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Urbanization, malaria transmission and disease burden in Africa

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Abstract

Many attempts have been made to quantify Africa's malaria burden but none has addressed how urbanization will affect disease transmission and outcome, and therefore mortality and morbidity estimates. In 2003, 39% of Africa's 850 million people lived in urban settings; by 2030, 54% of Africans are expected to do so. We present the results of a series of entomological, parasitological and behavioural meta-analyses of studies that have investigated the effect of urbanization on malaria in Africa. We describe the effect of urbanization on both the impact of malaria transmission and the concomitant improvements in access to preventative and curative measures. Using these data, we have recalculated estimates of populations at risk of malaria and the resulting mortality. We find there were 1,068,505 malaria deaths in Africa in 2000 — a modest 6.7% reduction over previous iterations. The public-health implications of these findings and revised estimates are discussed.

We have become accustomed to the rapid growth of the human population and are no longer surprised to read that there was a fourfold increase in the size of the human population (from 1.65 billion to 6.1 billion) between 1900 and 2000. Eighty percent of this increase occurred after 1950 (REF. 1; FIG. 1a). It is perhaps less well known that at the start of the twenty-first century 2.9 billion people were living in urban areas, and that almost all of the 2.2 billion people estimated to be born between 2000 and 2030 will become urban residents. By 2008, it is predicted that the number of urban dwellers will exceed the rural population for the first time[2].

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SUPPLEMENTARY INFORMATION: See online article: S1 (table) | S2 (table) **Access to this links box is available online.**

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These population dynamics have significant public-health implications[3–5]. A shift in human populations from rural to urban environments will change global patterns of disease and mortality[6–8]. In rural areas of low-income countries morbidity and mortality are mainly due to infectious diseases, whereas in urban areas morbidity and mortality are generally caused by non-communicable diseases (for example, chronic, degenerative and cardiovascular diseases); however, the evolving HIV pandemic has begun to influence these patterns due to its higher prevalence in urban areas[9]. In Africa, the world's most rapidly urbanizing continent, this transition will be particularly acute (FIG. 1b). In 2003, 39% of 850 million Africans were living in urban areas, and this is projected to increase to 54% by 2030 (REF. 10).

Malaria and global public health

Natural transmission of malaria infection occurs by exposure to the bites of infective female anopheles mosquitoes[11]. The alternation between the human host and the mosquito vector represents the biological cycle of malaria transmission[12]. *Plasmodium falciparum* is the most common and clinically serious of the four malaria parasite species that infect humans and is found throughout the tropics and subtropics[13]. Climate, particularly temperature and rainfall, affects the ability of malaria parasites and anophelene vectors to coexist long enough to enable transmission. The result is a diversity of *P. falciparum* exposure across the world and the African continent[14,15]. This diversity presents a great challenge to those attempting to define disease burden, as the distribution of the risk and its correlation with the location of the human population needs to be quantified objectively[16]. Despite reducing the extent of global malaria distribution by almost 50% during the twentieth century, approximately 3 billion people (almost half the global population) inhabit areas where there is a risk of acquiring malaria infection[17]. This number is greater than at any time in history[17] due to the inexorable growth of the human population.

The World Health Organization (WHO) estimates that each year there are between 300 million and 500 million clinical attacks of malaria globally, resulting in more than 1 million deaths[18]. It is also assumed that about 85% of these deaths occur in Africa, mostly in young children[18]. In Africa, malaria is the main cause of mortality in children less than five years old (20%) and constitutes 10% of the overall disease burden[19]. It is responsible for approximately 40% of public-health expenditure, 30–50% of inpatient admissions and up to 50% of outpatient visits in areas with high rates of malaria transmission[19]. In addition to the morbidity and mortality that are directly attributed to *P. falciparum* (that we aim to better quantify here), there are other consequential and indirect effects on mortality that are linked to each step of the infection and disease processes[20]. Chronic, sub-clinical infections cause anaemia or can exacerbate undernutrition, which in turn can increase susceptibility to severe clinical outcomes of subsequent infection with *P. falciparum* or other pathogens. During pregnancy, asymptomatic infection of the placenta markedly reduces birth weights and infant survival rates. Patients who survive severe disease can be left with debilitating neurological sequelae.

Malaria not only poses a risk to survival, but the repeated clinical consequences of infection during early life place a burden on individual households, the health service and, ultimately, the economic development of communities and nations[21]. It has been argued that the persistence of endemic malaria in the tropics and sub-tropics significantly contributes to a perpetual state of depressed economic growth[22]. These economic arguments, in addition to humanitarian ones, provide clear support for the Roll Back Malaria (RBM) partnership (see the Online links box), which is a renewed effort launched by the WHO and which aims to halve malaria mortality rates by the year 2010 (REF 23). This goal has been conceived at a time when existing, affordable therapeutics are failing, health-service provision does not

keep pace with population growth, there is no immediate prospect of widespread vaccination and poverty continues to afflict most countries where malaria is endemic. Despite these challenges, malaria is a preventable infection and a curable disease. Effective intervention strategies are aimed at increasing access to insecticide-treated nets and prompt effective treatment, as well as providing intermittent treatment to women during pregnancy and to at-risk infants regardless of disease status. International initiatives, such as RBM, require a sound evidence base on which to prioritize allocation of the limited resources that are available for therapeutic intervention. Improving our estimates of morbidity and mortality in Africa by increasing our understanding of the impact of urbanization can be considered part of this wider objective.

We are not the first to be concerned with the link between urbanization and health, and there have been several reviews comparing the health status of urban and rural populations[7,24–27], including some that have explored the impact of urbanization on parasitic[28] and vector-borne[29,30] diseases such as malaria[31–35]. The specific purpose of this article, by contrast, is to quantify the consequences of urbanization on the malaria burden in Africa in 2000. We first describe evidence that shows African urban populations are healthier and have reduced malaria transmission rates compared with their rural counterparts. We then use objective criteria to define urban areas in Africa and a systematic meta-analysis of ANNUAL *P. FALCIPARUM* ENTOMOLOGICAL INOCULATION RATES (A*Pf*EIR) to examine in detail the entomological evidence for reduced malaria risks in urban environments. Finally, these A*Pf*EIR data are compared with simultaneously collected PARASITE PREVALENCE RATIO (PR) data — a more commonly used marker of malaria risk — to remodel the assumed malaria mortality burden in Africa in 2000.

A health status divide

Contrary to common perception, the improved health status of urban populations compared with rural populations in Africa has been observed by many studies. For example, infant mortality rates and childhood mortality rates are lower in urban populations compared with rural populations, as shown by 59 national demographic and health surveys conducted in sub-Saharan Africa between 1988 and 2002 (TABLE 1). These same surveys show that, compared with those living in rural areas, mothers and children living in urban communities have better nutritional status indicators; fewer morbid events; increased vaccine coverage; better physical access to health services; and greater use of insecticide-treated nets (ITN)[36–38]. These improved health indicators in urban communities reflect enhanced access to preventative and curative services that might be related to wealth[21,26,39,40], education[39,41] and/or simple physical access to services[42,43]. For the purposes of this appraisal on the continental scale we do not consider differences within urban areas, such as conditions that are associated with the relatively poorly studied ‘slum’ communities[44], but note that the demographic and health surveys were structured specifically to derive nationally representative samples of the populations surveyed (see Demographic and Health Surveys in the Online links box).

A malaria transmission divide

As a general rule, cities are unhealthy for the malaria parasite. The most extensive set of investigations on the effect of urbanization on malaria epidemiology was conducted by Trape *et al.* in Brazzaville, Congo, in the early 1980s (REFS 45–49). After a review of the demographical development of Brazzaville and previous malaria-related entomological and parasite surveys[46], a series of papers were published that describe how the inhabitants of Brazzaville were subject to reduced anopheline biting rates (0–7.36 versus 35–96 bites per person per night)[47]; reduced transmission intensities (an A*Pf*EIR of 22.5 versus 250 infected bites per person per annum (ib/p/a))[48]; reduced PR (0.351 versus 0.764)[49] and reduced

malaria-specific mortality rates (0.43 versus 12.9 per 1,000 people between 0–4 years of age)[45] compared with rural Congolese.

The above findings were corroborated in west Africa (for example, Benin[50], Burkina Faso[51–56], The Gambia[57], Ghana[58–61], Liberia[62,63], Niger[64] and Nigeria[65]); Central Africa (for example, Cameroon[66,67], Congo[45–49]. Democratic Republic of Congo[68,69] and Gabon[70]); eastern and Horn of Africa (for example, Ethiopia[71,72], Kenya[73], Sudan[74], Tanzania[73,75] and Uganda[73]); and southern Africa (for example, Namibia[76], Zimbabwe[77] and Zambia[78,79]). In the large and diverse continent of Africa, exceptions that prove the rule can be found — the low A*Pf*EIR in the rural fishing villages surrounding Cotonou, the capital of Benin[80], for example, is due to the confounding influence of the coast and lagoons, which favour *Anopheles melas*, a relatively inefficient malaria vector that is tolerant of brackish water.

In summary, there is clear evidence that urbanization affects anopheline species in the environment — diversity, numbers, survival rates, infection rates with *P. falciparum* and the frequency with which they bite people are all affected. So, fewer people acquire malaria infection, become ill and/or die of its consequences in urban areas. The most common explanation is lower vector densities that result from a paucity of clean freshwater breeding sites[57]. As has been eloquently detailed[48], however, the process of urbanization effects changes in indices of mosquito and malaria abundance not only by eliminating open spaces for breeding, but also by increasing pollution of the remaining breeding sites, thereby limiting the dispersion opportunities for adult mosquitoes. With increased human densities, malaria exposure per capita also decreases[48,81].

Overview of methods

The qualitative evidence described above strongly indicates that urban populations have access to better health, nutrition and services, and are at lower risk of malaria transmission than rural populations. To quantify these differences, however, it is necessary to determine where the urban and rural populations of Africa are located. We first describe a method to partition objectively the population of Africa into URBAN, PERI-URBAN and RURAL classes. This is achieved by investigating the population density that is associated with the largest urban areas in Africa and how it decreases with increasing distance from the urban centre. Once these population density groupings are defined they can be extrapolated to the whole continent with human population distribution maps. After such a map has been created it is then possible to overlay entomological survey data onto these population classes to examine the extent to which transmission (A*Pf*EIR) is reduced when moving from rural to urban population densities. This method has the advantage of avoiding any ambiguity in the definition of urban and rural.

As national registration systems for malaria are often inadequate, malaria burden estimates for Africa are generated by calculating the morbidity and mortality rates at intensively studied sites and associating these rates with malaria risk classes[82,83]. These risk classes and human population distribution have been mapped in Africa, so morbidity and mortality figures can be calculated across the wider continent. In addition, recent work has shown that these risk classes are linearly related to the PR[84]. To link these two approaches, we elaborate on previous work that has demonstrated a correlation between A*Pf*EIR and PR in a community[85]. We can therefore use the A*Pf*EIR data to quantify the impact of urbanization on transmission in Africa and its impact on PR and the malaria risk classes with which they are associated. Estimates of malaria morbidity and mortality for Africa in 2000 can then be adjusted for the effect of urbanization.

Defining urban and rural

There is little consensus among national governments and international agencies on the definition of an urban area or how to describe the process of urbanization[86,87]. Of the 228 countries for which the United Nations Population Division (UNPD) has data[2,86], 108 use administrative definitions (for example, living in a city), 51 use size and density (for example, the number of people per square kilometre), 39 use functional characteristics (for example, the amount of non-agricultural economic activity), 22 have no definition whatsoever and 8 define all or none of their populations as urban. This global diversity of urban definitions is reflected in Africa and is shown in the online supplementary information S1 (table). Large-area statistics on urbanization obviously depend on the way in which urban populations are categorized in space and how these categorizations have changed over time[87].

It is opportune therefore that recent advances in mapping urban areas make it easier to be objective and avoid inherent subjectivity in definitions[87,88]. Here, we use the global database of urban extents that was developed as part of the Global Rural–Urban Mapping Project (GRUMP)[89,90] by the Centre for International Earth Science Information Network (CIESIN), Columbia University, the International Food Policy Research Institute (IFPRI), the World Bank and the International Centre for Tropical Agriculture (CIAT). The GRUMP urban extent map was developed at 1 × 1 km spatial resolution using data on night-time lights (NTL)[91] and Landsat satellite sensor imagery[92], in combination with other geographical data (for example, Digital Chart of the World populated places[93], Tactical Pilotage Charts produced by the Australian Defence Imagery and Geo-Spatial Organization, and national census data)[89,90]. So far, it is the only product of its kind — although other maps of global urban extents are being constructed[94], as are investigations into their fidelity[88,95] (REF. 88; A.J.T., A. M. Noor and S.I.H., manuscript submitted).

Numerous studies have shown that the blooming effect of NTL imagery, where light from bright urban areas contaminates surrounding unilluminated rural areas, leads to an overestimation of urban extent[94,95,96]. Yet recent efforts to estimate the incidence of clinical attacks of malaria in urban Africa[35] have compounded this problem by multiplying NTL estimates by a factor of 2 to 3 as lower and upper bounds of their urban area estimates. This results in 1.7–2.6% of African land defined as urban. The GRUMP urban extents, which are acknowledged as overestimates[89,90], classify only 0.8% of Africa as urban. In a validation exercise in Kenya, GRUMP and NTL overestimated urban areas consistently (A.J.T., A. M. Noor and S.I.H., manuscript submitted). Owing to the preliminary nature of these urban surfaces, and the problems of blooming, we outline a more conservative approach that uses the population density of the largest URBAN AGGLOMERATIONS (UA) to define urban areas.

Africa had 37 UA with more than one million inhabitants in 2003 (REF. 10). On average, these UA had 2.7 million inhabitants and accounted for 10% of the total population and 25% of the urban population of their respective countries[10]. By identifying these UA on the GRUMP map[89,90] and overlaying their locations on the gridded population of the world version 3 (GPWv3) map[97] it is possible to determine the population-density ‘footprint’ of these UA (FIG. 2a). The population densities that are characteristic of urban, peri-urban and rural locations in Africa can therefore be identified across the continent using the GPWv3 map (FIG 2b). This process identifies 0.2% of the African landmass as urban, 1.1% as peri-urban, 3.9% rural 1 and 94.8% rural 2 (FIG. 2a,b). The equivalent percentages for the African population in 2000 are 18.7% urban, 17.0% peri-urban, 21.7% rural 1 and 42.6% rural 2. These surfaces provide new opportunities to examine the spatial epidemiology of malaria. Here, they are first used to revisit and quantify the impact of urbanization on the A*Pf*EIR data.

Urbanization and APfEIR

The first synthesis of A*Pf*EIR data found 159 spatially distinct records in Africa, post-1980, and determined a mean A*Pf*EIR estimate of 121 ib/p/a (the data had a range of 0–884) across the continent[98]. The study defined the A*Pf*EIR as *P. falciparum* infected bites per adult per night indoors, using human biting rates that were averaged over one year and standardized to human bait catch equivalents[99] on adults[100]. Considerable geographical heterogeneity was observed and there was a marked difference between areas of predominantly rural (146 ib/p/a; range 0–884) and urban (14 ib/p/a; range 0–43) land use. Further work revealed a mean A*Pf*EIR of 7.1 ib/p/a in city centres, 45.8 ib/p/a in peri-urban areas and 167.7 ib/p/a in rural areas, and showed that this influence of urbanization held in both 'dry savannah and desert' and 'wet savannah and forest' zones[34]. Both these analyses rely on subjective definitions of urban and rural by the authors of the original studies. In the current modelling exercises this problem was avoided by using population density associated with the largest UAs to define urban–rural partitions across the continent (FIG. 2a,b).

The information provided in the previous meta-analysis of A*Pf*EIR[98], together with 74 additional studies, is presented in the online supplementary information S2 (table). Identical search criteria (restricted to the peer-reviewed literature published between 2000 and 2004), data exclusion and geo-referencing procedures were used to identify new data sources[98]. To achieve temporally and spatially independent samples within a 1-km radius in areas with multiple surveys, we selected (in order of preference) the survey with simultaneously collected *P. falciparum* PR data in children (<15 years); the longest duration; or the most recent date (online supplementary information S2 (table)). There is uncertainty associated with the estimation of A*Pf*EIR, especially in areas with low transmission rates (REF. 102; Smith, D. L., Dushoff, R.W.S. and S.I.H., manuscript in preparation), although, as in our previous work, efforts were made to standardize A*Pf*EIR measurements between the different studies[98].

The above selection process resulted in temporally and spatially distinct A*Pf*EIR estimates (n = 233) from 22 countries across Africa between 1980 and 2004 (see online supplementary information S2 (table)). In areas with A*Pf*EIR surveys, the arithmetic mean is 112 ib/p/a with considerable spatial heterogeneity (range 0–1,030 ib/p/a). Using the population density criteria for urban, peri-urban, rural 1 and rural 2 areas (FIG. 2b), the average A*Pf*EIR in urban areas was 18.8 (± 4.6) ib/p/a, peri-urban 63.9 (± 20.0) ib/p/a, rural 1 111.4 (± 28.4) ib/p/a and rural 2 141.1 (± 16.5) ib/p/a (FIG. 3a).

The decrease in A*Pf*EIR with urbanization extent (FIG. 3a) was also shown to be true when the data were stratified by 'dry' and 'wet' zones (FIG. 3b), as has been observed elsewhere[34]. The differential between urban and rural was more marked in the wet areas because malaria endemicity, and therefore transmission intensity, reach greater values. The ambiguity of using ecozones[34] or author-defined definitions of land use from the original studies[98] was avoided by using the mean annual normalized difference vegetation index (NDVI)[102,103] characteristic of the site (FIG. 3b).

PR and APfEIR

The PR has been used as a marker of malaria endemicity owing to its widespread availability[73], although as a community prevalence measure it does not quantify infection rate like the A*Pf*EIR[75,104]. The form of the PR–A*Pf*EIR relationship and its geographical coherence has implications for the use of the PR in transmission risk mapping and malaria burden estimation. We were able to match contemporaneous PR survey data to 130 of the

A*Pf*EIR observations (online supplementary information S2 (table)) and investigate this relationship further.

Beier and co-workers have shown that the PR increases logarithmically with A*Pf*EIR (PR = 24.68 + (24.2 * $\log_{10}$ A*Pf*EIR); $r^2 = 0.71$; $n = 29$ excluding two outliers)[85]. With increased data we performed a similar analysis ($n = 130$ surveys; online supplementary information S2 (table)) and tested for SPATIAL DEPENDENCE, which was suspected to be a problem due to clustering of the sites of entomological surveys in Africa[98]. A strong linear correlation was again found between $\log_{10}$ A*Pf*EIR and PR (adjusted $r^2 = 0.63$, $P < 0.001$, $n = 121$) with no outliers removed[85], but zero values were excluded owing to the logarithmic transformation (FIG. 4). The residuals from this regression model were normally distributed and the VARIOGRAM revealed minimal spatial dependence (results not shown), so the assumption of spatial independence among the survey samples was justified and the resulting correlation robust. In addition, the log-linear relationship has now been supported as a good approximation to theoretical predictions in additional work (REF. 101; Smith, D. L., Dushoff, J., R.W.S. and S.I.H., manuscript in preparation). As the influence of urbanization on A*Pf*EIR is known reliably, its effects on PR can now be predicted.

Malaria infection risk and disease

There have been several attempts to examine the relationship between malaria infection rate and disease outcomes in Africa[20,105–110]. There continues to be some debate about whether functional immunity that is acquired from birth leads to a saturation of malaria mortality at increased intensities of parasite transmission[107–109,111,112]. There is agreement, however, that there are rapid increases in all-cause (FIG. 5a) and malaria-specific (FIG. 5b) fatal outcomes over small increases of transmission from marginal risk areas to those of acute seasonal transmission and stable endemic transmission[73,83].

We have therefore used data from carefully conducted, prospective studies of malaria mortality (FIG. 5) to define mortality rates that are associated with *P. falciparum* risk and extrapolated in accordance with populations living under different epidemiological risks[82,83] (FIG. 6). Applying endemicity-specific estimates of mortality rates to spatially congruent malaria risk and population distributions in Africa has become the benchmark approach to describing the malaria burden on Africa by the WHO[19,82] and the World Bank[83]. So far, however, none of these burden estimations have considered the effects of urbanization[83].

Remodelling malaria burden in Africa

Having quantified the reduction in A*Pf*EIR by urbanization (FIG. 3a), we can apply the reduction in A*Pf*EIR that is caused by urbanization to the A*Pf*EIR–PR relationship (FIG. 4) to establish the effect of urbanization on PR. Moreover, PR is linearly related to the FUZZY CLIMATE SUITABILITY (FCS) values that are derived from the MARA (Mapping malaria risk in Africa) malaria transmission climate suitability model[14] in Kenya[73].

Therefore, the influence of urbanization on FCS can be hypothesized (FIG. 7). The peri-urban and rural 1 classes do not affect FCS markedly because the midpoint endemicity values do not move between FCS classes (FIG. 7). The urban areas, however, do affect FCS values and, on average, lead to a reduction from class 4 to 3 (solid arrow, FIG. 7), from class 2 to 1 (solid arrow, FIG. 7), but not from class 3 to 2 (dashed arrow, FIG.7). These changes were converted into decision rules for the influence of urbanization on FCS class, and the population at risk and malaria mortality burden were recalculated. We applied the same methodology as has been used previously[83] with the more accurate population map, GPWv3 (REFS 97,114), to calculate the reduction in the numbers at risk in the urban populations residing in classes 1 and 4 (FIGS 6,7). The decision rules are implemented on a categorical

basis due to the inherent uncertainties in our ability to measure both parasite challenge (A*Pf*EIR) and the PR.

Most of the above reductions result in persons being moved to lower risk FCS classes, so the total population at risk decreases only 1.3% from 551,859,326 to 544,906,568 (TABLE 2). This translates to a percentage mortality reduction of 5.4% from 1,129,330 (interquartile range (IQR) 693,155, 1,583,232) to 1,068,505 (IQR 620,500, 1,416,947) when urbanization is considered in this study (TABLE 3). Most of this change is due to the 43,555,892 persons who move from stable endemic to acute seasonal risk. Although we have been conservative in our estimates of urban extent, the impact is significant and will increase as the population of Africa becomes increasingly urban.

Discussion

Effective targeting of limited resources for malaria control should be driven by an appreciation of need and based on a credible understanding of risk. The lack of an evidence-based platform to understand the comparative risks of infection and disease outcomes in relation to *P. falciparum* and urbanization in Africa has been partly addressed in this article. We have quantified the extent by which urbanization reduces transmission through an objective categorization of urban populations — a comprehensive meta-analysis of A*Pf*EIR data and have related this to PR markers of endemicity, thereby recalculating Africa's malaria burden in 2000. These estimates account for urbanization so that the total population at malaria risk has decreased by 2.2% and mortality by 6.7% compared with previous estimates[83]. The revised best estimate is for 1,068,505 (IQR 625,500, 1,416,947) malaria deaths in Africa in 2000 (TABLE 3).

We have attempted to dispel some 'urban myths' in relation to urbanization and malaria in Africa. Urbanization has marked entomological, parasitological and behavioural effects on malaria risks, which would in turn have profound consequences on the public-health burden. Perhaps one of the most striking empirical demonstrations of the temporal impact of urbanization on malaria burden is a reconstruction of a historical, clinical and demographic time-series for Nairobi, Kenya, over thirty years prior to independence (BOX 1).

Our evaluation of the impact of urbanization in Africa is markedly divergent from a recent study that suggests 6–28% of the entire global malaria incidence might occur in African urban populations[35,113]. Despite specific concerns about how the authors used NTL data to define the urban population of Africa (S.I.H. and A.J.T., manuscript submitted), and therefore derive their incidence estimates, arguments that are based solely on numbers ignore some obvious policy implications of these findings. Urban populations are on average subject to reduced levels of malaria transmission and severe disease than rural ones. They are also able to access better healthcare facilities and consequently suffer less morbidity and mortality from malaria and several other conditions. Urban populations do not therefore constitute the most biologically or economically vulnerable of populations in Africa. 'Pro-poor' policies that simultaneously target the greatest burden dictate that we would do better to identify, access and treat malaria infection and disease in rural populations.

Crucial to estimating disease burdens due to vector-borne pathogens is an understanding of their spatial patterns of risk in relation to population[15,17,19,82,83,114]. Much of the uncertainty in the current approaches to estimating the malaria burden in Africa concern the definition of urban extents. Thankfully, considerable international effort is now devoted to urban area cartography (REFS 87–89; A.J.T., A. M. Noor and S.I.H., manuscript submitted). Despite the predictable impact of urbanization on health, however, there has been no quantitative consideration of how the demographic transition will impact on future malaria burden (or a

range of other important infectious diseases), whereas there has been much speculation about the future impacts of climate change on these disease systems[115–119]. This is particularly intriguing as population growth in space[87] and time[120,121] is relatively more predictable than changes in climate. Our future work will be directed to making such projections. In addition, there has been no consideration of the effects of urbanization on the malaria burden outside of Africa. Although it is unlikely to be so straightforward, due to the well-documented urban tolerances of *Anopheles stephensi* in India[122] and *Anopheles claviger* in the Middle East[123,124], most non-African malaria-endemic countries report reduced malaria transmission in their major cities[18]. A further current challenge, therefore, is to extend these analyses to other continents.

In summary, it has been shown that the dual effects of behavioural changes and transmission reduction that are associated with urbanization make for profound decreases in morbidity and mortality from malaria in Africa. The construction of global maps of urban extents provides scientists and policy makers with new opportunities to quantify and perhaps predict the coincidental transitions in population and disease in low-income nations over time.

Online Links

FURTHER INFORMATION

Simon I. Hay's laboratory: http://users.ox.ac.uk/~hay/

Demographic and Health Surveys: http://www.measuredhs.com/

Roll Back Malaria: http://www.rbm.who.int/

World Population Prospects database: http://esa.un.org/unpp

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Glossary

ANNUAL *P. FALCIPARUM* ENTOMOLOGICAL INOCULATION RATE	The number of *P. falciparum* positive mosquito bites per person per year.
FUZZY CLIMATE SUITABILITY	The suitability of local climate to support *P. falciparum* malarial transmission in an average year. It is represented as a value between 1 and 0, where 1 is completely suitable and 0 completely unsuitable.

PARASITE PREVALENCE RATIO	The proportion of a sampled population who have *P. falciparum* in their blood.
PERI-URBAN	Defined here for Africa as locations with 250–1,000 persons per km^2.
RURAL	Defined here for Africa as locations with fewer than 250 persons per km^2. This is further subdivided into rural 1 (100–250 persons per km^2) and rural 2 (<100 persons per km^2).
SPATIAL DEPENDENCE	The tendency for observations close in space to be more highly correlated than those further apart.
URBAN	Defined here for Africa as locations with more than 1,000 persons per km^2.
URBAN AGGLOMERATION	An area with population within the contours of contiguous territory inhabited at urban levels of residential density without regard to administrative boundaries of 1 million inhabitants or more in 2003.
VARIOGRAM	A function used to represent spatial dependence.

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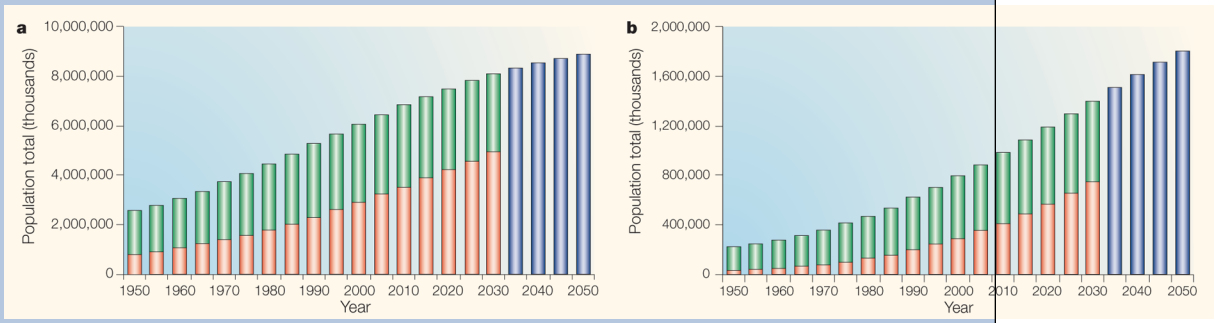
Box 1

Urbanization in time: a Nairobi case study



Nairobi provides an example of the influence of urbanization on malaria in a large African urban agglomeration. Urban patterns of transmission reduction, improved human access to services and protection measures are reflected over time during the establishment of Nairobi as Kenya's capital city. We focus on the 1930–1964 time interval, when data were available from the annual medical reports of Nairobi municipality[125]; see the figure, which shows locally notified cases of malaria (the line and the left axis show total malaria cases and the bar and the right axis show total population). The population increased from ~50 thousand to ~350 thousand over this period and reached 2.6 million in 1999 (REF. 126). Locally notified malaria cases show a collapse of autochthonous malaria transmission from an average of 1,182 cases in the 1930s to 317 cases in the 1940s, 250 in the 1950s and, finally, 49 cases in the 1960s. Population growth, urbanization and the collapse in malaria transmission are therefore inextricably linked. We provide these data for historical interest and realize that many factors will have combined to generate these trends.

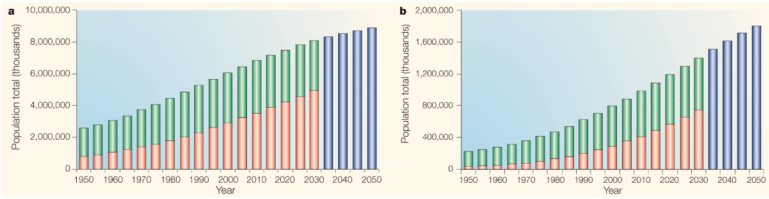


Figure 1. Population totals from 1950–2050

The bar plots are stratified by urban (red) and rural (green) for the world (**a**) and Africa (**b**). Note that urban and rural population projections stop in 2030, after which total population is shown (blue). Data from the World Population Prospects Database (see the Online links box).

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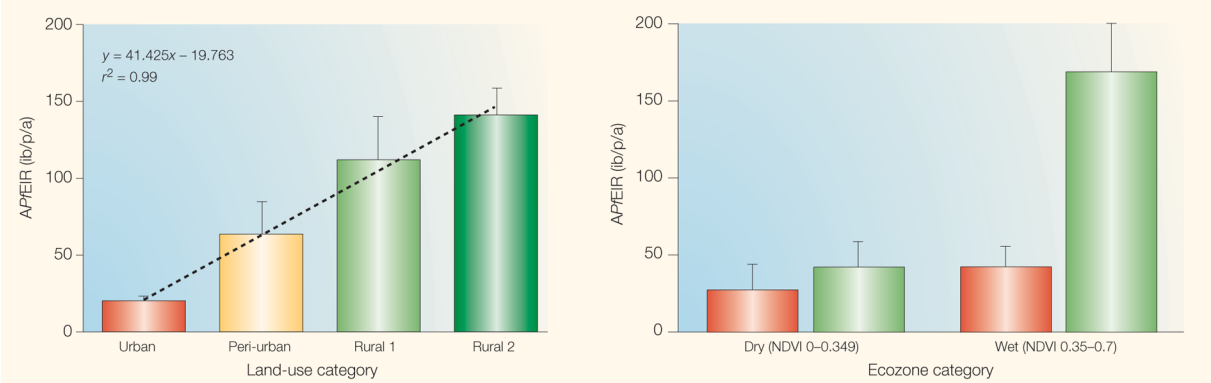

Figure 2. Mean population density (persons per km^2) in 2000 of the 37 urban agglomerations (UA) in Africa

a | The bar plot shows the mean population density in 2000 of the 37 UA with more than one million inhabitants in 2003 at successive 5 km buffers from the UA edge. The Global Rural–Urban Mapping Project (GRUMP) urban mask[89,90] defined the spatial extent of the 37 UA. Overlaying this on the gridded population of the world version 3 (GPWv3)[97] allowed the population associated with these classes to be shown in the bar plot: urban (red), peri-urban (yellow), rural 1 (light green) and rural 2 (dark green, off the scale). The core UA (defined by its GRUMP extent) has a population density >1,000 persons per km^2; peri-urban areas (defined as between 5–15 km from the UA edge) have population densities 250–1,000 persons per km^2 and rural areas (defined as >20 km from UA edge) have densities of <250 persons per km^2. To prevent this rural stratification being biased towards high population densities (as we conservatively trained population density on the largest African UAs), the rural class was further divided into rural 1 (100–250 persons per km^2 at 20–55 km from UA edge) and rural 2 (<100 persons per km^2 at >55 km from the UA edge). The vertical lines indicate the standard errors. **b** | The map shows these population densities plotted across Africa using GPWv3 (REF. 97). The map is exaggerated vertically to help resolve small-area urban and peri-urban classes across Africa.

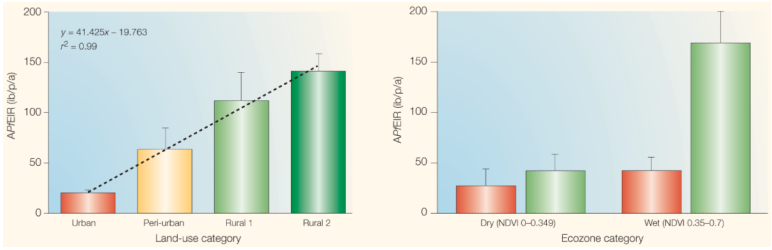

Figure 3. Bar plots of A*Pf*EIR by land-use category (a) and APfEIR by dry or wet ecozone (b)
The red bars in part **b** are pooled urban and peri-urban data and the green bars are pooled rural 1 and rural 2 data. Dry and wet areas were defined by satellite sensor measurements of <0.35 and >0.35 normalized difference vegetation index (NDVI)[127,128], respectively. The NDVI is a measure of photosynthetic activity or 'greenness' of a region[127,128] and is often used as a surrogate for moisture availability. In both plots the solid vertical lines indicate the standard error. A*Pf*EIR, annual *P. falciparum* entomological inoculation rates; ib/p/a, infected bites per person per annum; NDVI, normalized difference vegetation index.

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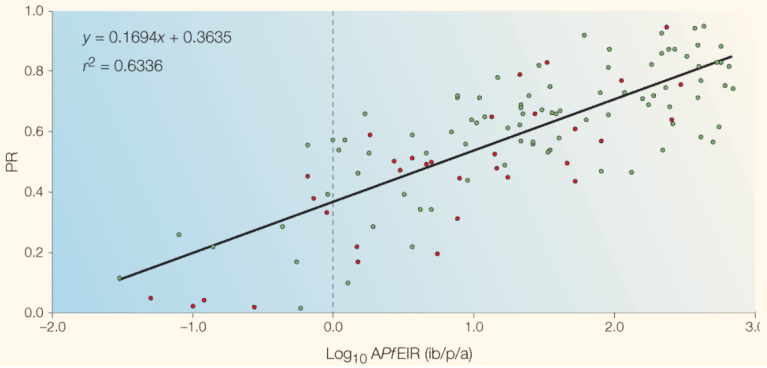

Figure 4. Plot of PR by $\log_{10}$ A*Pf*EIR

The red dots are pooled urban and peri-urban data ($n = 33$) and the green dots are pooled rural 1 and rural 2 data ($n = 88$). The fit line is drawn for all data. A*Pf*EIR, annual *P. falciparum* entomological inoculation rates; ib/p/a, infected bites per person per annum; PR, parasite prevalence ratio.

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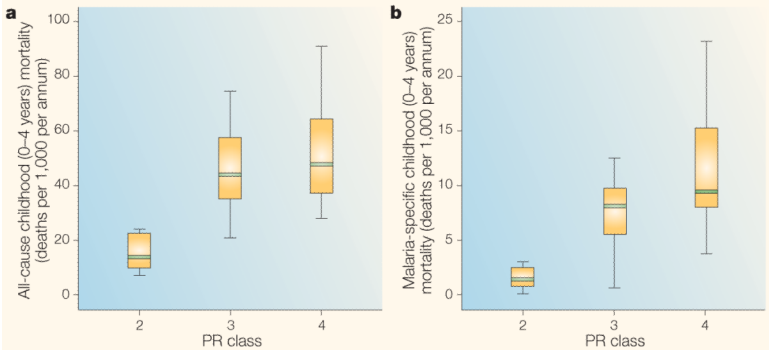

Figure 5. Childhood mortality in Africa

a | A box-and-whisker plot of all-cause childhood mortality in children (0–4 years of age), shown as deaths per 1,000 children per annum. **b** | A box-and-whisker plot of malaria-specific mortality in children (0–4 years of age), shown as deaths per 1,000 children per annum. The median (thick central line) is shown with the 25% and 75% percentile ranges (box depth) and the maximum and minimum (T-bars). These data are plotted against categorical classes of the PR (class 2, marginal risk areas, PR 0–0.25, $n = 8$; class 3, acute seasonal transmission PR 0.25–0.75, $n = 24$; class 4, stable endemic transmission, PR >0.75, $n = 15$)[73,83] (see FIG. 6). PR, parasite prevalence ratio.

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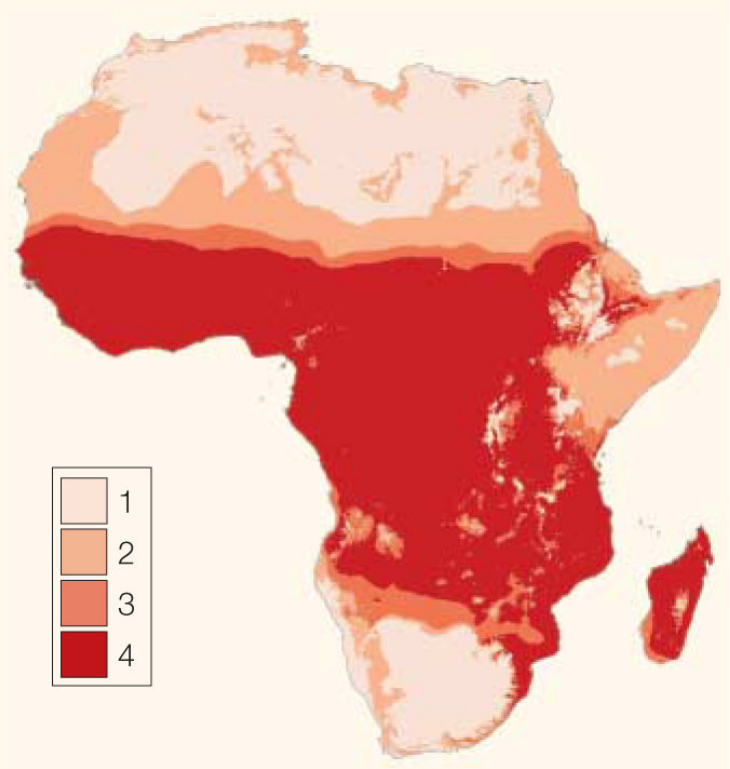

Figure 6. The MARA model[14] of fuzzy climate suitability (FCS) for *P. falciparum* malaria transmission

FCS values vary between zero (totally unsuitable) to 1 (totally suitable) in an average year. The data are grouped into class 1 zero risk (FCS = 0), class 2 marginal risk (FCS 0–0.25), class 3 acute seasonal transmission (FCS 0.25–0.75) and class 4 stable endemic transmission (FCS >0.75)[73,83].

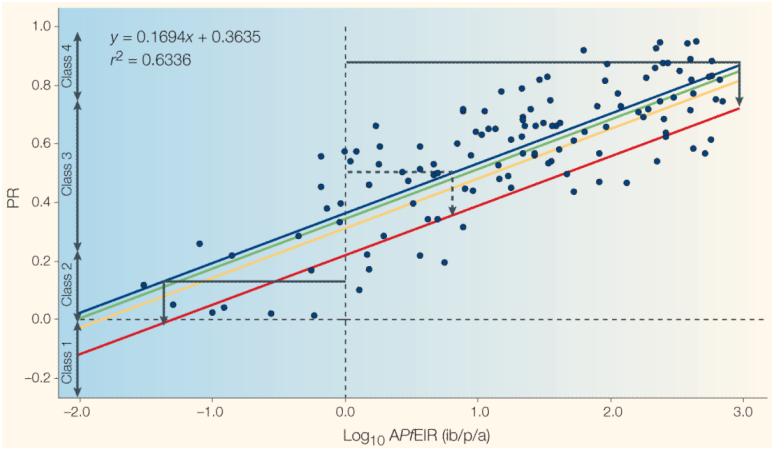

Figure 7. Plot of PR by $\log_{10}$ A*Pf*EIR

The blue circles and blue fit-line are the original data (see FIG. 4). The red (urban), yellow (peri-urban) and green (rural 1) fit-lines show the change in the relationship between A*Pf*EIR and PR predicted by the decrease A*Pf*EIR with urban intensity outlined in FIG. 3a. Class 1–4 emphasize the categorical separation of PR across Africa (FIG. 6). The solid arrows show levels of urbanization that result in movement between classes and dashed arrows those that do not. These categorical changes are then used in the population at risk estimates to calibrate burden estimates (TABLE 2). A*Pf*EIR, annual *P. falciparum* entomological inoculation rates; ib/p/a, infected bites/person/annum; PR, parasite prevalence ratio.

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Table 1

Urban–rural differentials in selected health indicators in sub-Saharan Africa

Health indicator	Urban environment			Rural environment		
	25%	Median	75%	25%	Median	75%
Infant mortality rates: deaths per 1,000 in >1 year of age cohort in the 10 years before survey; *n* = 59, 1988–2002	60.0	74.7	84.6	78.9	96.8	115.3
Childhood mortality rates: deaths per 1,000 in 1–4 year age cohort in the 10 years before survey; *n* = 59, 1988–2002	41.8	57.3	72.5	60.3	83.1	104.4
Nutritional status: maternal body mass index; *n* = 40, 1992–2002	22.5	22.9	23.5	21.7	21.4	20.9
Morbid events: percentage children (<3 years) ill with fever during the two weeks preceding the survey; *n* = 54; 1988–2002	29.9	35.1	39.0	32.7	41.6	47.8
Vaccine coverage: percentage children (12–23 months) who received bacille Calmette–Guérin, measles, three doses of diphtheria–pertussis–tetanus and polio vaccines by the time of the survey; *n* = 58; 1988–2002	46.5	57.9	72.0	23.2	38.5	60.0
Physical access to health services: average distance to a health facility in km; *n* = 6; 1999–2002	14.5	16	18.3	44.8	47.6	49.8
Use of insecticide-treated nets (ITN): percentage of households with ITN; *n* = 7; 1999–2002	30.9	32.9	41.9	7.8	10.1	29.2

For each indicator the number of demographic and health surveys and date range are given. The median and interquartile range (shown as 25 and 75 percentiles) are given. All data for sub-Saharan Africa from national Demographic and Health Surveys (see the Online links box), accessed 1 September 2004.

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Table 2

Populations at malaria risk during 2000 with and without urban corrections

Age	N. Africa exclusion	SA at no malaria risk (1–3)	SA at malaria risk (4)	ROA at no malaria risk (1)	ROA at low stable/epidemic risk (2–3)	ROA at stable endemic risk (4)	Total at malaria risk
0–4 years							
Original	16,370,666	5,037,177	2,194,347	7,109,982	21,861,405	72,303,528	96,359,280
Urban corr.	16,370,667	5,424,679	1,879,895	7,825,147	28,684,533	64,765,236	95,329,664
% change	0.00	7.69	−14.33	10.06	31.21	−10.43	−1.07
5–14 year							
Original	33,576,109	10,105,604	4,278,645	11,346,503	34,431,168	113,699,656	152,409,469
Urban corr.	33,576,110	10,878,074	3,656,880	12,558,596	45,284,467	101,634,264	150,575,611
% change	0.00	7.64	−14.53	10.68	31.52	−10.61	−1.20
≥ 15 years							
Original	93,187,646	27,180,408	10,091,855	22,052,882	68,166,114	224,832,608	303,090,577
Urban corr.	93,187,640	29,135,719	8,570,892	24,621,186	89,550,002	200,880,400	299,001,294
% change	0.00	7.19	−15.07	11.65	31.37	−10.65	−1.35
Total							
Original	143,134,421	42,323,189	16,564,847	40,509,367	124,458,687	410,835,792	551,859,326
Urban corr.	143,134,416	45,438,472	14,107,666	45,004,929	163,519,002	367,279,900	544,906,568
% change	0.00	7.36	−14.83	11.10	31.38	−10.60	−1.26

corr., corrected; ROA, rest of Africa; SA, southern Africa. Numbers in brackets denote the fuzzy climate suitability class (FIG.6).

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Table 3

Urban corrected estimates of malaria-specific deaths

Age	SA at malaria risk (4)	ROA at low stable/ epidemic risk (2–3)	ROA at stable endemic risk (4)	Total at malaria risk
0–4 years				
25%	150	–	477,967	478,118
Median	244	75,153	604,260	679,658
75%	395	–	943,629	944,024
5–14 years				
25%	293	–	67,079	67,371
Median	475	42,567	160,582	203,625
75%	768	–	281,527	282,295
≥ 15 years				
25%	686	–	74,326	75,011
Median	1,114	63,581	120,528	185,223
75%	1,800	–	188,828	190,627
Total				
25%	1,129	–	619,372	620,500
Median	1,834	181,301	885,370	1,068,505
75%	2,963	–	1,413,984	1,416,947

Median and interquartile range (IQR) shown as the 25 and 75 percentile using the IQR of mortality estimates (TABLE 1). ROA, rest of Africa; SA, southern Africa. Numbers in brackets denote the fuzzy climate suitability class (FIG.6).