots').

Table 1: Malaria risk factors (including vulnerability indicators) for eastern Africa

Indicator name	Date	Resolution ^b	Sign ^c	Weight	Data source
Hazard					
Entomological inoculation rate (EIR)	2010	~50km	+	0.5	D3.4 (Leedal 2014)
Vulnerability					
<i>Generic susceptibility (SUS)</i>					
Number of women	2010	1 km	+	0.0272	AfriPop:demography
Population change	1970-2010	2.5-arc minutes	+	0.0314	GPWv3, UNEP-APD
Travel time to closest urban center	2000	30 arc-seconds	+	0.0229	JRC/WorldBank
Distance to roads	2010	Line layer	+	0.0286	OSM, ESA GlobCover, SRTMv4
Conflict density (km ²)	1997-2009	Point layer	+	0.0429	ACLED
Number of people living on less than 2 USD per day	2010	2.5 arc-minutes	+	0.1214	CGIAR CSI
<i>Lack of capacity to anticipate (C2A)</i>					
Secondary/higher education (%)	2007/08	Point layer	-	0.0571	DHS
Child did not sleep under net last night (%)	2007/08	Point layer	+	0.2100	DHS
<i>Biological susceptibility (BIO)</i>					
Number of children under the age of 5 ^d	2010	1 km	+	N/A	AfriPop:demography
Number of women of childbearing age	2010	1 km	+	0.0414	AfriPop:demography
Prevalence of stunting children under the age of 5	2010	5 arc-minutes	+	0.0843	FAO
Immunity	2010	1 km	-	0.1614	Malaria Atlas Project
HIV prevalence among 15-49 year olds (%)	2010	Polygon layer	+	0.0857	USAID
<i>Lack of capacity to cope (C2C)</i>					
Distance to closest hospital	2010	Point layer	+	0.0671	OSM, ESA GlobCover, SRTMv4
Number of dependents	2010	1 km	+	0.0186	AfriPop:demography

^aBased on the outcomes of the literature survey, expert consultation and data availability; ^bRefers to the spatial resolution of the original datasets (i.e., before the data was resampled to 10 x 10 km² grids); ^cSign indicates if high indicator values increase (+) or decrease (-) risk; ^dThis indicator was removed from the analysis to reduce existing multicollinearities in the data.

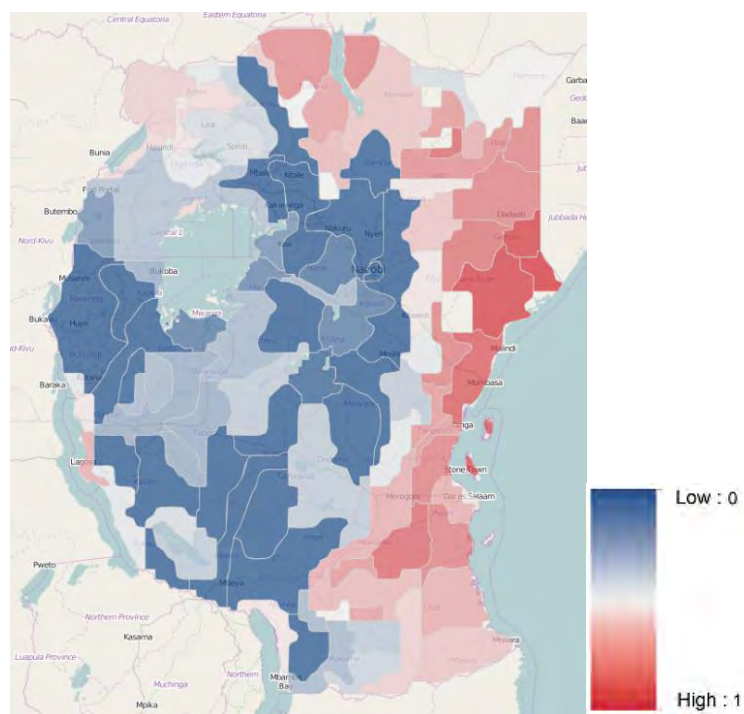


Figure 20: Risk map for malaria in the study area

This pattern and distribution of present day malaria risk may surprise, as presently different or other hot spots of malaria risk are known. Especially lower risk values would be expected along the Kenyan coast, higher values for significant areas in Uganda, and in central Tanzania (e.g. when compared with Gething *et al.*, (2011)). To identify the causes for this pattern, it is required to decompose malaria risk into its sub-domains of hazard (reflected through the EIR) and its social vulnerability, the later modelled as a multi-dimensional and spatially-explicit composite indicator. Both sub-domains - present day EIR values (as the hazard proxy) and social vulnerability to malaria - are shown in Figure 21.

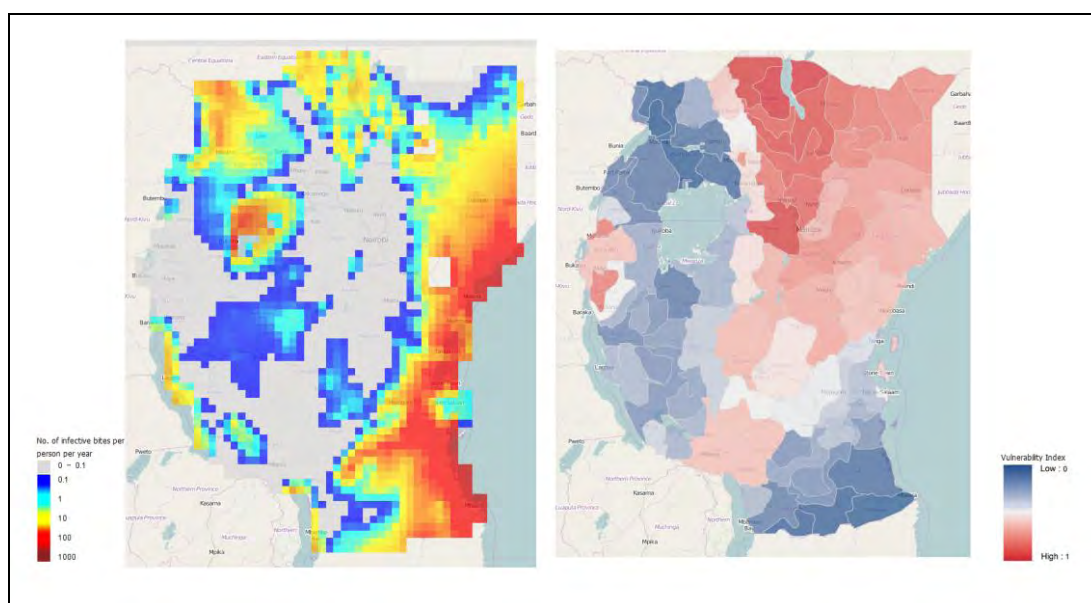


Figure 21: Present day EIR values (left) and social vulnerability to malaria (right)

As we can depict from this figure 21, the malaria model results (derived from the VECTRI model presented in D3.4 (Leedale 2014)) in high EIR values along the east African coast, 'above' Lake Victoria and in northern Kenya. The combination of the EIR values with the social vulnerability by the means of an equally weighted geometric mean amplifies areas along the Tanzanian/Kenyan coast and northern Kenya towards high risk values as they are characterised by high social vulnerability values. In these regions the high social vulnerability values are mainly due to low immunity, deficits in education, the minor use of protective measures, and partly due to limitations in access to health facilities as well as higher dependency ratios. Additional social vulnerability hot spots in Rwanda and Burundi, as well as south-western Tanzania do receive lower risk values due to lower EIR values. This vulnerability pattern is the result of high biological susceptibility (low immunity), relatively poor use of bed nets (where Rwanda is partly better off) and higher rates of poverty. As a consequence the final malaria risk mapping result needs to be contextualised within the capabilities of a dynamical malaria modelling approach. This requires further discussion and uptake, where a joint interpretation with malaria modelling experts needs to be carried out before translating that into clear policy recommendations (Leedale 2014).

Future scenarios for EIR have been modelled based on the RCP4.5 and RCP8.5 scenarios (see above) based on two malaria models, LMM and VECTRI respectively. The two models are discussed in detail in D3.4 (Leedale 2014), but do rely mainly on varying variables of temperature and rainfall. These scenarios are therefore climate scenarios and impact on the hazard layer only. The results are provided for both models as decadal means up to 2100. Though economic scenarios for these two time scale exist, their outputs are at national level. To downscale these scenarios to sub-regional level and make them spatially explicit for each of the indicators is a whole research on its own and currently such data are not available. Therefore, vulnerability maps have been kept constant to present day conditions and not been adjusted for these scenarios.

RCP4.5 assumes a stabilisation of radiative forces after about 40 years. Figure 22 presents the model outputs of D3.4 for present-days conditions, mid-century (decadal mean of 2046-2055), and end-century (decadal mean of 2086-209) for VECTRI and LMM. From a global perspective both models provided similar results as well as comparable minimum and maximum values of EIR. However, significant differences can be identified in northern and north-eastern Kenya as well as in the region of Rwanda and Burundi and scattered model

results in the east African highland area. When looking at the change (‘delta’) of the EIR values between the decadal means of 2006-2015(present-days) and 2086-2095 (far future) according to the LMM model a decrease of EIR values will occur along the coast and parts of southern and central Tanzania (close to Lake Victoria), with a scattered increase in the east African highlands as well as the area of Rwanda and Burundi. Comparing that to the VECTRI model outputs there is not only a difference in the EIR delta value magnitude but also the change pattern differs with a general increase throughout the study area with minor decreases in the north-eastern part of Kenya. The results for both scenarios and its implications are, as already mentioned, in more details discussed in D3.4. However, it is important to reflect and be aware about trends and differences as they impact the pattern of future malaria risk.

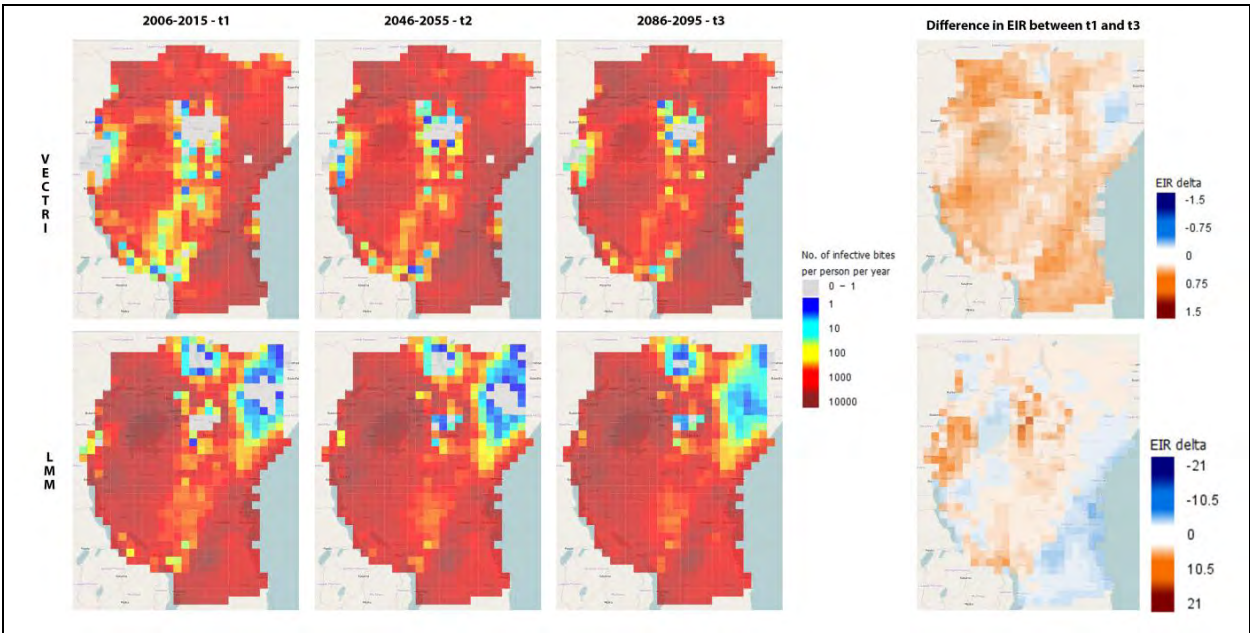


Figure 22: RCP4.5 EIR scenarios for LMM and VECTRI model results

RCP8.5 assumes a steady increase in radiative forces until 2100. The very general pattern is comparable to the RCP4.5 scenario, however this ‘worst-case’ scenario is more amplified with higher values throughout the region. Towards the end of the century large parts of east Africa might have relatively high EIR values in the LMM model with the exception of high mountain regions and the northern and north-eastern parts of Kenya (Figure 23).

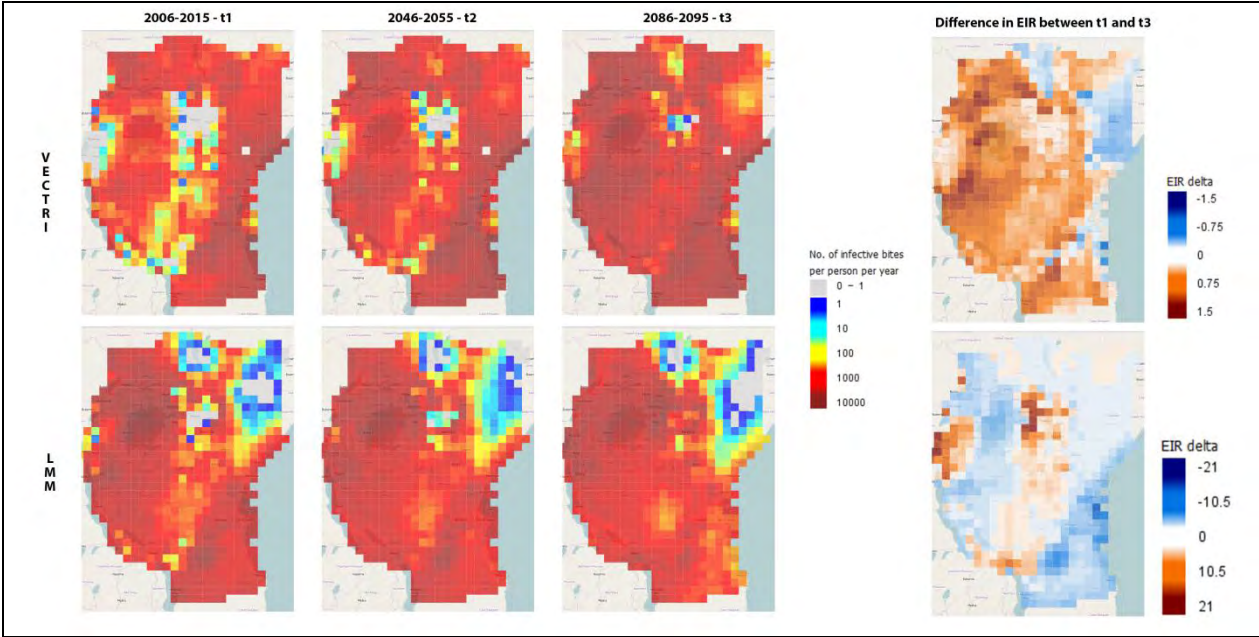


Figure 23: RCP8.5 EIR scenarios for LMM and VECTRI model results

As mentioned above, in the future risk assessment social vulnerability is kept constant, whereas changes in the risk pattern are due to changes in the hazard layer. In summary, malaria risk seems to be relatively high throughout the region, whereas the two model outputs provide different patterns as described above. This leads either to future hot spots in the east African highlands and cold spots in the northern and north-eastern parts of Kenya following the LMM model results (Figure 24 – see also Annex 1). Based on the VECTRI model outputs (Figure 25 – see also Annex 1), present-day cold spots are the regions of Rwanda and Burundi where changes will occur for both scenarios, with much critical changes and increases in the RCP8.5 scenario. The highest risk values are and might be observed in the northern and north-eastern regions of Kenya, which is in contradiction with the LMM model outputs (as

discussed).

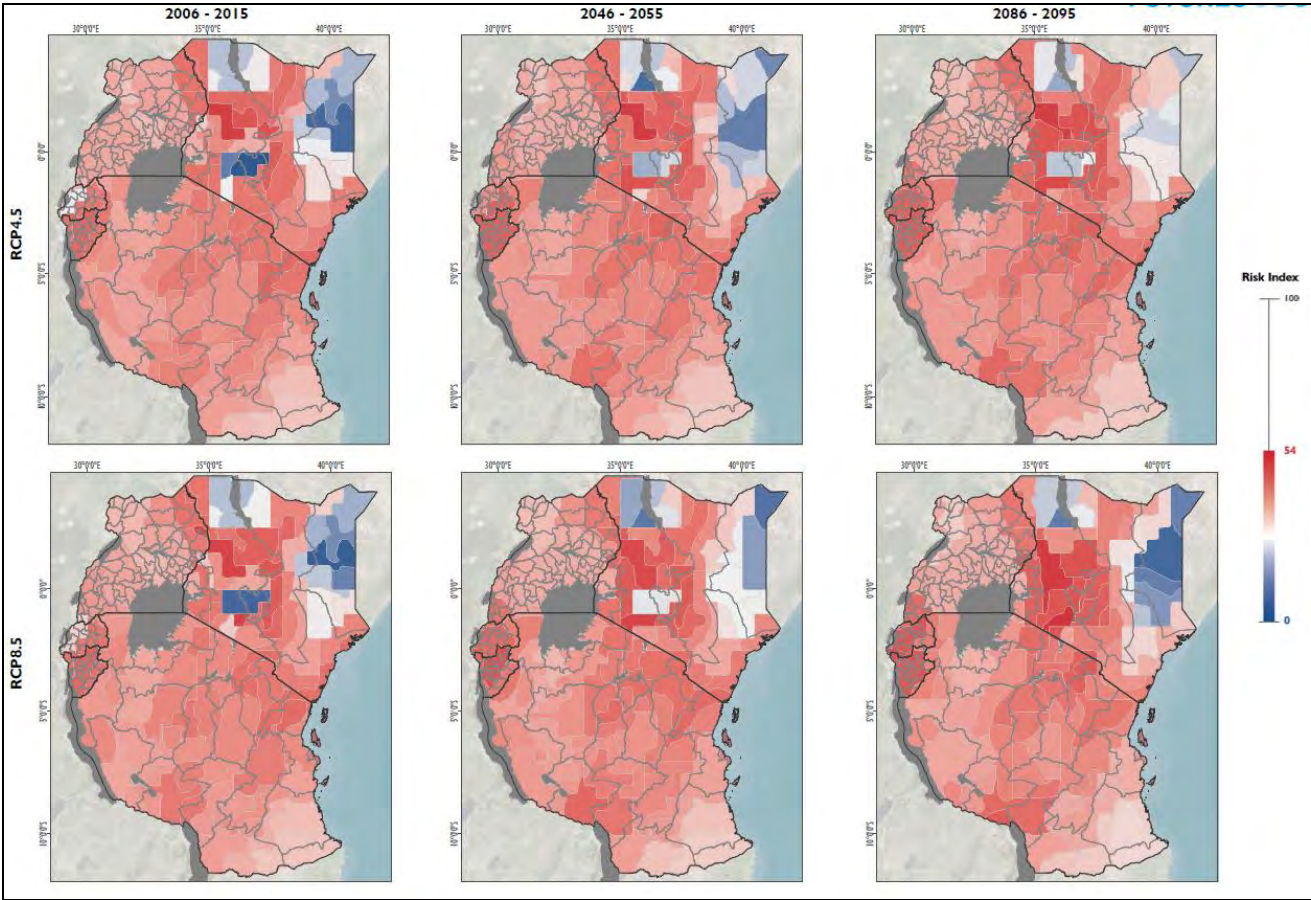


Figure 24: Present and Future Malaria Risk (LMM model)

A preliminary conclusion from the malaria risk assessment is that the validity of modelling outputs – for both the hazard and social vulnerability domain – and respectively inputs to the final risk aggregation are key. There are uncertainties and limitations included based on methodological challenges as well as the quality of data and in regard to the understanding of a complex and non-linear behaviour, especially when integrating environmental with socioeconomic data. However, we do believe that we demonstrated also within the malaria case study the integrative nature of the new IPCC framing of risk and applied it successfully for the malaria case study. Based on that, it is possible to decompose the different domains of risk into its underlying components. Combined with spatially-explicit modelling approaches this allows the identification of appropriate intervention measures in a multi-dimensional spectrum of environmental and social drivers. The research underlies also the need to view malaria risk from a holistic and integrative perspective, which not only addresses the direct causes of malaria, but also its root causes related to the socioeconomic conditions and the vulnerability of different population groups.

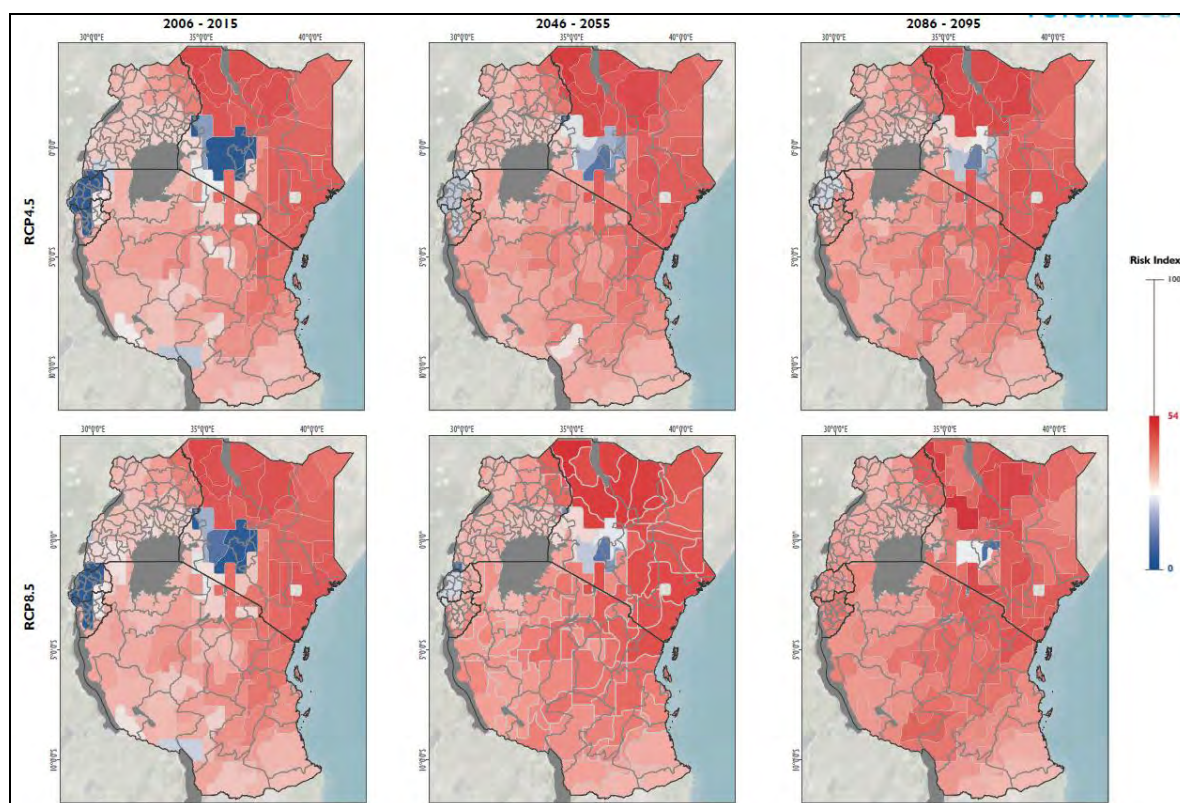


Figure 25: Present and Future Malaria Risk (VECTRI model)

2.2. Schistosomiasis

Table 2 provides an overview of the hazard (i.e. disease-related) and vulnerability indicators that were integrated into the spatial risk assessment. The risk assessment results for schistosomiasis shown in Figure 26 for the present days is characterised by high risk values throughout the study region. This high values and their regions are altered by cold spots in north-eastern Kenya and north of Nairobi in the east African highlands. Lower risk values also occur in parts of Rwanda and Burundi, along the central coast and in parts of southern Tanzania.

This pattern and distribution of present day schistosomiasis risk is influenced by the sub-domains of risk, namely hazard and social vulnerability (both are presented in Figure 27). The hazard proxy for the potential distribution of schistosomiasis is the suitable temperature for transmission. Therefore this is a purely climatological indicator reflecting the variations of temperature in the region. More details on the mathematical modelling approach are presented in D3.4 (Leedale 2014). Suitable temperature conditions therefore occur in the western parts of the study region, with modifications in the highlands of Rwanda and Burundi, ‘above’ the lakes and along the coast.

Table 2: Schistosomiasis risk factors (including vulnerability indicators) for eastern Africa

Indicator name	Date	Resolution ^b	Sign ^c	Weight	Data source
Hazard					
Temperature suitability for transmission	2010	~50km	+	0.5	D3.4 (Leedal 2014)
Vulnerability					
<i>Generic susceptibility (SUS)</i>					
Number of school aged children	2010	1 km	+	0.0617	AfriPop:demography
Population change	1970-2010	2.5-arc minutes	+	0.0150	GPWv3, UNEP-APD
Travel time to closest urban center	2000	30 arc-seconds	+	0.0367	JRC/WorldBank
Distance to roads	2010	Line layer	+	0.0300	OSM, ESA GlobCover, SRTMv4
Conflict density (km ²)	1997-2009	Point layer	+	0.0433	ACLED
Number of people living on less than 2 USD per day	2010	2.5 arc-minutes	+	0.1000	CGIAR CSI
Prevalence of non-improved toilets (%)	2007/08	Point layer	+	0.1800	DHS
Prevalence of non-improved sources of water (%)	2007/08	Point layer	+	0.2283	DHS
<i>Lack of capacity to anticipate (C2A)</i>					
Secondary/higher education (%)	2007/08	Point layer	-	0.1050	DHS
<i>Biological susceptibility (BIO)</i>					
Prevalence of stunting children under the age of 5	2010	5 arc-minutes	+	0.0450	FAO
HIV prevalence among 15-49 year olds (%)	2010	Polygon layer	+	0.0417	USAID
<i>Lack of capacity to cope (C2C)</i>					
Distance to closest hospital	2010	Point layer	+	0.0683	OSM, ESA GlobCover, SRTMv4
Number of dependents	2010	1 km	+	0.0133	AfriPop:demography

^aBased on the outcomes of the literature survey, expert consultation and data availability; ^b Refers to the spatial resolution of the original datasets (i.e., before the data was resampled to 10 x 10 km² grids); ^c Sign indicates if high indicator values increase (+) or decrease (-) risk.

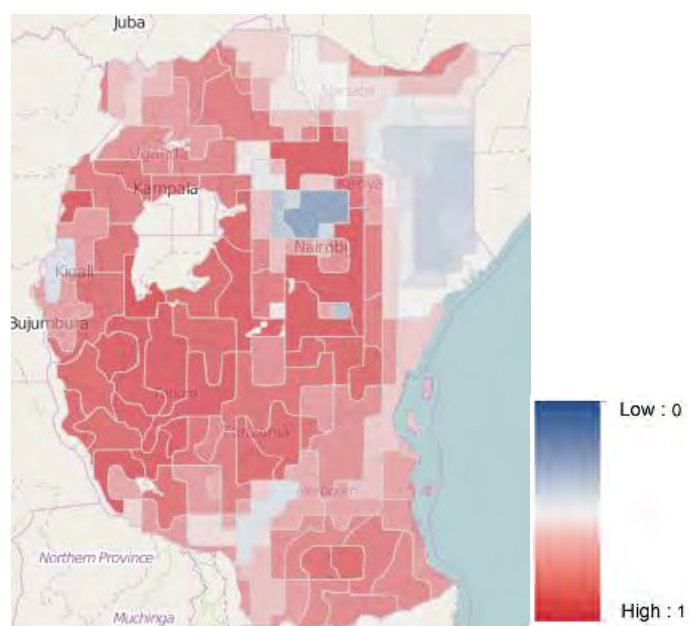


Figure 26: Risk map for schistosomiasis in the study area

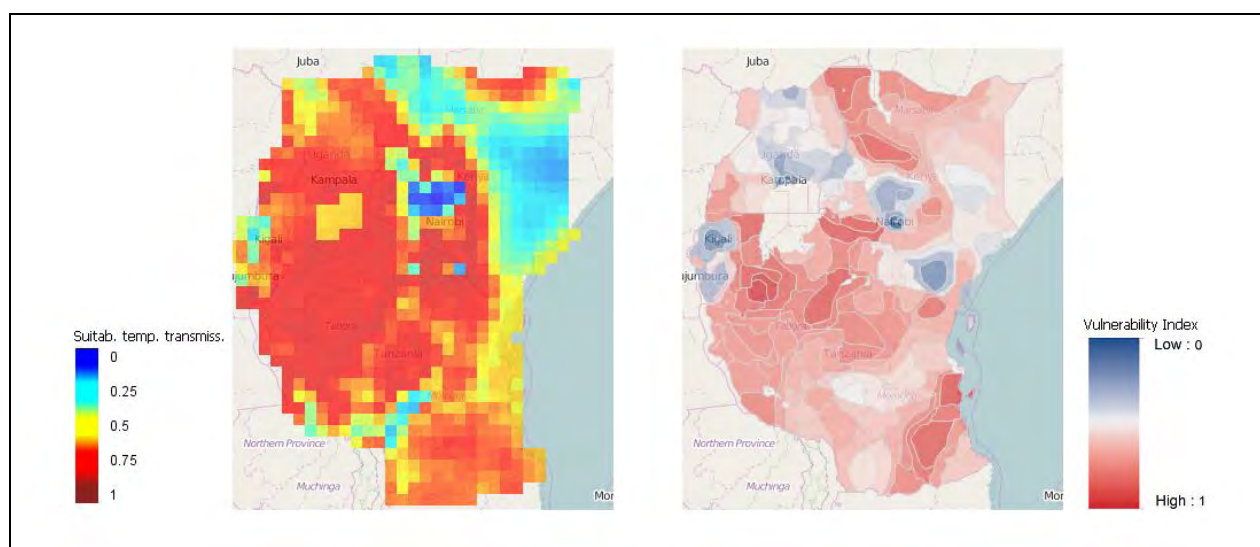


Figure 27: Present day suitable temperature values for schistosomiasis transmission (left) and related social vulnerability (right)

Non-suitable conditions characterise the area north of Nairobi. The very hot regions in northern and north-eastern Kenya also reduce the suitability for schistosomiasis transmission significantly. It is important to mention that the modelling does not include the availability of water bodies, and therefore provides an indication of potential schistosomiasis transmission only. Lower social vulnerability levels occur north of Nairobi as well as in the Rwandan and Burundian highlands. This is due to better situations related to sanitation, improved drinking water sources and the dependency ratio compared to other regions. In the Rwandan and Burundian highlands a general better situation compensates the higher lack of education. A similar pattern can be found throughout Uganda, where still challenges on sanitation, education and high dependency ratios exist. The social vulnerability hot spots occur partly

spread out through the region, especially in very rural areas. Again this are characterised by a high deficit in accessibility but with critical values in the domain of sanitation, improved drinking water sources, lack of higher education of head of family and high dependency ratios. There is a slight variation among the hot spots which provides them with a unique characteristic among the listed factors. In combination with the hazard layer, the large number of hot spot regions becomes evident, with the above mentioned cold spots in specific regions characterised, by low hazard and/or lower vulnerability values as mentioned above.

Future scenarios for suitable temperature transmission have been modelled based on RCP4.5 and RCP8.5 scenarios (see above) until 2050 only. Again the modelling approach is discussed in detail in D3.4 (Leedale 2014). The results are provided as decadal means up to 2050. Social vulnerability maps have been kept constant to present day conditions and not been adjusted for these scenarios.

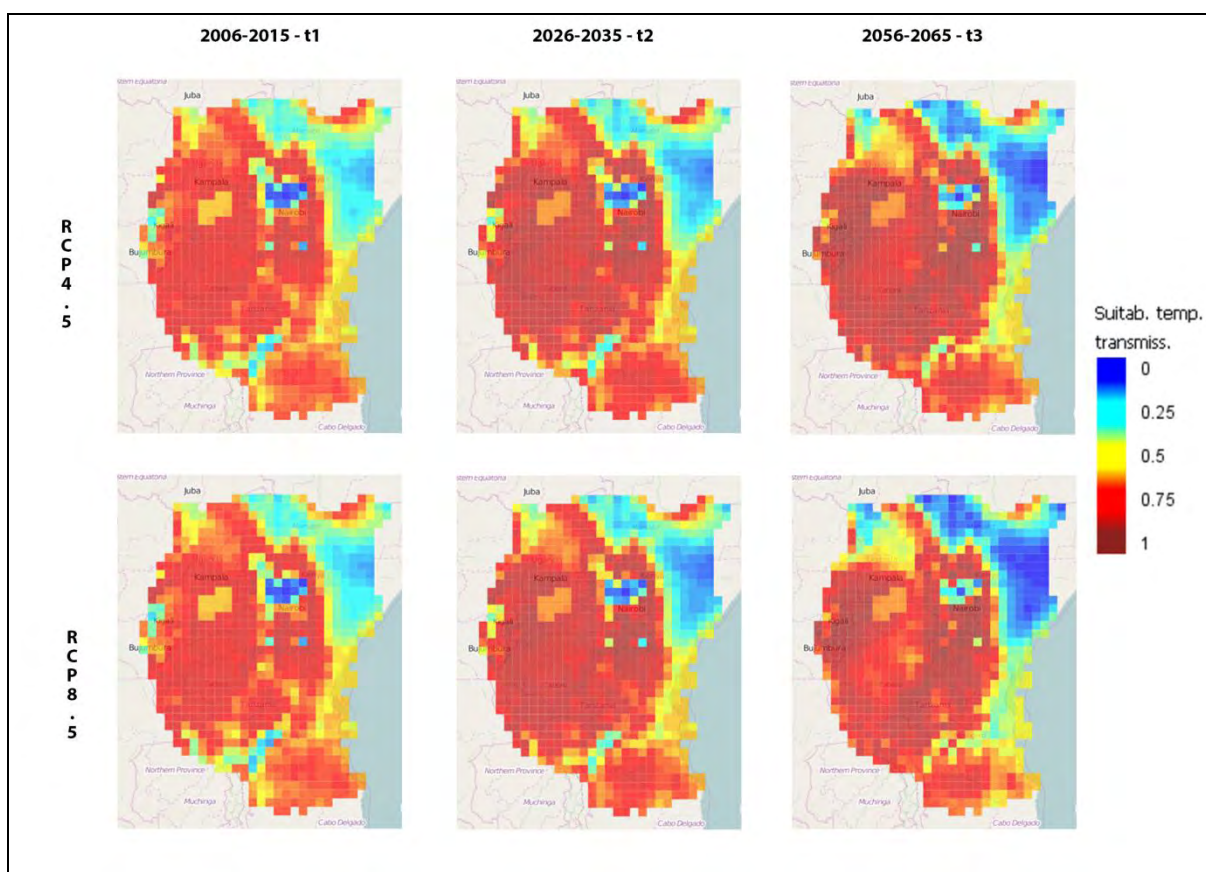


Figure 28: RCP4.5 and RCP8.5 scenarios for suitability of schistosomiasis transmission

As it can be seen in Figure 29, there is not much variation between the two scenarios as climate change may have significant differences between the two scenarios beyond 2050 only. However, looking at the maps it can be seen that there will be a general increase in temperature suitability throughout the region. Some former less suitable areas will become

more suitable, such as Rwanda and Burundi and related highlands. The area north of Nairobi will increase its suitability in general but will still remain very low until 2050. Those areas with presently less suitable temperature will become even more unsuitable such as the northern and north-eastern parts of Kenya but also along the coast in the study region. Interestingly to observe is the change in north-western Uganda in the Lake Albert region, where schistosomiasis temperature dependent transmission will decrease in the future.

As mentioned above, in the future risk assessment social vulnerability is kept constant, whereas changes in the risk pattern are due to changes in the hazard layer (see Figure 29).

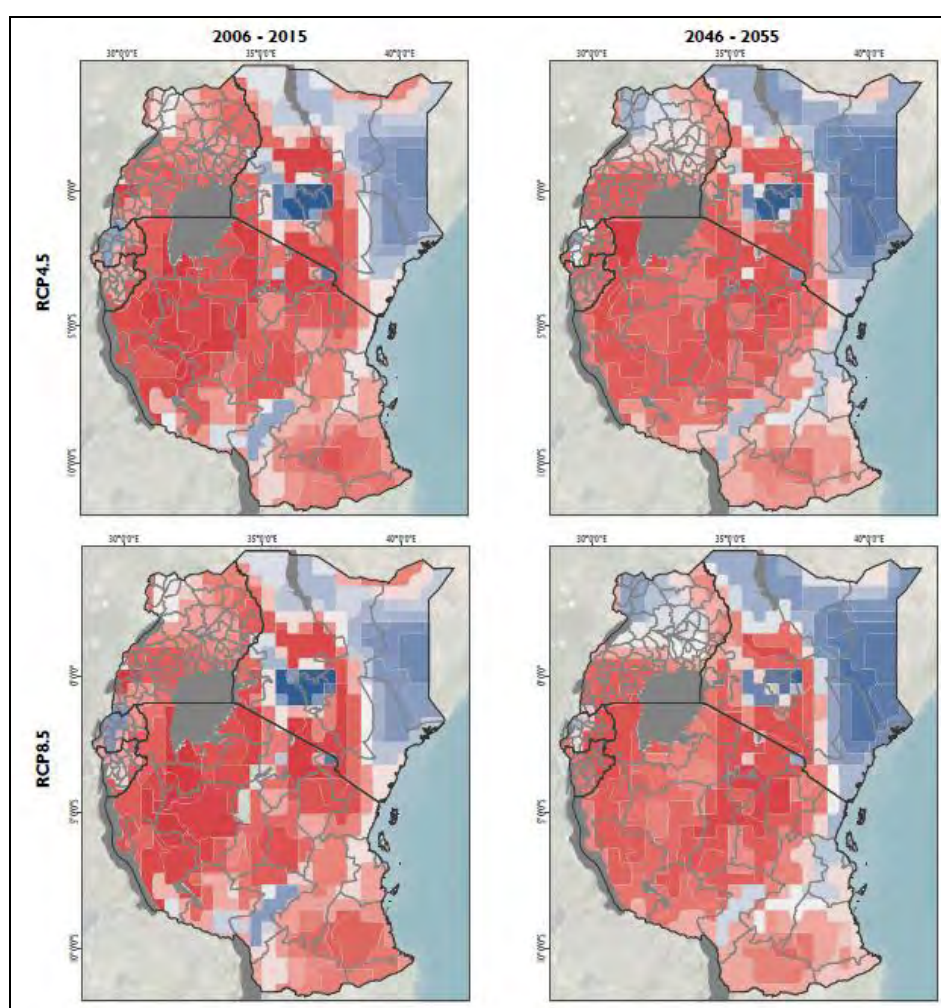


Figure 29: Present and future schistosomiasis risk

In combination with the social vulnerability maps it is interestingly to observe that some regions will be characterised in the future by lower risk values. This is specifically true for the northern part of Uganda, northern and north-eastern part of Kenya as well as along the coast. However, there will be an increase in risk in Rwanda and Burundi as well as throughout central Tanzania.

A concluding point in relation to the schistosomiasis risk assessment is that patterns will also vary throughout the study region significantly. Not only based on different amplitude of values of specific factors but also in combination and relation of these. Next to that, further emphasis needs to be given to better identify future environmental driven risk factors (such as water availability and quality) and changes in socioeconomic development, which are key for schistosomiasis prevalence.

2.3. Rift Valley fever

Table 3 provides an overview of the hazard (i.e. disease-related) and vulnerability indicators that were integrated into the spatial risk assessment. The risk assessment as shown in Figure 30 for the present days point out two particular hotspots in the East African region to Rift Valley fever: in central Kenya, the central western area of Lake Victoria, namely in Rwanda and Burundi, as well as northern and south-western part of Tanzania.

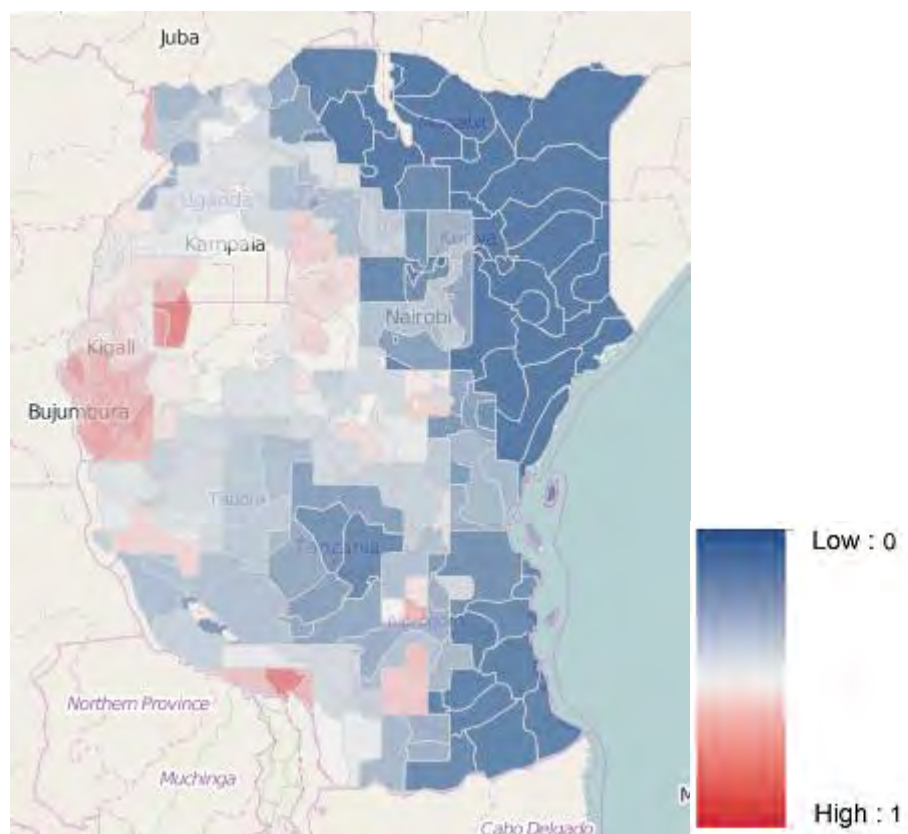


Figure 30: Risk map for RVF in the study area

These findings compare favourably with those presented earlier by (Anyamba *et al.* 2002) although the present results have not captured other potential hotspots in the north and north-eastern parts of Kenya. These hot spots represent ecologies where the primary and secondary vectors of RVF (floodwater *Aedes* spp., *Culex* spp., *Mansonia* and other mosquito species) and hosts (livestock and wildlife) thrive and interact. They also represent areas where flooding readily occurs following prolonged precipitation. This is because the areas generally have a gentle topography, with clayey soils that readily get clogged within a short period of time. It is believed that the RVF virus is maintained through inter-epidemic periods in the eggs of floodwater *Aedes* mosquitoes that remain dormant in the soils.

Table 3: Rift Valley fever risk factors (including vulnerability indicators) for eastern Africa

Indicator name	Date	Resolution ^b	Sign ^c	Weight	Data source
Hazard					
Entomological inoculation rate (EIR) - culex	2010	~50km	+	0.5	D3.4 (Leedal 2014)
Vulnerability					
<i>Generic susceptibility (SUS)</i>					
Tropical livestock unit	2006	0.00833 degrees	+	0.1679	FAO
Aridity index	2009	0.00833 degrees	+	0.1419	CGIAR CSI
Number of people living on less than 2 USD per day	2010	2.5 arc-minutes	+	0.2228	CGIAR CSI
<i>Lack of capacity to anticipate (C2A)</i>					
Secondary/higher education (%)	2007/08	Point layer	-	0.0522	DHS
Knowledge/experience with RVF	2014	Point layer	-	0.1717	FAO IMPRESS
Households with radio (%)	2007/08	Point layer	-	0.0626	DHS
Households with mobile phone (%)	2007/08	Point layer	-	0.0418	DHS
<i>Lack of capacity to cope (C2C)</i>					
Travel time to urban centers (proxy for distance to markets)	2010	Point layer	+	0.0695	OSM, ESA GlobCover, SRTMv4
Distance to road networks	2010	Line layer	+	0.0695	OSM, ESA GlobCover, SRTMv4

^a Based on the outcomes of the literature survey, expert consultation and data availability; ^b Refers to the spatial resolution of the original datasets (i.e., before the data was resampled to 10 x 10 km² grids); ^c Sign indicates if high indicator values increase (+) or decrease (-) risk; ^d This indicator was removed from the analysis to reduce existing multicollinearities in the data.

This risk map can be decomposed by hazard and by vulnerability. Our results present two hazard maps, for each scenario, based on Culex EIR and the other on Aedes EIR mosquitoes. In principle, these rates ought to be combined given that RVF transmission involves both vectors; Aedes spp is a primary vector that often initiates the transmission while Culex and other mosquitoes play a major role in the amplification of the disease. The hazard map based on Aedes EIR indicates areas where RVF is likely to persist, while that based on Culex EIR shows areas that have high potential for RVF epidemics when the virus is introduced.

The EIR of Culex shown in Figure 31 shows two hot spots, namely around Kampala and Lake Victoria as well as central and South Tanzania. Most of Kenya has a low EIR and therefore hazard probability. This might be because a large part of the country is arid and semi-arid where mosquito populations are always low in most periods of the year. The EIR of Aedes in Figure 31 looks pretty different. Its two hotspots, one in the Western part of Lake Victoria and one at the Ugandan and Kenyan borders are bigger. North East of Kenya as well as Tanzania are cold spots.

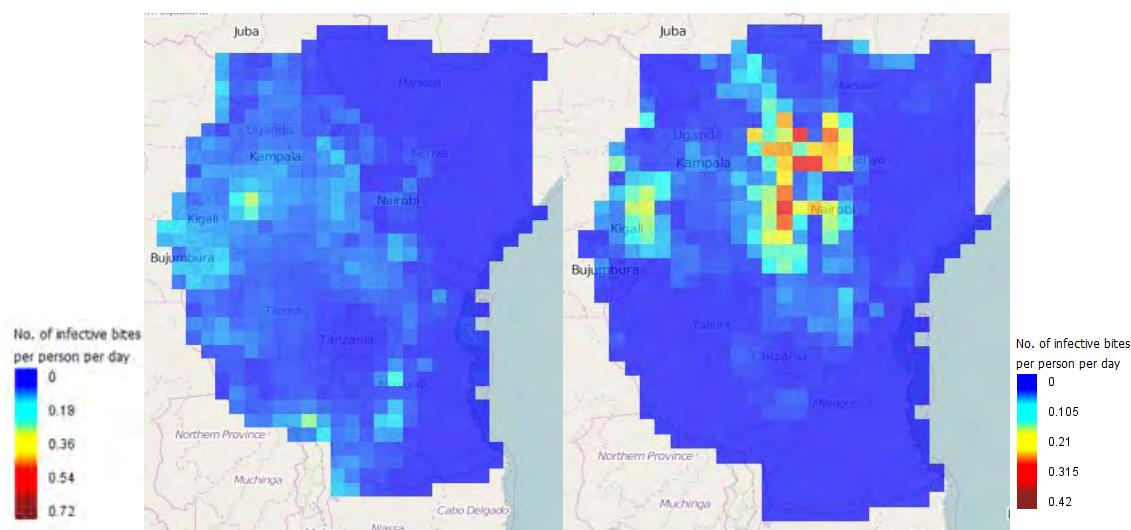


Figure 31: EIR for Culex (left) and EIR for Aedes (right)

Given the difference between the two maps, the choice of one of the two maps results is a crucial assumption. The Culex map has been retained in this context because it is the main mosquito responsible for spreading RVF.

Vulnerability is introduced with its subcomponents, generic susceptibility capacity to anticipate, capacity to cope and recover. Note that for RVF there is not a biological susceptibility as for the other two VBD. This is because we assess RVF for livestock and little is known about biological susceptibility of RVF in livestock. The vulnerability map shown in Figure 32 suggests that the central eastern part of the study area, namely around Nairobi to the coast to a certain extent Tanzania are least vulnerable to RVF. This pattern is the result of relatively low poverty, easy access to market and information as well as higher education levels. On the contrary all bordering areas of the study area are more vulnerable, mainly because of fewer infrastructures and to certain extent to lower education and lower access to media and phone.

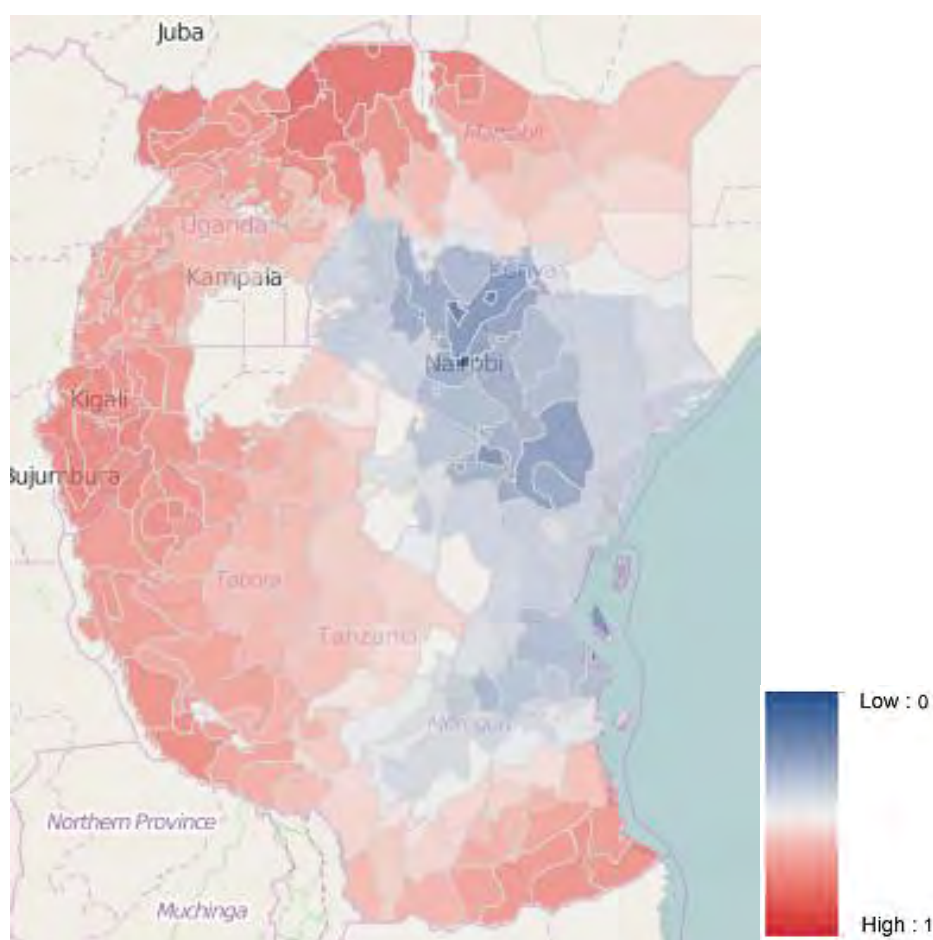


Figure 32: Vulnerability map for RVF

These patterns can be analysed in more detailed by breaking them down to the several components of vulnerability shown in Figure 33. Susceptibility is highest in the north-western part of the study area that is mainly driven by high number of livestock, relatively high poverty rate and aridity. Capacity to anticipate, shows very similar patterns that the vulnerability as a whole, whereas as the region from central part of the study area towards

the coast is much better off than the rest of the study area, and is mainly driven by access to information through phone or radio as well as knowledge about the disease. The latter assumes that smallholders who have been affected in the last outbreak know better how to recognise RVF and act accordingly. Finally the capacity to cope and recover is mainly driven by access to markets and roads, and northern Kenya as well as some parts in Tanzania are least best off.

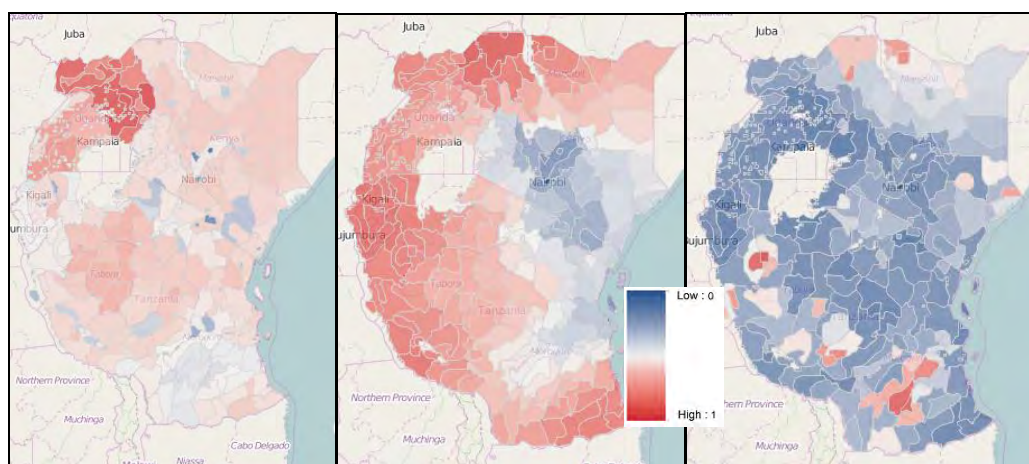


Figure 33: the different component of vulnerability, susceptibility (left), capacity to anticipate (middle), capacity to cope/recover (right)

Two of the four representative concentration pathways from IPCC, namely RCP4.5 and RCP8.5 have been modelled for in 40 and 80 years. The varying variables are temperature and rainfall only. Though other drivers, such as land use are known as important factors for RVF, their change were too complex to be modelled within the frame of this research. These scenarios are therefore climate scenarios and impact on the hazard layer only. They have been recomputed for both time scales in both scenarios. Though economic scenarios for these two time scale exist, their outputs are at national level. To downscale these scenarios to sub-regional level for each of the indicators is a whole research on its own. Therefore, the vulnerability map has not been adjusted for these scenarios and has been assumed constant. Because the Culex map has been used to map vulnerability, also assumes that the risk map will be changing over the next 80 years with the same patterns than the Culex maps.

The RCP8.5 scenario assumes a steady increase in radiative forcing until 2100. The hazard hot spot for Culex is surprisingly decreasing in the first 30 years and then starts increasing again around the current hot spot over the next 80 years. The hazard within the hot spots will be more important. Whereas north-eastern Kenya always has a relatively low hazard, Tanzania will have a lower hazard in 80 years. However the hot spot will be moving towards the centre of the study area.

The pattern for Aedes is very much similar; the high hazards hot spot decrease to the extent that the hot spot in the East of the study area disappears. However, hazard values become much higher in the current hot spots.

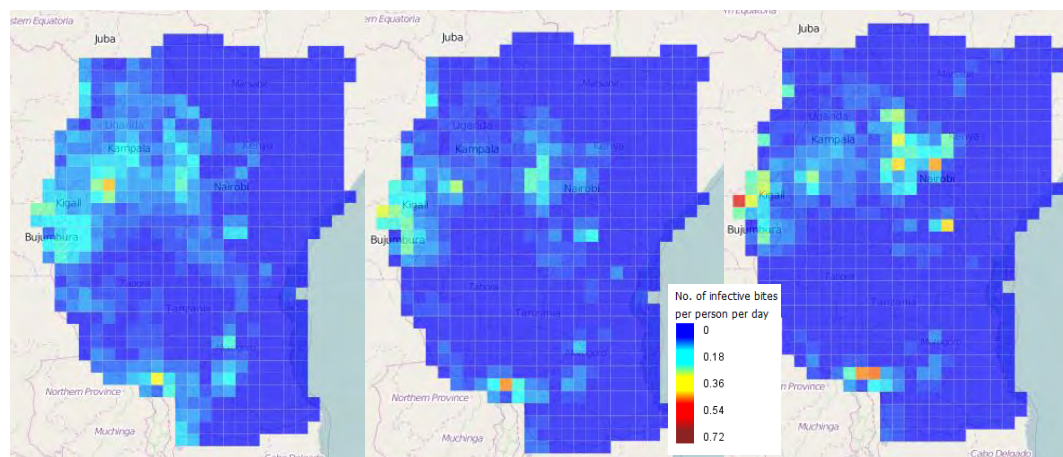


Figure 34: Scenarios for EIR for Culex, base run (left), after 40 years (middle), after 80 years (right) for scenario RCP 8.5

The RCP4.5 scenario (stabilisation without overshoot pathway to 4.5 W/m² at stabilisation after 2100) assumes a stabilisation of radiative forcing after about 40 years. As Figure 35 shows, the hazard hot spot for Culex is increasing around the current hot spot over the next 40 year and will cover the whole of the western part of the study area despite of the stabilisation of radiative forcing. After year 40 the hotspot decreases again. Kenya always remains relatively protected.

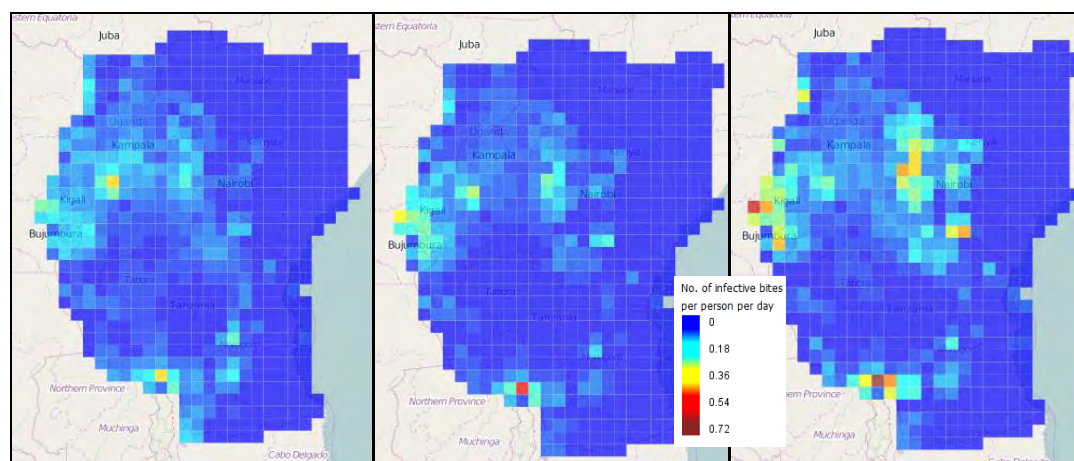


Figure 35: Scenarios for EIR for Culex, base run (left), after 40 years (middle), after 80 years (right) for scenario RCP 4.5

Interestingly the dynamics for Aedes is different as shown in Figure 36, whereas in the upcoming years the hazard hot spot is slightly increasing, it is actually reducing in the next 80 years, but the hazard within the hot spot located in the central area of the study area, is becoming higher.

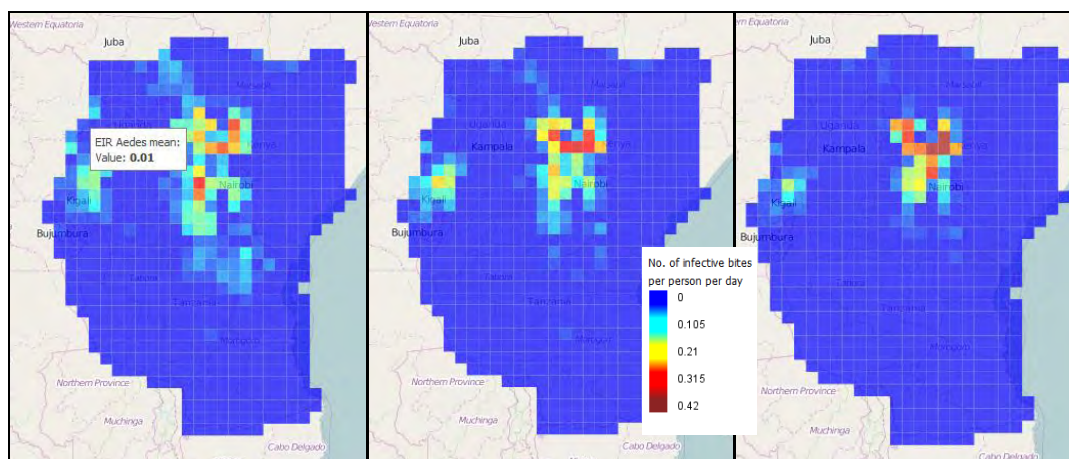


Figure 36: Scenarios for EIR for Aedes, base run (left), after 40 years (middle), after 80 years (right) for scenario RCP 4.5

In summary, RVF has most of its risk in two hot spots, namely Western part of Lake Victoria and its one spot in southern Tanzania. Figure 47 shows the risk map for the scenario RCP 8.5, and Figure 38 the risk maps for scenario RCP 4.5 both of which can also be found in the Annex 1. Note that EIR for Culex has been used as the hazard map. In both scenarios the hot spots are on similar locations and tend to move more eastwards over time. Scenario RCP 4.5 though assumes to have stabilisation after 40 years; the no risk zone is smaller than in scenario RCP8.5. Though the extent of the risk only marginally changes the intensity of the risk in scenario RCP8.5 is bigger in the hot spots over the years.

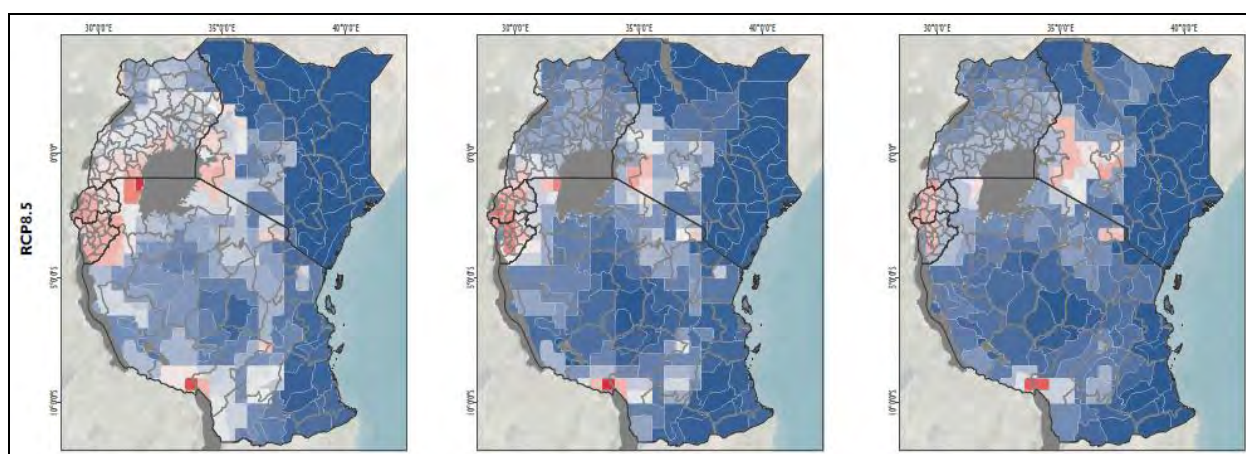


Figure 37: Scenarios for risk, base run (left), after 40 years (middle), after 80 years (right) for scenario RCP 8.5

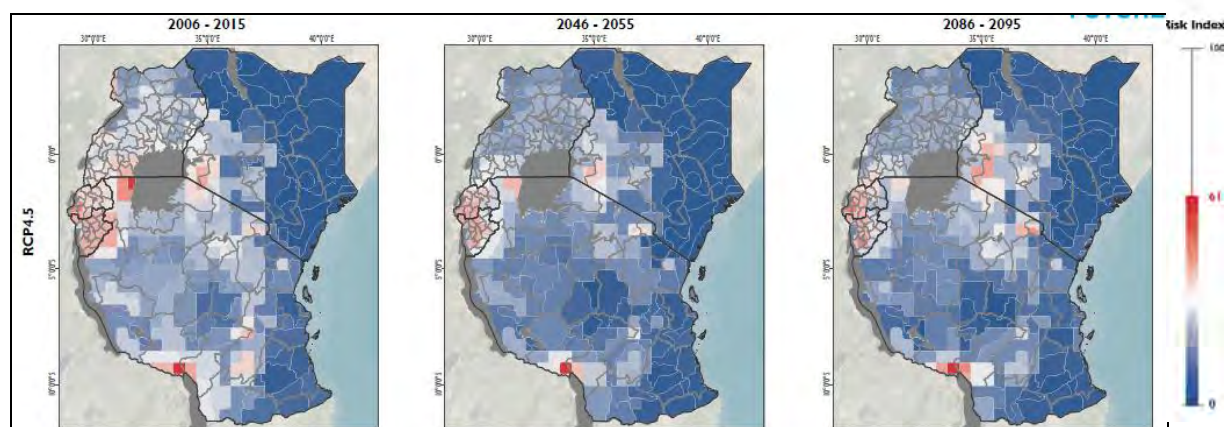


Figure 38: Scenarios for risk, base run (left), after 40 years (middle), after 80 years (right) for scenario RCP 4.5

The largest difference between the two scenarios can be found in western Tanzania, where scenario RCP4.5 predicts and increase in the risk whereas scenario RCP 8.5 predicts a much smaller increase. With breaks out every 6-10 years leading to major economic losses, RVF is quite different from other VBDs. Early warning systems are one of the most crucial approaches to deal with an outbreak to plan responses and mitigations efficiently. These early warning systems until today were mostly based on rainfall predications, they did not allow for identifying where the interventions will benefit most to people. This research has developed an approach to understand in which location populations might benefit most from mitigation interventions. Defining these population through the vulnerability concept allow now to target a multi-dimensional approach beyond relatively uncertain poverty measurement. This new type of information will provide policy-makers and other stakeholders who have the capacity to intervene in an outbreak better by allocating their limited resources more efficiently. The results from this research have shown that pastoralist areas are more vulnerable than the high potential areas of eastern Africa.

However, this research has also pointed out that RVF is the VBD that has data challenges. This is the limiting factor to develop better information for policy support. Better surveillance systems that reporting more consistently during outbreaks, will be needed to better understand the ecology of the disease and identify other locations at risk where no outbreaks have yet been observed. Also, more consistent reporting will allow to better assessing ex-post impacts not only on the producers but also the other actors of the meat value chain and economy as a whole.

3. Incorporation of the spatial assessment of risk and vulnerability to the decision support frameworks

Current status of targeted diseases prevalence has been presented and its spatial distribution assessed as well as situation of future diseases taking into account environmental and socio-economic features. While the research findings are easily accessible, each country possesses a proper planning, institutions and institutional arrangement that can use created knowledge in various ways in decision making to ensure surveillance for early detection and intervention in VBD outbreaks. To benefit from HF research outputs, there is a need for a shift toward proactive vector control approaches and integrated vector management strategies. Such approaches should consider, in addition to diseases prevention, surveillance, a wider context of environmental management including land use plan and implementation, reducing climate change impacts, etc. It has been observed that complex human–environment interactions constitute a fundamental component of diseases problem in East Africa. In the case of malaria in particular, complicated relationships exist between land use change for agriculture and malaria outcomes. Converting natural swamps to agriculture for instance led to an increase in temperatures and mosquito vectors, leading to higher malaria risks in these areas compared to areas where natural swampland was maintained (Lindblade *et al.*, 2000). There is a need for a strategy to integrate diseases risk management in various activities impacting on environment and health.

3.1. Decision support framework for RVF

In the framework of HF, DSFs were developed for the 3 VBDs: malaria, Schistosomiasis and RVF. For RVF, through different meetings and consultations, an existing DSF was refined. In all 4 meetings were organised by HF. Deliverable 5.4 is an output of such consultations. In terms of decision points, a sequence of events characterising the progressive increase and eventual decrease in risk of an RVF epidemic was determined starting from normal situation between epidemic, through the height of an epidemic with confirmation of case in both livestock & humans and the eventual return to inter-epidemic period. The sequence includes (D 5.4):

1. Inter-epidemic period
2. Pre-outbreak

- EW -Early warning of RVF issued and/or early warning of heavy rain by national meteorological departments
- Alert
- Localised, prolonged heavy rains reported by eye-witnesses
- Localised flooding reported by eye-witnesses
- Localised mosquito swarms reported by eye-witnesses

3. Outbreak

- Suspected outbreak – either in animals or humans
- First detection of suspected RVF in livestock by active searching and/or rumours from herders
- First rumour or field report of human RVF case
- Confirmed outbreak
- Laboratory confirmation of RVF cases in livestock
- Laboratory confirmation of first human RVF case

4. Recovery phase

- No new human cases
- No clinical livestock cases for 45 days
- Post-outbreak recovery and reflection

5. Inter-epidemic period (i.e. same as event 1)

In addition to decision point, categories of intervention (below) were defined and activities specified for both human and animal health (D5.4)

Under each of the decision points specified above, the following interventions may be carried out:

- Capacity building and training
- Communication, advocacy and public awareness
- National and regional coordination
- Early warning
- Surveillance
- Disease prevention
- Vaccination
- Vector control
- Infection, prevention and control

- Case management
- Regulation of trade and markets for livestock
- Resource mobilisation
- Establishing or strengthening institutions and policies
- Research
- Risk, impact and climate change assessment

The framework developed is put into use by decision makers when there is RVF outbreak.

It has been observed that decision makers are sometimes reluctant to take decision to prevent RVF outbreak with the risk of wasting resources if action is taken too early while there is no danger. The tendency is then to wait until an epizootic has begun and in this case it is too late to take effective preventive and control measures with all the loss of human lives, livestock and resources. Therefore there is need to reinforce the use of the framework as a preventive tool. In this way, the move can also accommodate better the impact of climate dynamic that impact on the spatial distribution of the hazard. The analysis of risk and vulnerability to RVF has revealed that areas likely to be affected today are not the ones to be affected in the future due to varying temperature. Also the vulnerability differs from one area/country to another, meaning that some areas need to be prioritised in intervention compared to other. What needs to be taken into consideration to integrate better the hazard and vulnerability dimension is also the fact that the hazard model outputs provide information on spatial distribution of risk that is useful at regional level of EAC. There is a need to integrate in the framework the variation of the spatial distribution between the present and futures hazards and the resulting social vulnerability. The changes are due to changing climatic conditions and socio-economic situation of affected community. For instance for the present day non suitable temperature values for schistosomiasis transmission, are located in north Nairobi, northern and north Eastern Kenya, in Rwandan and Burundian highland. Future scenario for suitable temperature transmission have been modelled based on RCP 4.5 and RCP 8.5 until 2050 and the output revealed that some former less suitable areas become more suitable, such as Rwanda and Burundi. The area of Nairobi will increase its suitability in general but will still remain very low until 2050. On the contrary, the north eastern Uganda around Lake Albert will know decreased transmission of the diseases. Therefore decision makers should take into consideration areas at risk from RVF risk maps, the RVF outbreak history in a specific area as well as possibility of occurrence in new areas.

3.2. Decision support framework for malaria and Schistosomiasis

In the framework of HF, a DSF for malaria and Schistosomiasis was developed as part of task 5.5. The framework was developed as a guide to respond to the disease in the face of climate change. Malaria has benefited from major international control programs in place and the risk based DSF is a complement to existing policy and strategy document. In the case of schistosomiasis, on the other hand, sustainable, international control efforts stemming from decisions made by local actors are lacking and the institutional context of schistosomiasis control within the East Africa region could benefit from further development. The deliverable 5.5 specifically examines how malaria epidemiology may be affected in the future by three inter-related phenomena: climate change; environment and land use change. The DSF takes into consideration the changing risk and vulnerability to malaria as a result of climate change. Through the consultation on the DSF for malaria, experts have agreed that the process of changing temperatures and rainfall pattern will reshape patterns of agriculture production, settlement and environmental suitability for vectors of disease. Some of these shifts will be dramatic when they occur and difficult to predict as they are driven by a complex interaction of climatic, social and politico-economic determinants (D 5.5.). As table 4 shows, the decision points for malaria are various and cover different areas. Risk and vulnerability have been taken into consideration in the DSF. However, more needs to be done to complement the information availed by the risk and vulnerability models as there are other factors impacting on diseases occurrence. So the framework should leave room for integration of other information particularly on land use dynamics and socio-economic changes.

Table 4: Decision point and action categories for malaria

Decision point	Action categories
<p>1. Pre-existing national program Describes assumptions about baseline malaria prevention and control programs activities in place</p>	<p>1. Management and coordination 2. Planning and strategy 3. Health Systems 4. Legislation 5. Communication and social mobilisation 6. Vector Control</p>
<p>2. Population migration and displacement: Events include increased migration or movement from malaria endemic areas into lower risk areas OR increase in susceptible population</p>	<p>7. Environmental management 8. Surveillance - Climate variability 9. Surveillance - Vector 10. Surveillance - Parasite 11. Monitoring and evaluation 12. Data management</p>

<p>(population growth and movement)</p> <p>3. Change in environment including land use change This was defined to changes in the environment that would affect malaria risk such as changes in land use and cover, changes in hydrology, etc. However, it was not defined to include climate change</p> <p>4. Climate change This event category includes long-term In temperature and rainfall</p>	<p>13. Policy 14. Finance 15. Human resources and capacity building 16. Advocacy 17. Risk, Impact and Climate Change Assessment 18. Research</p>
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The time horizon for the DSF for malaria is 20 years and 30 years for Schistosomiasis. Considering changes in diseases distribution modelled on the basis of projection of climate changes, this time horizon seems short if climate factors have to be fully integrated in the framework. The DSFs are implemented at country level. However, as the diseases outbreaks are the results of complex relation between climate, environment, land use human mobility, etc., regional collaboration can enable a better implementation of such framework.

In the case of schistosomiasis: The Schistosomiasis DSF is intended to be complementary to regional and national policies and strategies, as they may be lacking actions to be taken related to the effects of regional and local environmental change on schistosomiasis transmission. The DSF can play a strong role in catalysing appropriate

3. Conclusion

The spatial assessment of present day and future risks (and vulnerabilities) associated with the three target VBDs serves as a basis for exploring the complex nature of these three diseases in eastern Africa. The assessment of risk, hazard and social vulnerability was based on a conceptual framework that was developed within HF while adhering to the latest definitions of risk, vulnerability, resilience and adaptation by the IPCC (IPCC, 2014). The results of the spatial risk assessments illustrate the complexity of the three target diseases in eastern Africa and aim at encouraging a more profound examination of the manifold drivers of these three diseases in a spatial context. The study confirms what has already been widely discussed in literature as well by relevant key experts: that factors contributing to disease risk are spatially diverse and often distinct, meaning that any formulation of generalised

explanations cannot be drawn without ignoring a more complex reality. Thus, an integrative spatial-explicit view of risk, which integrates socioeconomic and demographic as well as environmental (disease-related) factors, has several advantages: the spatially explicit assessment clearly shows that substantial differences exist within the region, both in terms of the values and the nature of vulnerability of the population as well as regarding the spatial variability in disease prevalence. Since the composite risk index builds on a set of single disease-related and vulnerability indicators, each region (geon) can also be decomposed into its risk components (hazard, vulnerability), vulnerability domains (generic susceptibility, biological susceptibility, lack of capacity to anticipate, lack of capacity to cope), and into its factors (i.e. vulnerability indicators), enabling an identification of the contributions of the single indicators to the overall risk. In this way, the risk assessment can support the development of context-specific and spatial-explicit (for each region) interventions to reduce (1) disease prevalence with potential impacts and (2) prevailing vulnerabilities of the population. Limitations arise from the uncertainties which are introduced by concepts used, data quality and availability and methods applied. It is therefore of utmost importance that these uncertainties are communicated to users and decision makers in a transparent and meaningful manner. At the same time, users need to be aware of the strengths and weaknesses of such modelling results which should then allow a meaningful reasoning and interpretation of modelling and map outputs based on data and methods applied.

4. References

- Anyamba, A., Chretien, J.P., Small, J., Tucker, C.J., Formenty, P.B., Richardson, J.H. *et al.* (2009). Prediction of a Rift Valley fever outbreak. *Proceedings of the National Academy of Sciences of the United States of America*, 106, 955-959. doi:910.1073/pnas.0806490106.
- Anyamba, A., Linthicum, K.J., Mahoney, R. & Tucker, C.J. (2002). Mapping Potential Risk of Rift Valley fever outbreaks in African Savannas using Vegetation Index Time Series Data. *Photogrammetric Engineering & Remote Sensing* 68, 137-145.
- Baatz, M. & Schäpe, A. (2000). Multiresolution Segmentation – an optimization approach for high quality multi-scale image segmentation. In: *Angewandte Geographische Informationsverarbeitung* (eds. Strobl, J, Blaschke, T & Griesebner, G). Wichmann: Heidelberg, pp. 12-23.
- Bayne, C.J. & Loker, E.S. (1987). Survival within the snail host. In: *The Biology of Schistosomes from Genes to Latrines*. (eds. Rollinson, D & Simpson, AJG). Academic Press London, pp. 347-378. .
- Boelee, E. & Madsen, H. (2006). Irrigation and Schistosomiasis 99 in Africa: Ecological Aspects; International Water Management Institute; (IWMI Research Report 99, 39p.
- Brooker, S. & Clements, A.C.A. (2009). Spatial heterogeneity of parasite co-infection: Determinants and geostatistical prediction at regional scales. *International Journal of Parasitology*, 39, 591-597.
- Brooker, S., Kabatereine, N.B., Gyapong, J.O., Stothard, J.R. & Utzinger, J. (2009). Rapid mapping of schistosomiasis and other neglected tropical diseases in the context of integrated control programmes in Africa. *Parasitology*, 136, 1707-1718.
- Bukenya, G.B., Nsungwa, J.A., Makanga, B. & Salvator, A. (1994). Schistosomiasis mansoni and paddy-rice growing in Uganda: an emerging problem. *Annals of Tropical Medicine and Parasitology* 88, 379-384.
- Caminade, C., Ndione, J.A., Kebe, C.M.F., Jones, A.E., Danuor, S., Tay, S. *et al.* (2011). Mapping Rift Valley fever and malaria risk over West Africa using climatic indicators. *Atmospheric Science Letters*, 12, 96-103. doi: 110.1002/asl.1296.
- Chamwali, L.A. (2013). The economic burden of malaria, A Post Field Report Submitted to AERC in Partial Fulfillment of the Requirement for the Degree of Doctor of Philosophy (Economics) of the University of Dar es Salaam, November, 2013.
- Chandiwana, S.K. & Woolhouse, M.E. (1991). Heterogeneities in water contact patterns and the epidemiology of Schistosoma haematobium. *Parasitology* 103, 363-370.
- Chevalier, V., Rakotondrafara, T., Jourdan, M., Heraud, J.M., Andriamanivo, H.R., Durand, B. *et al.* (2012). An unexpected recurrent transmission of rift valley Fever virus in cattle in a temperate and mountainous area of madagascar *PLoS Neglected Tropical Diseases* 5, e1423.
- Clennon, J., Mungai, P., Muchiri, E., King, C. & Kitron, U. (2006). Spatial and temporal variations in local transmission of Schistosoma haematobium in Msambweni, Kenya. *American Journal of Tropical Medicine and Hygiene*, 75, 1034-1041.
- Clennon, J.A., King, C.H., Muchiri, E.M., Kariuki, H.C., Ouma, J.H., Mungai, P. *et al.* (2004). Spatial patterns of urinary schistosomiasis infection in a highly endemic area of coastal Kenya. *The American Journal Tropical Medicine and Hygiene* 70, 443-448.
- Cook, J.H. (1909). Distribution of Bilhaziasis on the Victoria Nyanza. 1:.
- Craig, M.H., Snow, R.W. & Le Sueur, D. (1999). A climate-based distribution model of malaria transmission in sub-Saharan Africa. *Parasitology Today*, 15, 105-111. .
- Daubney, R., Hudson, J.R. & Granham, P.C. (1931). Enzootic hepatitis or Rift Valley fever: an undescribed virus disease of sheep, cattle and man from East Africa. *Journal of Pathology and Bacteriology* 34, 545-579.

- Davies, F.G., Linthicum, K.J. & James, A.D. (1985). Rainfall and epizootic Rift Valley Fever. *Bulletin of the World Health Organization* 63, 941-943.
- Doumenge, J.P., Mott, K.E. & Reud Thomas, G. (1987). *Atlas of the Global distribution of schistosomiasis: Talence, CEGET-CNRS. Geneva: WHO Publication.*
- Drăgut, L., Csillik, O., Eisank, C. & Tiede, D. (2014). Automated parameterisation for multi-scale image segmentation on multiple layers. *ISPRS Journal of Photogrammetry and Remote Sensing*, 88, 119–127.
- Engels, D., Ndoricimpa, J. & Gryseels, B. (1993). Schistosomiasis mansoni in Burundi: progress in its control since 1985. *World Health Organization Bulletin*, 71, 07–214.
- Ernst, K.C., Adoka, S.O., Kowuor, D.O., Wilson, M.L. & Chandy, C.J. (2006). Malaria hotspot areas in a highland Kenya site are consistent in epidemic and non-epidemic years and are associated with ecological factors. *Malaria Journal*, 5, doi: 10.1186/1475-2875-1185-1178.
- Fritzsche, K., Schneiderbauer, S., Bubeck, P., Kienberger, S., Buth, M., Zebisch, M. et al. (2014). *The Vulnerability Sourcebook: Concept and guidelines for standardised vulnerability assessments. Deutsche Gesellschaft für Internationale Zusammenarbeit (GIZ) GmbH, Bonn und Eschborn, Germany, 180p.*
- Füssel, H.M. (2007). Adaptation planning for climate change: concepts, assessment approaches, and key lessons. *Sustainability Science*, 2, 265-275.
- Gething, P.W., Patil, A.P., Smith, D.L., Guerra, C.A., Elyazar, I.R.F., Johnstone, G.L. et al. (2011). A new world malaria map: Plasmodium falciparum endemicity in 2010. *Malaria Journal*, 10, <http://www.malariajournal.com/content/10/11/378>.
- Government of Rwanda (2006). Rwanda Demographic and Health Survey 2005; Final Report; National Institute of Statistics of Rwanda, July 2006.
- Government of Uganda (2014a). Annual Health Sector Performance Report Financial Year 2013/14, Government of Uganda, Ministry of Health, Kampala, page 111-112.
- Government of Uganda (2014b). Draft Uganda Malaria Reduction Strategic Plan 2014-2020, Government of Uganda, Ministry of Health, Kampala.
- Groen, E.T. & Jacobs, C. (2012). Risk Mapping Burundi Sector Disaster Risk Reduction & Emergency Aid. Cordaid, 6 p.
- Gryseels, B. (1994). Human resistance to Schistosoma infections: age or experience? . *Parasitology Today* 10, 380-384.
- Hagenlocher, M., Delmelle, E., Casas, I. & Kienberger, S. (2013). Assessing socioeconomic vulnerability to dengue fever in Cali, Colombia: statistical vs expert-based modeling. *International Journal of Health Geographics*, 12, <http://www.ij-healthgeographics.com/content/12/11/36>.
- Hagenlocher, M., Kienberger, S., Lang, S. & Blaschke, T. (2014). Implications of spatial scales and reporting units for the spatial modelling of vulnerability to vector-borne diseases. In: *GI_Forum 2014: Geospatial Innovation for Society* (eds. Jekel, T, Car, A, Strobl, J & Griesebner, G). Heidelberg: Wichmann Salzburg, Austria, pp. 197-206.
- Hoshen, M.B. & Morse, A.P. (2004). A weather-driven model of malaria transmission. *Malaria Journal*, 3, doi:10.1186/1475-2875-1183-1132.
- Imbahale, S.S., Mukabana, W.R., Orindi, B., Githeko, A.K. & Takken, W. (2012). Variation in Malaria Transmission Dynamics in Three Different Sites in Western Kenya. *Journal of Tropical Medicine and Hygiene*, ID 912408, 8 pages, <http://dx.doi.org/10.1155/2012/912408>.
- IPCC (2001). Impacts, Adaptation, and Vulnerability. The Contribution of Working Group II to the Third Scientific Assessment of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge.
- IPCC (2007). Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge, United Kingdom and New York, NY, US.
- IPCC (2012). Managing the Risks of Extreme Events and Disasters to Advance Climate Change Adaptation. A Special Report of Working Groups I and II of the Intergovernmental Panel on Climate Change (Field CB, Barros V, Stocker TF, Qin D,

- Dokken DJ, Ebi KL, Mastrandrea MD, Mach KJ, Plattner G-K, Allen SK, Tignor M, Midgley PM (eds.). Cambridge University Press, Cambridge, UK, and New York, NY, USA. .
- Isabwe, A., Ruberanziza, E., Mupfasoni, D., Ruxin, J., Clerinx, J. & White, P.T. (2012). Potential for transmission of schistosomiasis in Kayonza district. *Rwanda Medical Journal*, 69, 14-19.
- Ishengoma, D.S., B.P, M., Segeja, M.D., Alifrangis, M., Lemnge, M.M. & Bygbjerg, I.C. (2013). Declining burden of malaria over two decades in a rural community of Muheza district, north-eastern Tanzania. *Malaria Journal*, 12, doi:10.1186/1475-2875-1112-1338.
- Ivorra Cano, V. (1967). Paludisme. In Santé et maladies au Rwanda. Administration Générale de la Coopération au Développement Bruxelles, pp. 427-447.
- Ivorra Cano, V. (1983). Paludisme. In: *Santé et Maladies au Rwanda* (eds. Meheus, A, Butera, S, Eylenbocsh, W, Gatera, G, Kivits, M & Musafili, I). Administration Générale de la Coopération au Développement Bruxelles, pp. 427-447.
- Ivorra Cano, V. (1994). Evaluation of the malaria situation in Rwanda and in Rwandan refugee camps in Tanzania, Burundi and Zaire, 7 October-9 November, WHO (unpublished document).
- Jordan, P., Webbe, G. & Sturrock, R. (1993). *Human schistosomiasis*. Wallingford, England.
- Kabatereine, N.B., Brooker, S., Tukahebwa, E.M., Kazibwe, F. & Onapa, A.W. (2004). Epidemiology and geography of *Schistosoma mansoni* in Uganda: implications for planning control. *Tropical Medicine & International Health*, 9, 72-80.
- Kabatereine, N.B., Kazibwe, F. & Kemijumbi, J. (1996). Epidemiology of schistosomiasis in Kampala, Uganda. *East African Medical Journal* 73, 795-800.
- Kajangwa, D., Nindagiye, F. & Guajardo, J. (2013). Burundi Literacy Boost Midline Report. World Vision, 52 p.
- Kardoff R, Gabone RM, Mugashe C, Obiga D, CE, R., Mahler, C. *et al.* (1997). *Schistosoma mansoni* related morbidity in Ukerewe Island, Tanzania: Clinical, ultrasonographical and biochemical parameters. *Tropical Medicine & International Health*, 2, 230-239.
- Kienberger, S. & Hagenlocher, M. (2014). Spatial-explicit modeling of social vulnerability to malaria in East Africa. *International Journal of Health Geographics*, 13.
- Kienberger, S., Notenbaert, A., Vervoort, J. & Snel, D. (2013). Future socioeconomic changes. Deliverable Report 4.4 (D4.4) of the HEALTHY FUTURES research project (HEALTHY FUTURES FP7: 266327).
- Kinoti, G. (1964). Observations on the transmission of *Schistosoma haematobium* and *Schistosoma bovis* in the Lake Region of Tanganyika. *Bulletin of the World Health Organization*, 31, 815-823.
- Lafferty, K.D. (2009). The ecology of climate change and infectious diseases. *Ecology* 90, 888-900. <http://dx.doi.org/810.1890/1808-0079.1891>.
- Lang, S., Kienberger, S., Tiede, D., Hagenlocher, M. & Pernkopf, L. (2014). Geons – domain-specific regionalization of space. *Cartography and Geographic Information Science*, 41, 214-226. <http://dx.doi.org/210.1080/15230406.15232014.15902755>.
- Leedale, J. (2014). Integrated Disease Transmission Models. HEALTHY-FUTURES-Deliverable Report 3.4, 77p.
- Lichoti, J., K, Kihara, A., Oriko, A.A., Okutoyi, L.A., Wauna, J.O., Tchouassi, D.P. *et al.* (2014). Detection of Rift Valley Fever Virus Interepidemic Activity in Some Hotspot Areas of Kenya by Sentinel Animal Surveillance, 2009-2012. *Veterinary Medicine International*, 2014, doi:10.1155/2014/379010.
- Linthicum, K.J., Davies, F.G., Kairo, A. & Bailey, C.L. (1985). Rift Valley fever virus (family unyviridae, genus Phlebovirus). Isolations from Diptera collected during an inter-epizootic period in Kenya *Journal of Hygiene*, 95, 197-209.
- Maclean, G., Webbe, G. & Msangi, A.S. (1958). A report on a bilharzia and molluscan survey in the Tanga district of Tanganyika. *East African Medical Journal*, 35, 7-22.

- Magendantz, M. (1972). The biology of *Biomphalaria choanomphala* and *B. sudanica* in relation to their role in the transmission of *Schistosoma mansoni* in Lake Victoria at Mwanza, Tanzania. *Bulletin of the World Health Organization*, 47, 331-342.
- Magona, J.W., Galiwango, T., Walubengo, J. & Mukiibi, G. (2013). Rift Valley fever in Uganda: Seroprevalence and risk factor surveillance vis-à-vis mosquito vectors, anti-RVF virus IgG and RVF virus neutralizing antibodies in goats. *Small Ruminant Research*, 114, 176-181.
- Malenganisho, W.L., Magnussen, P., Friis, H., Siza, J., Kaatano, G., Temu, M. *et al.* (2008). *Schistosoma mansoni* morbidity among adults in two villages along Lake Victoria shores in Mwanza District, Tanzania. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 102, 532-541.
- Martin, V., Chevalier, V., Ceccato, P., Anyamba, A., De Simone, L., Lubroth, J. *et al.* (2008). The impact of climate change on the epidemiology and control of Rift Valley fever. Review of Science and Technology, Office of International Epizootics',. *Review of Science and Technology, Office of International Epizootics*, 27: , 413-426.
- Mazigo, H.D., Nuwaha, F., Kinung'hi, S., Morona, D., Pinot de Moira, A., Wilson, S. *et al.* (2012). Epidemiology and control of human schistosomiasis in Tanzania. *Parasites & Vectors* 5, doi:10.1186/1756-3305-1185-1274.
- Mboera, L.E.G., Senkoro, K.P., Mayala, B.K., Rumisha, S.F., Rwegoshora, R.T., Mlozi, M.R.S. *et al.* (2010). Spatio-temporal variation in malaria transmission intensity in five agro-ecosystems in Mvomero district, Tanzania. *Geospatial Health*, 4, 167-178.
- McCreech, N. & Booth, M. (2014). The effect of simulating different intermediate host snail species on the link between water temperature and schistosomiasis risk. . *PLOS ONE* 9, e87892.
- McCullough, F.S. (1972). The distribution of *Schistosoma mansoni* and *S.haematobium* in East Africa. *Tropical Geography and Medicine*, 24, 199-207.
- McKenzie, F. (2000). Why model malaria? . *Parasitology Today*, 16, 511-516.
- Meurs, L. (2014). *Schistosomamansoni* and *Schistosomahaematobium* infection and morbidity in a co-endemic focus: Integrated study of epidemiological, micro-geographical and immunological patterns. Leiden University, 20 p.
- Meyus, H., Lips, M. & Caubergh, H. (1962). L'état actuel du problème de paludisme d'altitude au Ruanda-Urundi. *Annales de la Société Belge de Médecine Tropicale*, 42, 771-782.
- Mupfasoni, D., Karibushi, B., Koukounari, A., Ruberanziza, E., Kaberuka, T., Kramer, M.H. *et al.* (2009). Polyparasite Helminth Infections and Their Association to Anaemia and Undernutrition in Northern Rwanda. *PLoS Neglected Tropical Diseases*, 3, e517. doi:510.1371/journal.pntd.0000517.
- Mutuku, F.M., King, C.H., Bustinduy, A.L., Munga, P.L., Muchiru, E.M. & Kitron, U. (2011). Impact of drought on the spatial pattern of transmission of *Schistosoma haematobium* in Coastal Kenya. *American Journal of Tropical Medicine and Hygiene*, 85, 1065-1070.
- Mwambungu, J.A. (1988). Transmission of *Schistosoma* in Mkwinda (Mbozidistrict, Mbeya region, Southern highlands of Tanzania. *Journal of Helminthology*, 62, 29-32.
- Ndayishimiye, O., Ortu, G., Magalhaes, R.J.S., Clements, A., Willems, J., Whitton, J. *et al.* (2014). Control of Neglected Tropical Diseases in Burundi: Partnerships, Achievements, Challenges, and Lessons Learned after Four Years of Programme Implementation. *PLoS Neglected Tropical Diseases* 8, e2684. doi:2610.1371/journal.pntd.0002684.
- Nkurunziza, H., Gebhardt, A. & Pilz, J. (2011). Geo-additive modelling of malaria in Burundi. *Malaria Journal*, 10.
- Notenbaert, A., Kienberger, S., Bett, B., Hagenocher, M., Zeil, P. & Omolo, A. (2012). Vulnerability assessment for the eastern African region to identify hotspots. Deliverable Report 4.1 (D4.1) of the HEALTHY FUTURES research project; HEALTHY FUTURES FP7: 266327.

- O'Meara, W.P., Mangeni, J.N., Steketee, R. & Greenwood, B. (2010). Changes in the burden of malaria in sub-Saharan Africa. *Lancet Infectious Diseases*, 10, 545–555. DOI:10.1016/S1473-3099(10)70096-70097.
- Ochomo, E., Nabie, O., Bayoh, M., Walker, E.D., Abongo, B.O., Ombok, M.O. *et al.* (2013). The efficacy of long-lasting nets with declining physical integrity may be compromised in areas with high levels of pyrethroid resistance. *Malaria Journal*, 12, doi:10.1186/1475-2875-1112-1368.
- OECD (2008). *Handbook on Constructing Composite Indicators: Methodology and User Guide; Organization for Economic Co-operation and Development (OECD); ISBN 978-92-64-04345-9*, Paris.
- Ojaka, D.I., Jarvis, J.D., Matilu, M.I. & Thiam, S. (2014). Acceptance of a malaria vaccine by caregivers of sick children in Kenya. *Malaria Journal*, 13, doi: 10.1186/1475-2875-1113-1172.
- PNILP (2005). Un avenir sans le paludisme: Plan de Lutte Contre les Epidémies de Paludisme au Rwanda 2005 -2010, Ministère de la Santé, Programme National Intégré de Lutte Contre le Paludisme (PNILP); Janvier 2005.
- République du Burundi (2008). Recensement General de la Population et de l'Habitat; Ministère de l'Intérieur et Du Développement Communal.
- République du Burundi (2013). Enquête sur les Indicateurs du Paludisme 2012; Ministère de la Santé Publique et de la Lutte contre le Sida, 126pp.
- Rich, K.M. & Wanyoike, F. (2010). An Assessment of the Regional and National Socio-Economic Impacts of the 2007 Rift Valley Fever Outbreak in Kenya. *The American Journal of Tropical Medicine and Hygiene*, 83, 52-57.
- Rubaihayo, J., Moghusu, E., Clouds, P. & Abaasa, A. (2008). Schistosomiasis transmission at high altitude crater lakes in Western Uganda. *Infectious Diseases*, 8,, doi:10.1186/1471-2334-1188-1110.
- Ruberanziza, E., Mupfasoni, D., Karibushi, B., Kabera, M., Karema, C., Nyatanyi, T. *et al.* (2010). A Recent Update of Schistosomiasis Mansonii Endemicity around Lake Rweru. *Rwanda Medical Journal*, 86, 5-9.
- Rusanganwa, A. (1999). Epidemiologic Micro-stratification of paludism: Index plasmodisques and its determinants in two basic medical zones of Rwanda. Work of end of studies of the DEA in sciences of health: specialization in statistical epidemiology, Université Libre de Bruxelles.
- Shoemaker, T., Boulianne, C., Vincent, M.J., Pezzanite, L., Al-Qahtani, M.M., Al-Mazrou, Y. *et al.* (2002). Genetic analyses of viruses associated with emergence of Rift Valley fever in Saudi Arabia and Yemen 2000-2001. *Emerging Infectious Diseases*, 8, 1415 - 1420.
- Sindato, C., Karimuribo, E. & Mboera, E.G. (2011). The epidemiology and socio-economic impact of rift valley fever in Tanzania: a review. *Tanzania Journal of Health Research*, 13, DOI: <http://dx.doi.org/10.4314/thrb.v4313i4315.4311>.
- Sindato, C., Karimuribo, E.D., Pfeiffer, D.U., Mboera, L.E.G., Kivaria, F., Dautu, G. *et al.* (2014). Spatial and temporal pattern of Rift Valley fever outbreaks in Tanzania; 1930 to 2007. *PLoS ONE* 9, e88897. doi:88810.81371/journal.pone.0088897.
- Soumare, P.O., Freire, C.C., Faye, O., Diallo, M., de Oliveira, J.V., Zanotto, P.M. *et al.* (2012). Phylogeography of rift valley fever virus in Africa reveals multiple introductions in Senegal and Mauritania. *PLOS ONE*, 7, e35216.
- Stocker, T.F., Qin, D., Plattner, G.-K., Tignor, M., Allen, S.K., Boschung, J. *et al.* (2013). Climate change 2013: The physical science basis. *Intergovernmental Panel on Climate Change, Working Group I Contribution to the IPCC Fifth Assessment Report (AR5)(Cambridge Univ Press, New York)*.
- Swai, E.S. & Schoonman, L. (2009). Prevalence of rift valley fever immunoglobulin g antibody in various occupational groups before the 2007 outbreak in Tanzania. *Vector Borne Zoonotic Diseases* 9, 579-582 .doi: 510.1089/vbz.2008.0108.

- Tompkins, A.M. & Ermert, V. (2013). A regional-scale, high resolution dynamical malaria model that accounts for population density, climate and surface hydrology. *Malaria Journal*, 12, doi:10.1186/1475-2875-1112-1165.
- Tonui, W.K., Otor, S.C.J., Kabiru, E.W. & Kiplagat, W.K. (2013). Patterns and trends of malaria morbidity in western highlands of Kenya. *International Journal of Education and Research*, 1, 1-8.
- Tukahebwa, E.M., Magnussen, P., Madsen, H., Kabarereine, N.B., Nuwaha, F., Wilson, S. *et al.* (2013). A very high infection intensity of *Schistosoma mansoni* in a Ugandan Lake Victoria Fishing Community is required for association with highly prevalent organ related morbidity. *PLOS Neglected Tropical Diseases*, 7, e2268. doi: 2210.1371/journal.pntd.0002268.
- USAID (2012). Evaluation of the Impact of Malaria Interventions on Mortality in Children in Mainland Tanzania. 114 P.
- Vermeylen, M. (1967). Répartition des Anophèles de la République du Rwanda et Burundi. . *Rivista di Malariologia* 46, 13-22
- Webbe, G. (1959). A bilharzia and molluscan survey in the Handeni and Korogwe Districts of Tanganyika. *Journal of Tropical Medicine and Hygiene*, 62, 37-42.
- WHO (2007). Gender, Health and Malaria, World Health Organization; Department of Gender, Women and Health (GWH) Family and Community Health (FCH); Geneva, Switzerland, June 2007.
- WHO (2013). World Malaria Report 2013, World Health Organization; Geneva, Switzerland, 255 pp.
- Wielgosz, B., Kato, E. & Ringler, C. (2014). Agro-ecology, household economics and malaria in Uganda: empirical correlations between agricultural and health outcomes. *Malraia Journal*, 13, <http://www.malariajournal.com/content/13/11/251>.
- Wielgosz, B., Mangheni, M., Tsegai, D. & Ringler, C. (2013). Malaria in Uganda: Improved Outcomes when the Health Sector joinsforces with Agriculture. International Food Policy Research Institute, May 2013.
- World Health Organization (2012). Accelerating work to overcome the global impact of neglected tropical diseases: a roadmap for implementation. Geneva.
- Yawe, B.L. (2014). Changes in climatic factors and malaria in Uganda. World Institute for Development Economics Research. 18 p.

ANNEX 1

Present and Future Malaria Risk (LMM model)
Present and Future Malaria Risk (VECTRI model)
Present and Future Rift Valley Fever Risk (Culex)
Present and Future Schistosomiasis Risk

Present and Future Malaria Risk (LMM model)

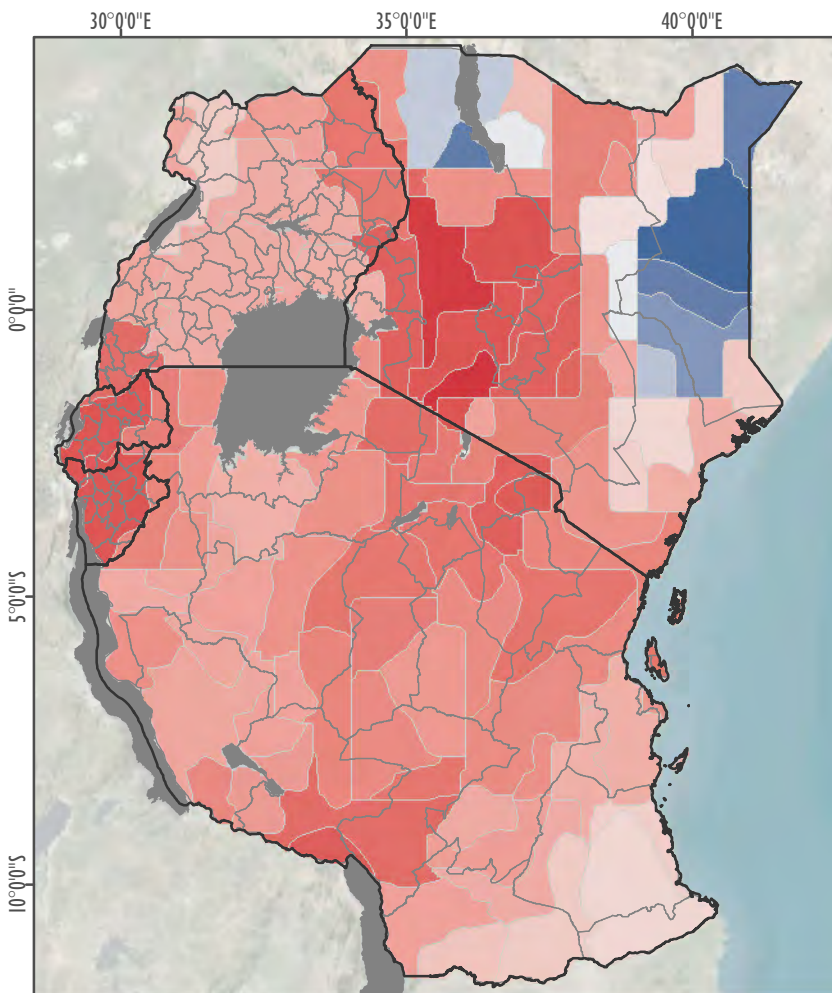
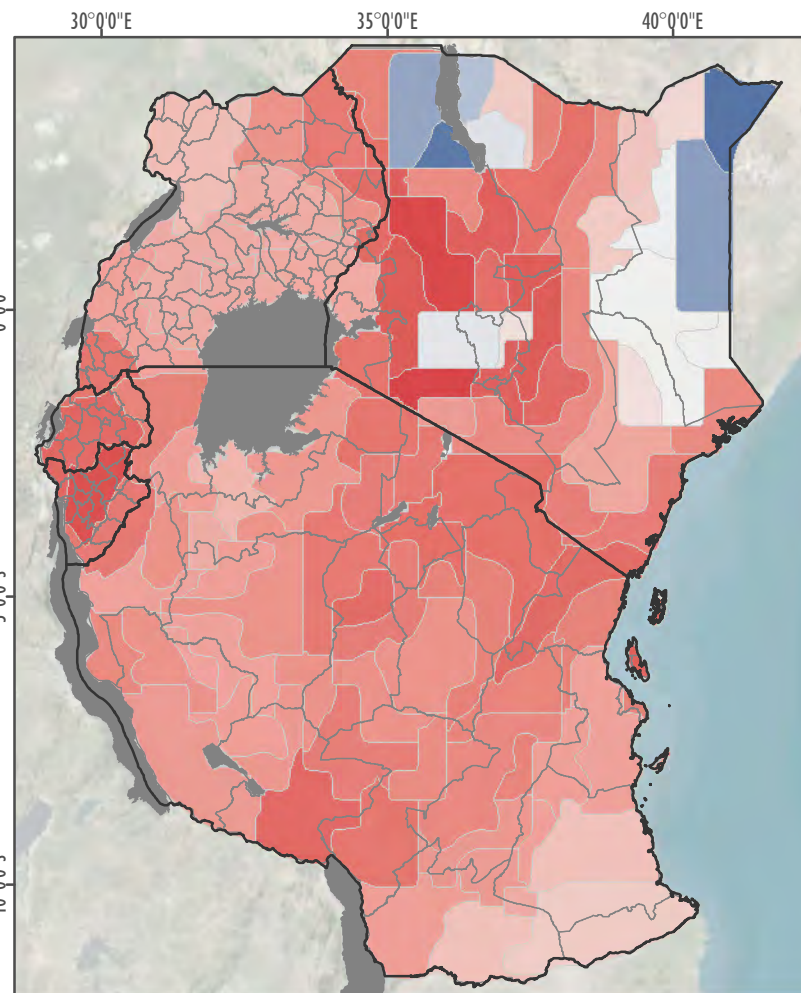
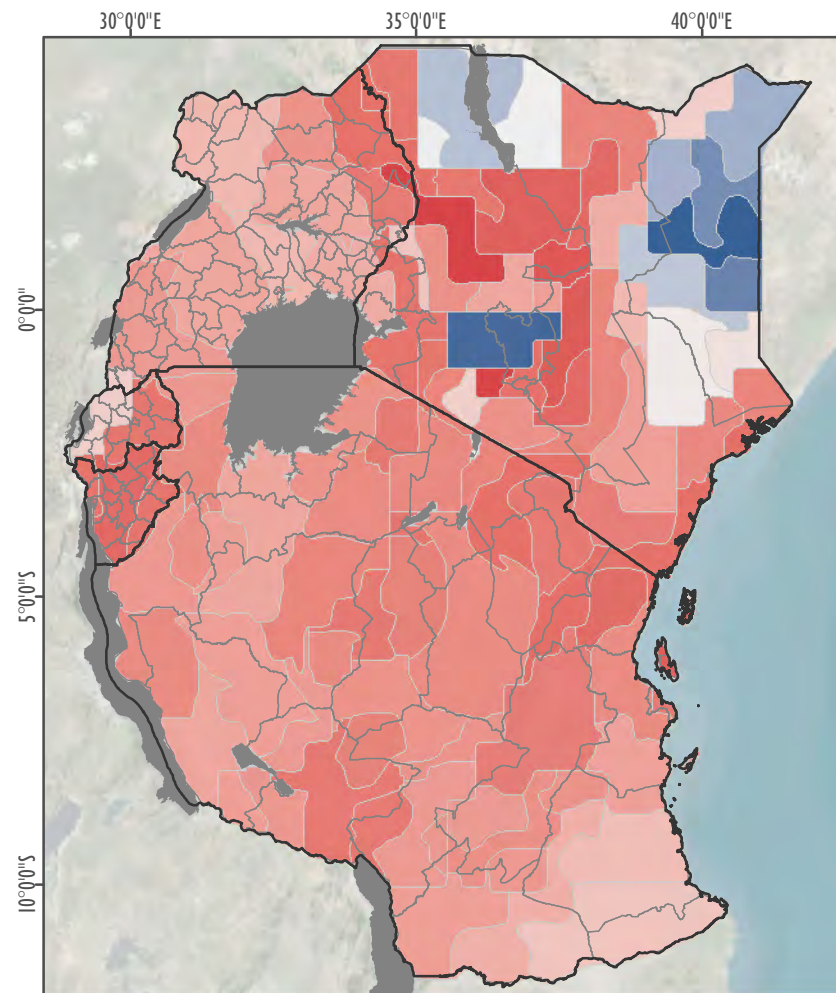
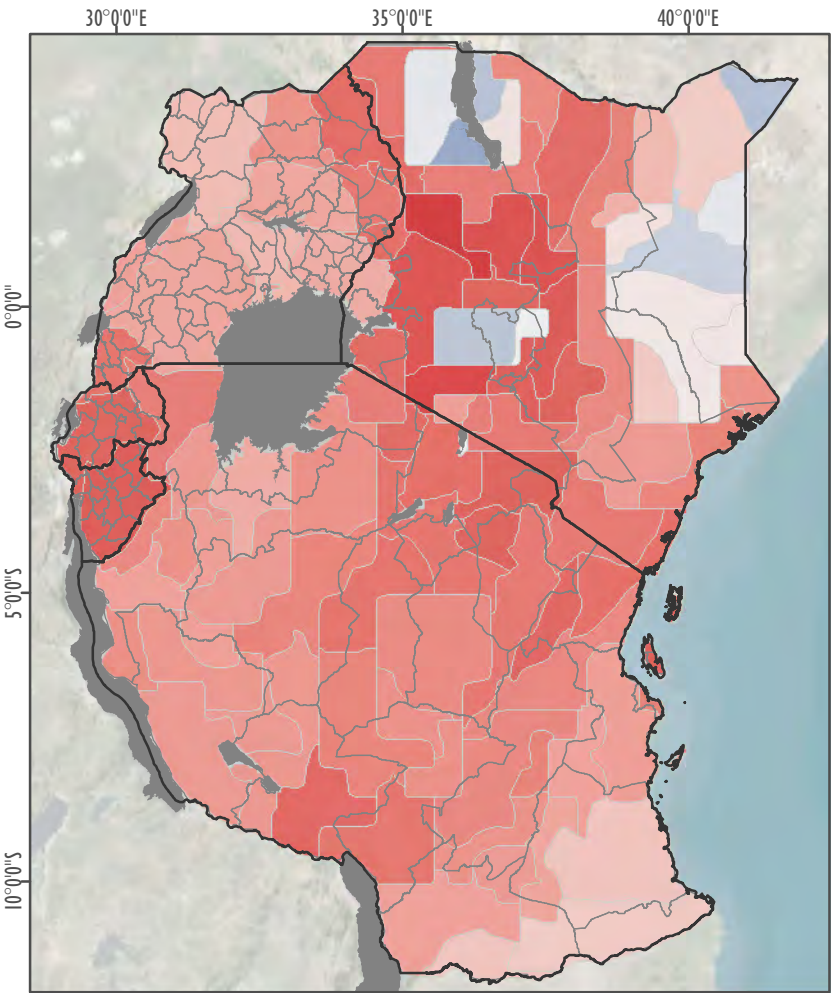
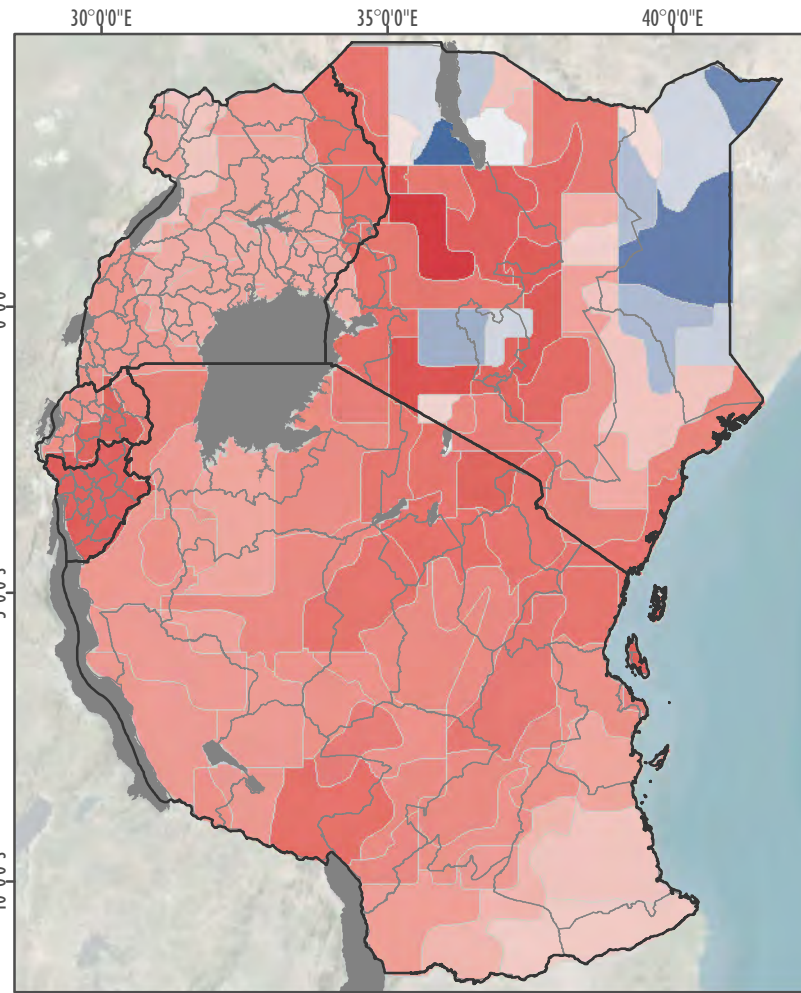
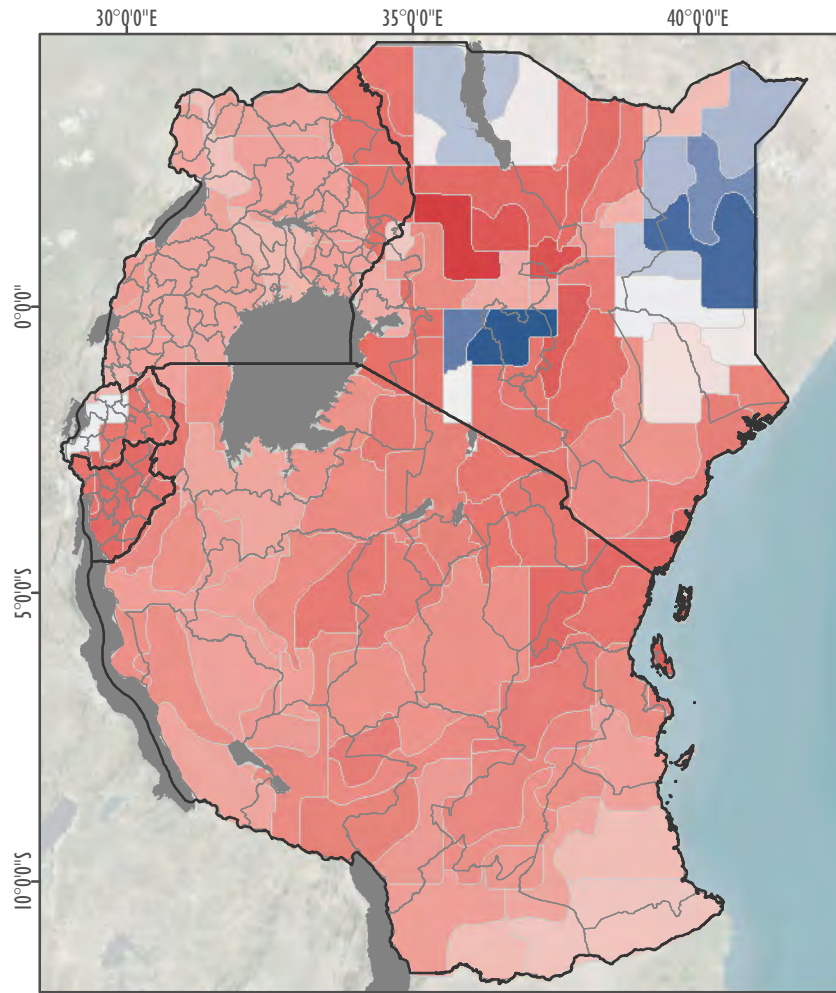
2006 - 2015

2046 - 2055

2086 - 2095

RCP4.5

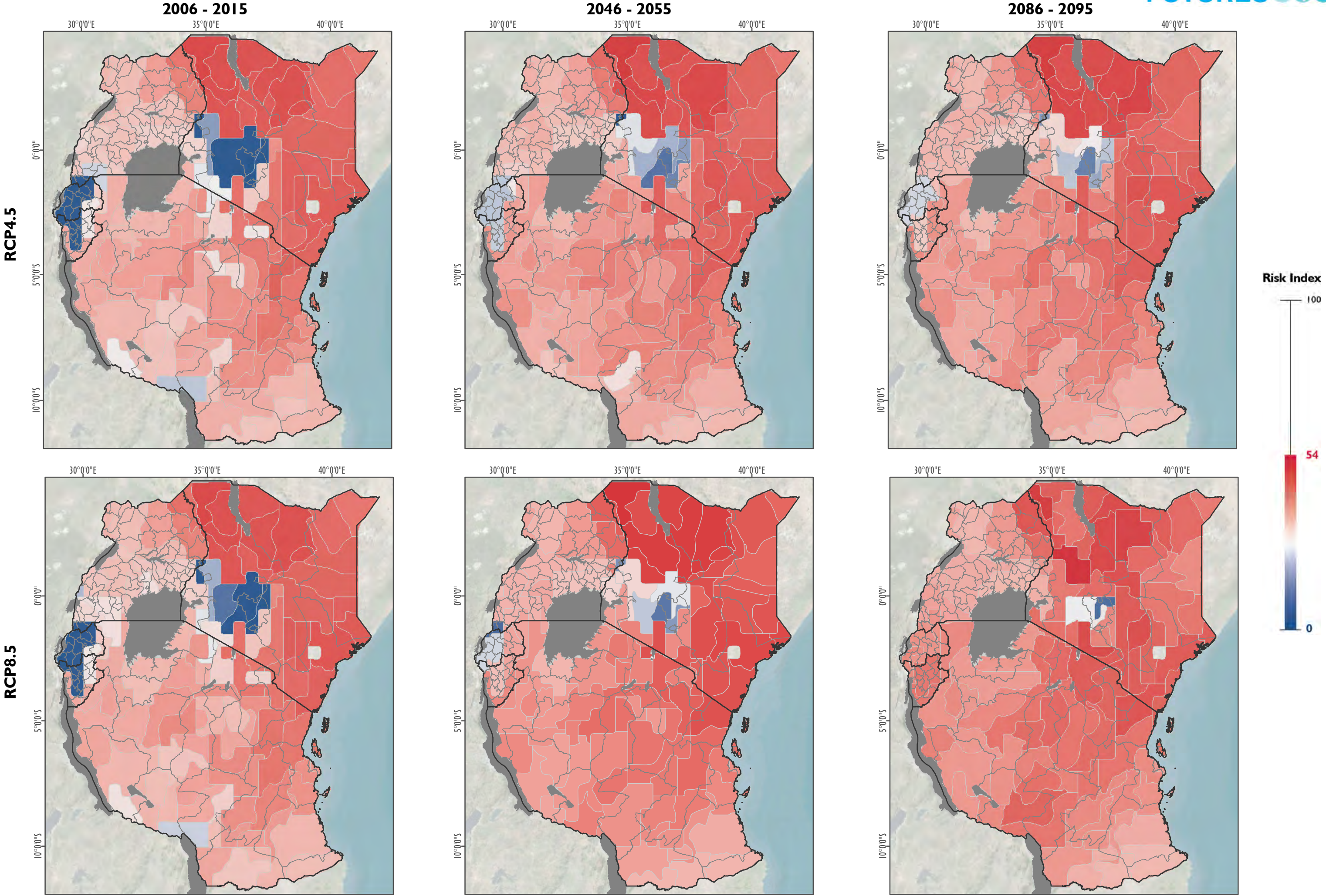
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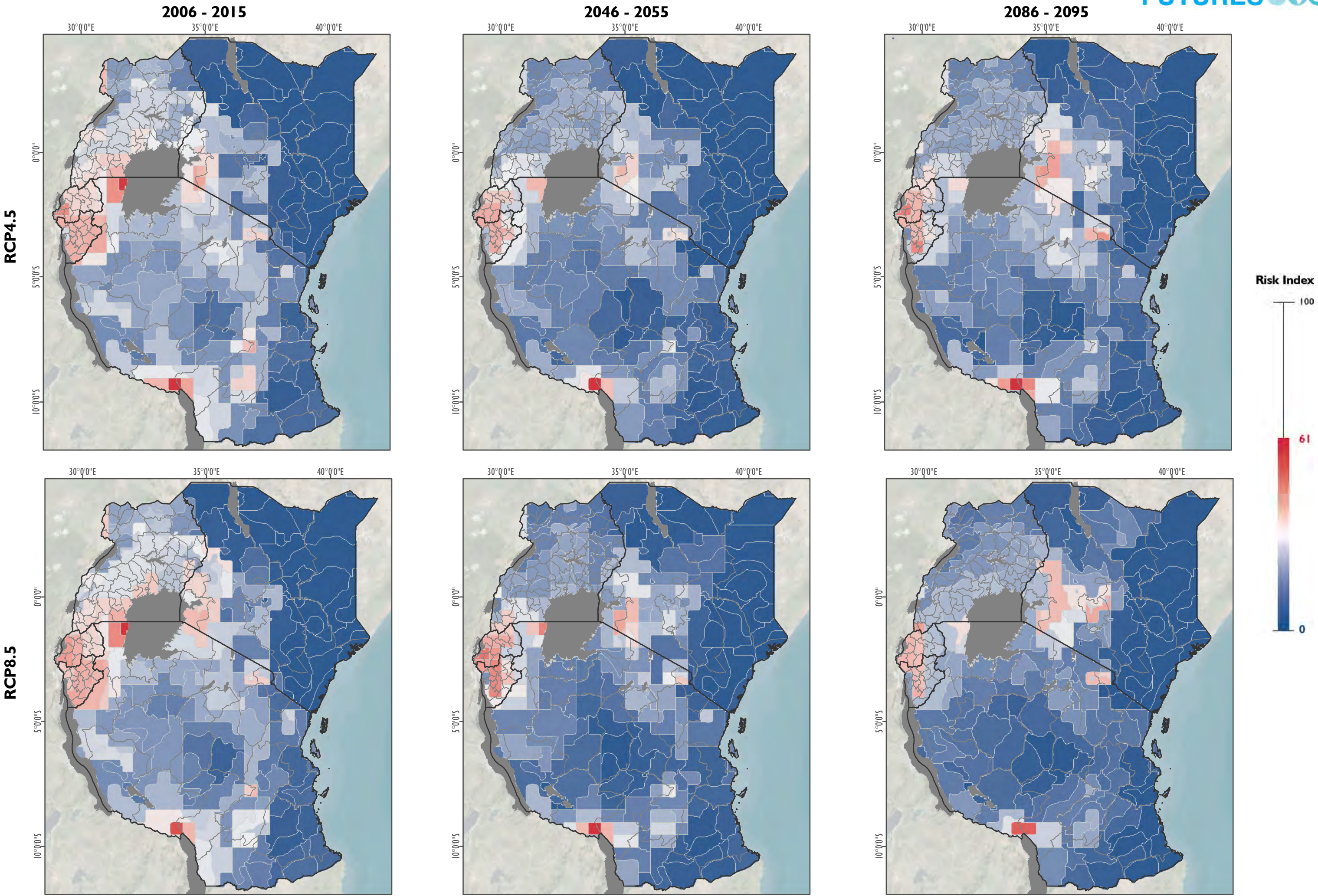
Risk Index



Present and Future Malaria Risk (VECTRI model)



Present and Future Rift Valley Fever Risk (Culex)



Present and Future Schistosomiasis Risk

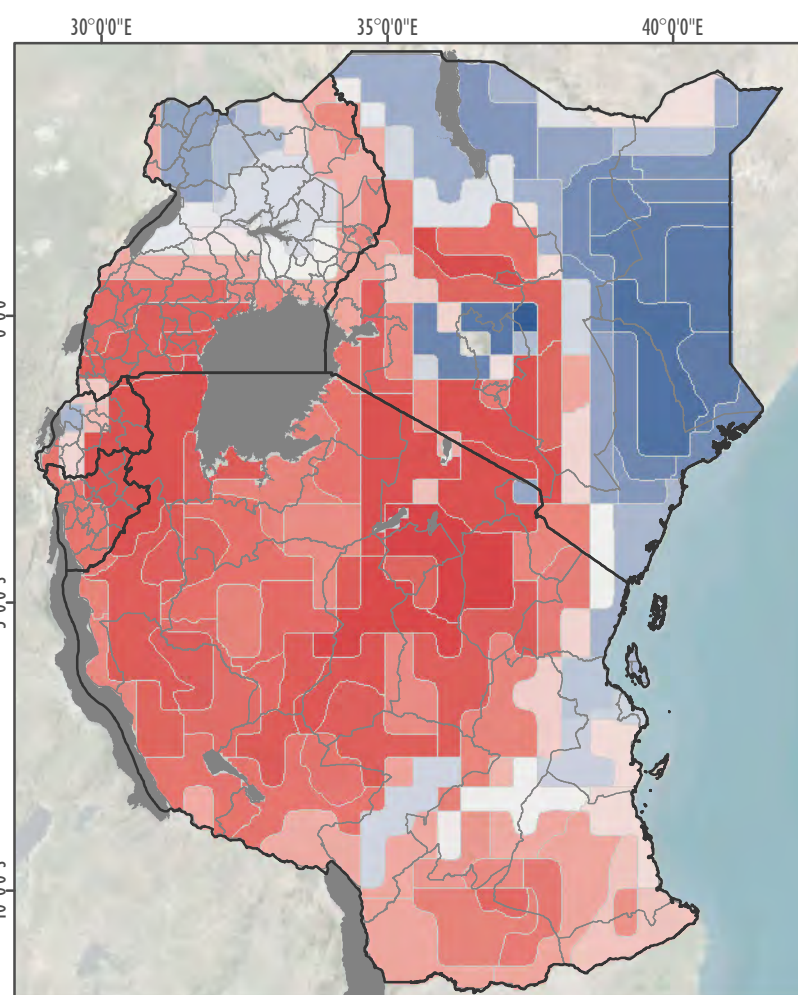
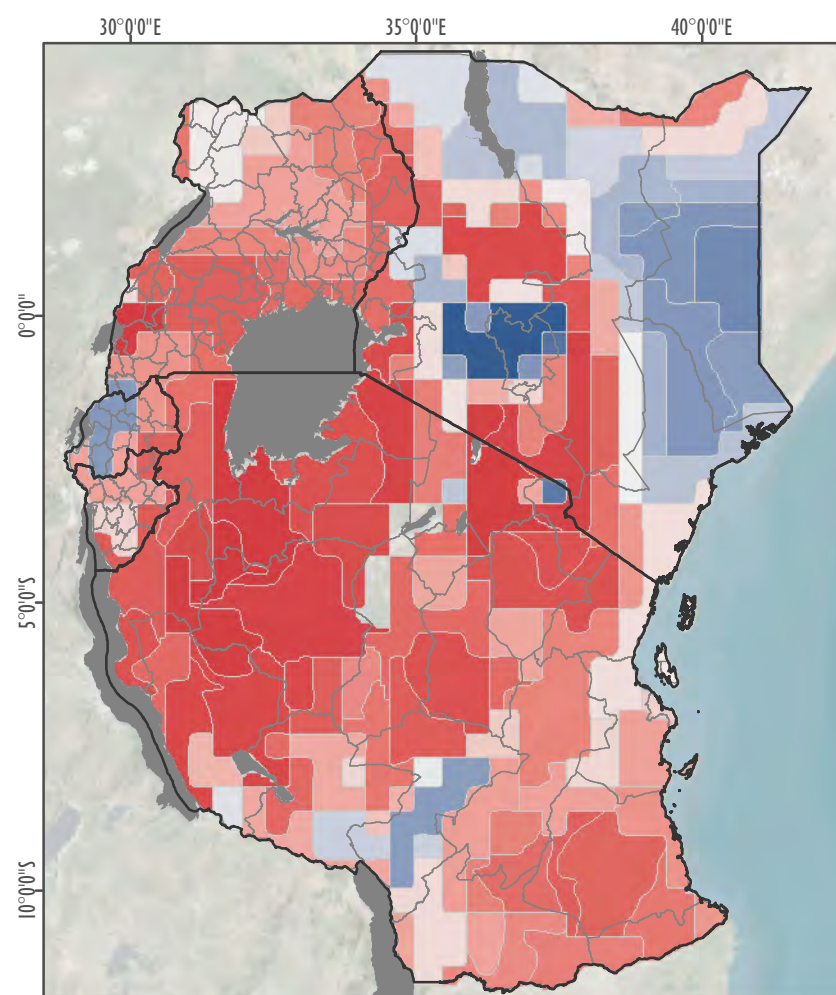
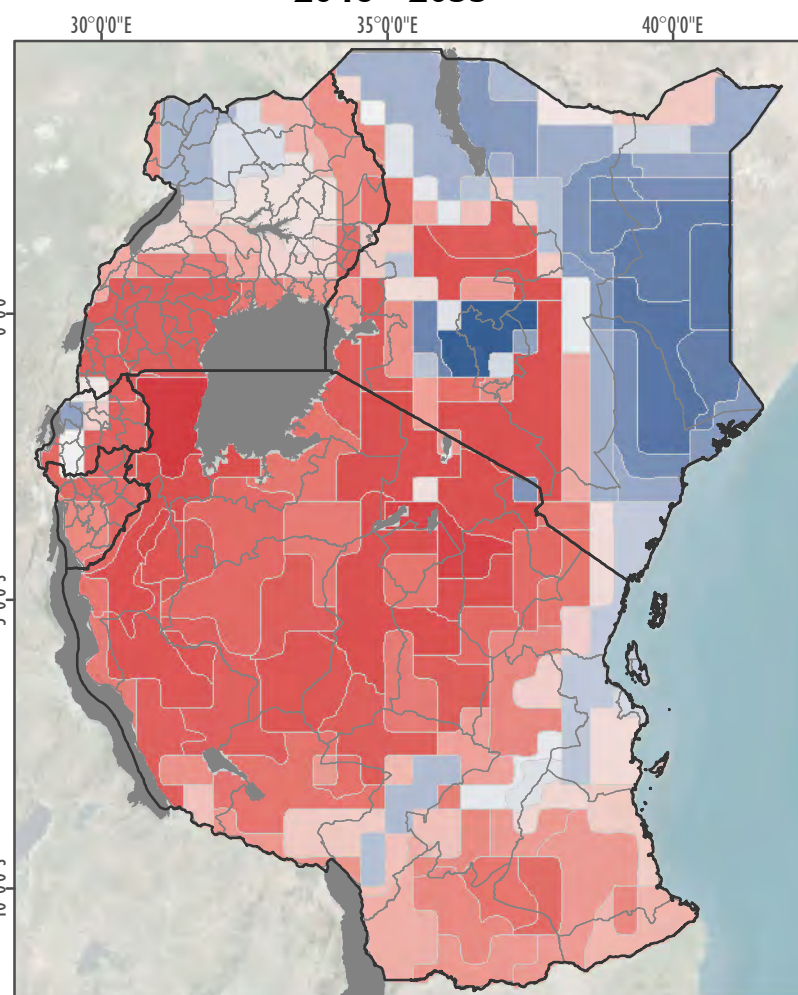
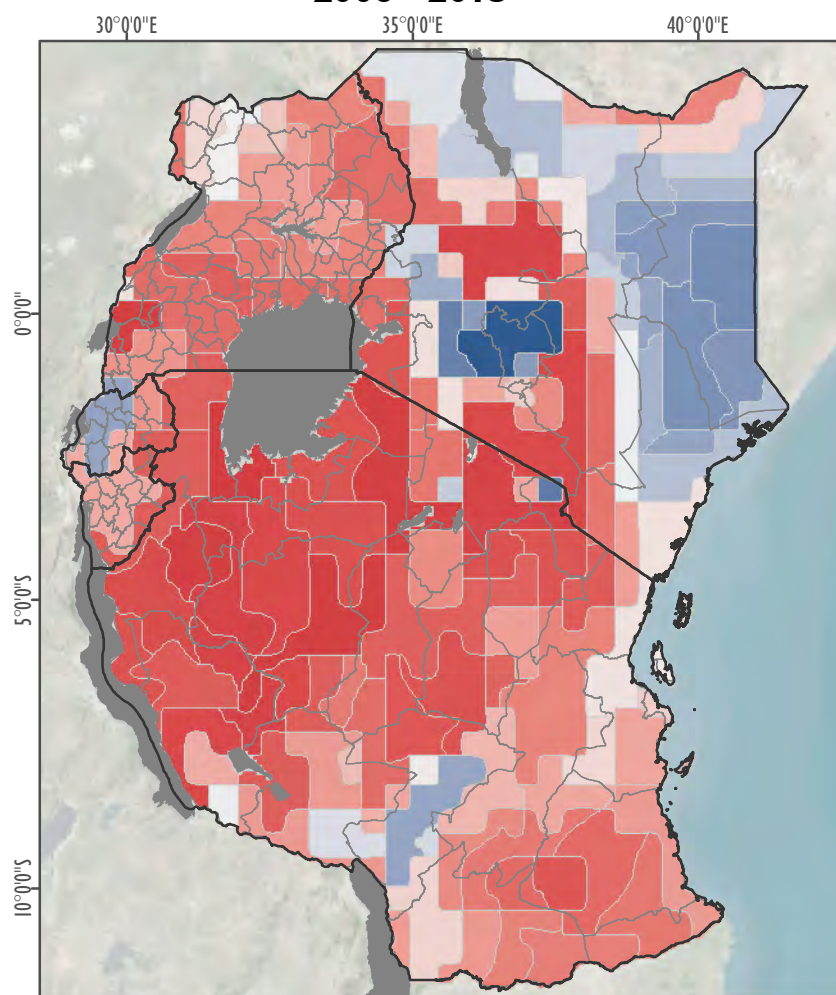
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RCP4.5

RCP8.5



Risk Index

