LETTER

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The prevalence of *Plasmodium falciparum* in sub-Saharan Africa since 1900

Robert W. Snow[1,2], Benn Sartorius[3], David Kyalo[1], Joseph Maina[1], Punam Amratia[1], Clara W. Mundia[1], Philip Bejon[1,2] & Abdisalan M. Noor[1]

Malaria transmission is influenced by climate, land use and deliberate interventions. Recent declines have been observed in malaria transmission. Here we show that the African continent has witnessed a long-term decline in the prevalence of *Plasmodium falciparum* from 40% prevalence in the period 1900–1929 to 24% prevalence in the period 2010–2015, a trend that has been interrupted by periods of rapidly increasing or decreasing transmission. The cycles and trend over the past 115 years are inconsistent with explanations in terms of climate or deliberate intervention alone. Previous global initiatives have had minor impacts on malaria transmission, and a historically unprecedented decline has been observed since 2000. However, there has been little change in the high transmission belt that covers large parts of West and Central Africa. Previous efforts to model the changing patterns of *P. falciparum* transmission intensity in Africa have been limited to the past 15 years[1,2] or have used maps drawn from historical expert opinions[3]. We provide quantitative data, from 50,424 surveys at 36,966 geocoded locations, that covers 115 years of malaria history in sub-Saharan Africa; inferring from these data to future trends, we would expect continued reductions in malaria transmission, punctuated with resurgences.

Although short-term seasonal cycles are fundamental to malaria epidemiology, longer-term climate anomalies and shifting environmental and intervention landscapes also alter the likelihood of contact between mosquitoes and humans or the duration of host infection. The supra-seasonal, long-term cycles of transmission are poorly defined for *P. falciparum* malaria in Africa.

To provide an empirical basis for defining the long-term nature of malaria transmission cycles, we used data on the *P. falciparum* parasite rate (the proportion of persons positive for malaria infection among those examined). These data were derived from a repository assembled over the past 21 years (see Supplementary Information 1). To our knowledge, these data (available through the Harvard Dataverse, http://dx.doi.org/10.7910/DVN/Z29FR0) represent the largest repository assembled for any parasitic disease in Africa, derived from over 50,000 community-based surveys across sub-Saharan Africa since 1900[4] (Extended Data Figs 1, 2, Supplementary Information 1). We have used the space–time cube of data to leverage power from neighbouring areas and preceding data points in time[5] in a conditional autoregressive spatial and temporal model, in order to compute a smoothed median estimate for approximately five-year intervals (since 1900) across 520 sub-national administrative polygons (Extended Data Fig. 3) and within the changing limits of *P. falciparum* transmission (Fig. 1).

The median posterior predictions of *P. falciparum* prevalence provide a summary of several major cycles in the history of malaria transmission across the continent (Fig. 2). The impact of interventions and/or climate on these cycles can be assessed only by temporal plausibility rather than quantitative analysis. Between 1900 and 1944, efforts to control malaria focused on areas of European economic influence, primarily by targeting vector larvae or through mass quinine administration campaigns targeting the parasite itself[6]; we did not observe declines in transmission associated with these efforts.

We observed two precipitous declines in infection prevalence, both of which followed rises in prevalence. One of these declines occurred between 1945 and 1949 and another between 2005 and 2009: dichlorodiphenyltrichloroethane (DDT) and chloroquine were introduced between 1945 and 1949, and the widespread introduction of insecticide-treated bed nets and artemisinin-based combination therapy occurred between 2005 and 2009 (Fig. 2). Indoor residual house-spraying with DDT was introduced through comparatively small projects during the 1950s and expanded in the 1960s only in southern Africa, Ethiopia, Sudan, Somalia and Madagascar. After successful trial projects, the expansion of the use of insecticide-treated bed nets to national scales took over a decade, reaching moderate levels of coverage in Africa by 2010[7].

The period from 1960 to 1984 was characterized by a slow decline in malaria prevalence across Africa (Fig. 2). Counter-intuitively, this coincided with a cessation of malaria elimination ambitions across much of sub-Saharan Africa[8], with an emerging resistance among mosquitoes to organochloride insecticides[9] and with a period in which malaria was integrated into broader health agendas that focused on the presumptive treatment of fevers with choroquine, a cheap, widely available and efficacious drug. This interval also included drought periods across much of the Sahel[10] (Fig. 2), which rendered some areas unsuitable for malaria transmission[11]. Therefore, although several interventions may have influenced the observed trends, no single factor appears sufficient to account for them all.

Between 1985 and 2004, however, median malaria prevalence rose to levels similar to those witnessed fifty years earlier, before the introduction of DDT (Fig. 2). This period also saw a rapid expansion of chloroquine resistance across Africa[12], climate anomalies connected with changes in Pacific Ocean sea surface temperatures[10] (Fig. 2), and the failure of many national health agencies to prioritize the growing malaria epidemic because of a lack of international donor assistance[13]. Despite impressive gains in the coverage of effective interventions since 2005, the rate of reduction of malaria prevalence has slowed during the interval 2010–2015 (Fig. 2). Continued challenges to malaria control include difficulties in ensuring access to artemisinin-based combination therapies, the threat of drug resistance, rapidly emerging insecticide resistance and inadequate funding plans for replacing long-lasting insecticide-treated nets[14].

There has been an overall decline in malaria transmission intensity over the past 115 years. Independent abiotic factors related to economic growth may have contributed to this overall decline, but the constant growth in sub-Saharan African GDP, urbanization and/or female education charted by the World Bank (World Development Indicators,

[1]Kenya Medical Research Institute-Wellcome Trust Collaborative Programme, Nairobi, Kenya. [2]Centre for Tropical Medicine and Global Health, Nuffield Department of Clinical Medicine, University of Oxford, Oxford, UK. [3]Public Health Medicine, School of Nursing and Public Health, University of KwaZulu-Natal, Durban, South Africa.

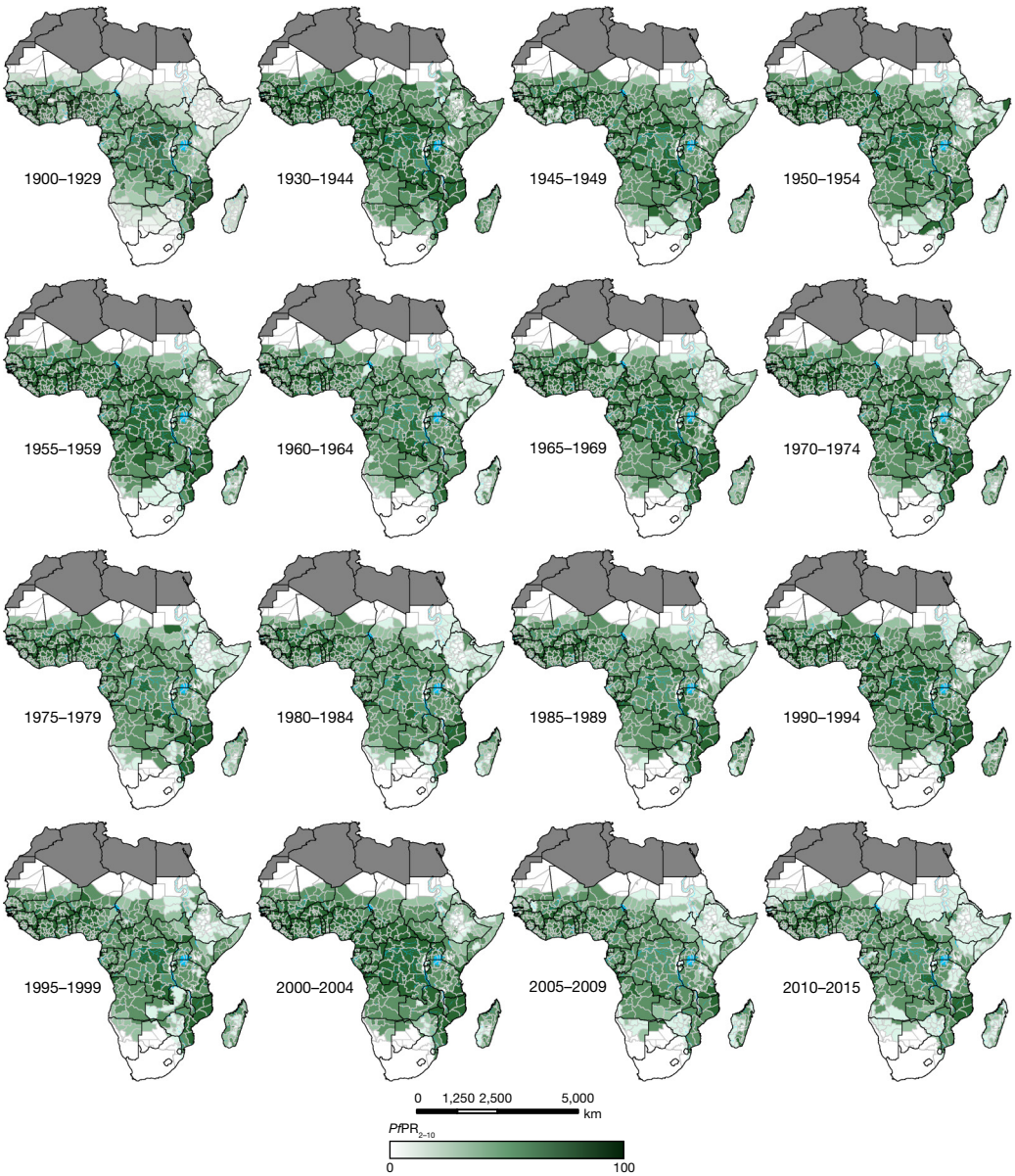

Figure 1 | Changing spatial patterns of *P. falciparum* endemicity in sub-Saharan Africa since 1900. Predicted posterior predictions of age-standardized *P. falciparum* prevalence ($PfPR_{2-10}$) per administrative unit on mainland sub-Saharan Africa and Madagascar, masked (white) according to biological- or control-related absence of transmission (see Methods and Supplementary Information 2.2) and the reported changing spatial extents of malaria transmission (see Methods, Supplementary Information 2.3, and Source Data).

accessed 8 April 2017)[15] and United Nations (World Urbanization Prospects, accessed 8 April 2017)[16] cannot account for the emergence of the 1985–2004 malaria epidemic. Conversely, minimum temperatures across sub-Saharan Africa have risen by over 1 °C since the 1970s[10] (Fig. 2); the linear phenomena of global warming cannot explain the precipitous declines in malaria prevalence witnessed after 2004. The interplay among malaria, climate, effective or failing intervention, human settlement and development is inevitably complex. Our analysis highlights the fact that a focus on simple, single factors fails to adequately explain the cycles of parasite prevalence.

The reduction in malaria transmission intensity has not occurred equally between countries or within countries (Fig. 1), with more substantive declines and 'shrinking of the map' occurring at the margins of the historical range of *P. falciparum* transmission than in the heartland of Africa's most efficient vector species, *Anopheles gambiae sensu stricto* and *Anopheles coluzzii*. This heartland forms a densely populated belt from West Africa through Central Africa toward Mozambique, and represents the most severely impacted area of the contemporary malaria-endemic world: it was ignored after 1960[17,18] and risks being ignored today[19]. Our previous and current armoury of interventions has not eliminated malaria in this part of the world, and there is little indication that it will do so in the foreseeable future.

Although caution is required in predicting a complex future, if past trends remain consistent we would expect further reductions in the range and intensity of malaria transmission in Africa, punctuated with resurgences. We show the implausibility of simple explanations for temporal trends over the past 115 years, and therefore caution against using similar explanations for the trend of the past 15 years (for example, in ascribing this trend to human intervention alone). The unique malaria endemicity that prevails in Africa cannot be ignored in global efforts to eliminate *P. falciparum*, nor should we wait for future rises in malaria prevalence to re-galvanize interest in a parasite that remains entrenched across large parts of the continent.

Online Content Methods, along with any additional Extended Data display items and Source Data, are available in the online version of the paper; references unique to these sections appear only in the online paper.

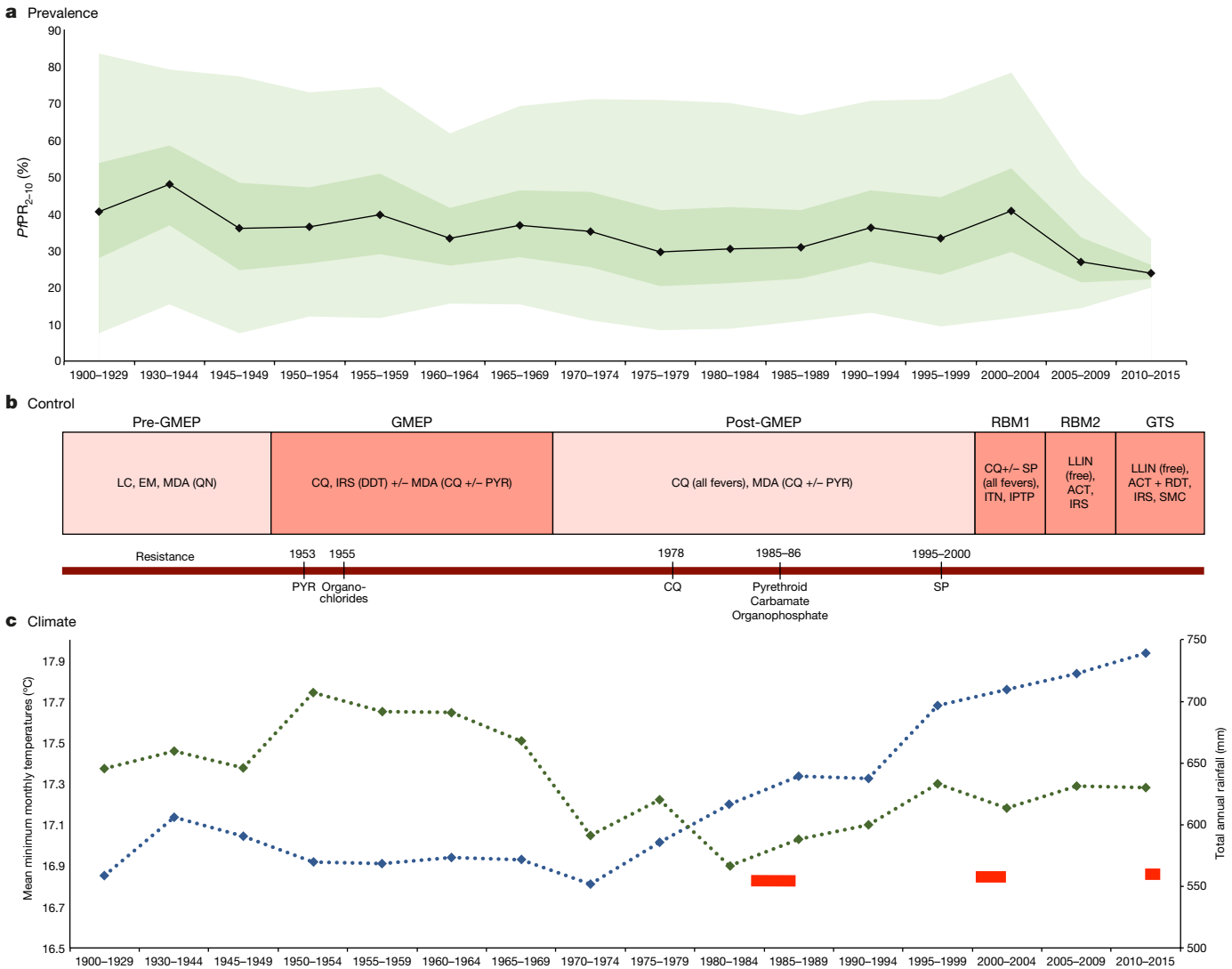

Figure 2 | Summary and plausibility framework of *P. falciparum* transmission cycles in sub-Saharan Africa since 1900. a, The median (dark green line) and 25–75% (medium green boundaries) and 2.5–97.5% (light green boundaries) interquartile credibility range of the posterior predictions of $PfPR_{2-10}$ (see Source Data). **b**, Six periods of major intervention: (1) 1900–1949, restricted efforts through larval control (LC), environmental management (EM) and mass drug administration (MDA) using quinine (QN); (2) 1950–1969, launch of global malaria eradication programme (GMEP) in 1955, introduction of DDT and pilot elimination projects involving indoor residual house-spraying (IRS), accompanied later by MDA using chloroquine (CQ) and pyrimethamine (PYR); (3) 1970–1999, end of most vector control efforts, presumptive treatment of fevers with chloroquine, use of chloroquine in MDA to school children; (4) 2000–2004, the roll back malaria (RBM) initiative with insecticide-treated nets (ITN) for vulnerable children and pregnant women, expansion of intermittent presumptive treatment of malaria in pregnancy (IPTp) and failing first line treatment with sulfadoxine–pyrimethamine (SP) and/or chloroquine; (5) 2005–2010, distribution of long-lasting insecticide-treated nets (LLIN) on a large scale, IRS expansion, and switch from chloroquine or sulfadoxine–pyrimethamine to artemisinin-based combination therapy (ACT); (6) 2010–2015, increased IRS in many countries, scale-up of rapid diagnostic tests (RDTs), launch of the Global Technical Strategy (GTS) in 2012, which re-invigorated a global ambition for eradication and seasonal malaria chemoprevention (SMC) in West African countries. Vector resistance to organochlorines was detected in 1955 in Nigeria; organophosphate, carbamate and pyrethroid resistance was detected in the late 1980s and has expanded rapidly since the late 1990s[20]; chloroquine resistance was detected in 1978; sulfadoxine–pyrimethamine resistance was detected in 1953, with substantial clinical failure rates in 2000[12]. **c**, Mean annual rainfall across the Sahara (green)[10] and monthly minimum temperatures (blue)[10]; El Niño events leading to serious climate anomalies, including flooding in 1997–1998 in East Africa and drought in the Horn of Africa in 2014–2015 (red bars). Climate data from National Oceanic and Atmospheric Administration, US Department of Commerce (http://www.cpc.ncep.noaa.gov/products/analysis_monitoring/ensostuff/ensoyears.shtml).

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Supplementary Information is available in the online version of the paper.

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Author Contributions R.W.S. assembled the data, designed the experiment and wrote the paper; B.S. undertook the statistical analysis; P.B. provided support for data interpretation; A.M.N. provided support for data assembly and analysis; and D.K., J.M., P.A. and C.W.M. all provided assistance in locating survey reports, abstraction of data and geo-coding. All authors have access to the data and have reviewed the paper and Supplementary Information.

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METHODS

No statistical methods were used to predetermine sample size.

Data Assembly. Over the past 21 years, we have sourced unpublished and published materials related to community-based malaria infection prevalence at European, United Nations and African national libraries, archives and ministry of health repositories. We undertook standard electronic data searches of peer-reviewed publications, and contacted malaria scientists, regional health research institutes, and government and non-government agencies involved in the delivery and monitoring of malaria interventions (Supplementary Information 1.3, 1.4). The minimum data requirements for the survey included the date and location, age range and numbers for participants examined, infection prevalence by species, and parasite detection method. A total of 50,424 parasite prevalence surveys were included[4] (Extended Data Figs 1, 2, Supplementary Information 1.5).

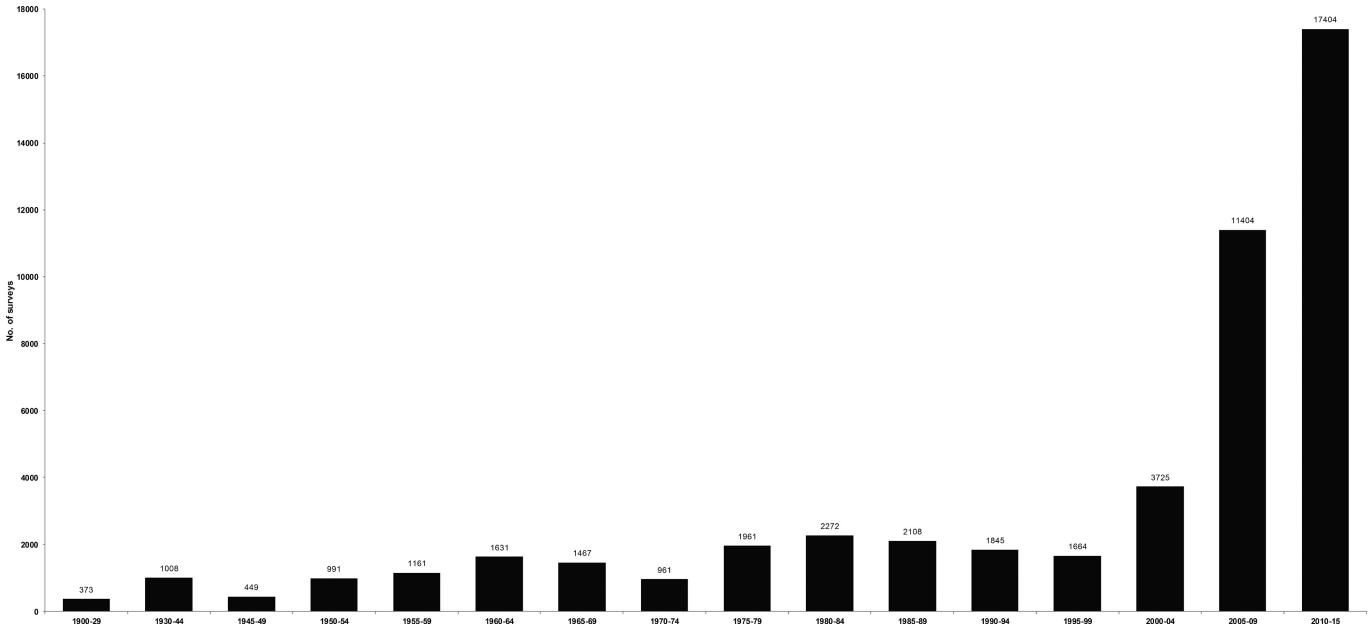
Spatial limits and resolution of malaria predictions. We excluded previously endemic North African countries (Morocco, Algeria, Tunisia, Libya and Egypt), off-shore islands and countries where malaria has not been described (Western Sahara and Lesotho). Guided by the United Nations' GAUL project (http://www.fao.org/geonetwork/srv/en/metadata.show?id=12691), we used current national and sub-national first-level administrative boundary units, with adaptations for the margins of natural *P. falciparum* risk and for disputed boundaries; we also dissolved the boundaries of small urban municipalities and ensured contiguous shapes between sub-national units. Rwanda, Burundi, Djibouti, Swaziland and The Gambia were treated as single polygons (Supplementary Information 2.1, 2.2; see Source Data of Fig. 1). The natural spatial limits of *P. falciparum* risk were derived from expert opinion, national maps and biological constraints (Supplementary Information 2.2; see Source Data of Fig. 1). The selection of 520 spatial polygons at the natural range of *P. falciparum* transmission is shown in Extended Data Fig. 3. Changing limits were mapped using data from national reports of malaria incidence from the 1960s onward (Supplementary Information 2.3; see Source Data of Fig. 1).

Statistical methods. We used a Bayesian hierarchical binomial model that simultaneously estimates stable spatial and temporal structured patterns and departures from these stable components[5]. The input data were as follows: observed number of children aged 2–10 years with *P. falciparum* ($Pf\mathrm{PR}_{it}$) and total number of tested children aged 2–10 years (n_{it}) for subnational region (i, from 1 to 520) and time periods ($t = 1$–16, corresponding to 1900–1929, 1930–1944, five-year periods from 1945–1949 to 2004–2009, and one six-year period, 2010–2015). The model was fitted using Markov chain Monte Carlo (MCMC) simulation using non-informative priors. Posterior distributions of parameters were obtained using WinBUGS software (Supplementary Information 3; see Source Data of Fig. 1). Gelman–Rubin statistics were used to assess model convergence (Extended Data Fig. 4). Output was validated using observed versus fitted $Pf\mathrm{PR}_{2-10}$ from the full model (Extended Data Fig. 5).

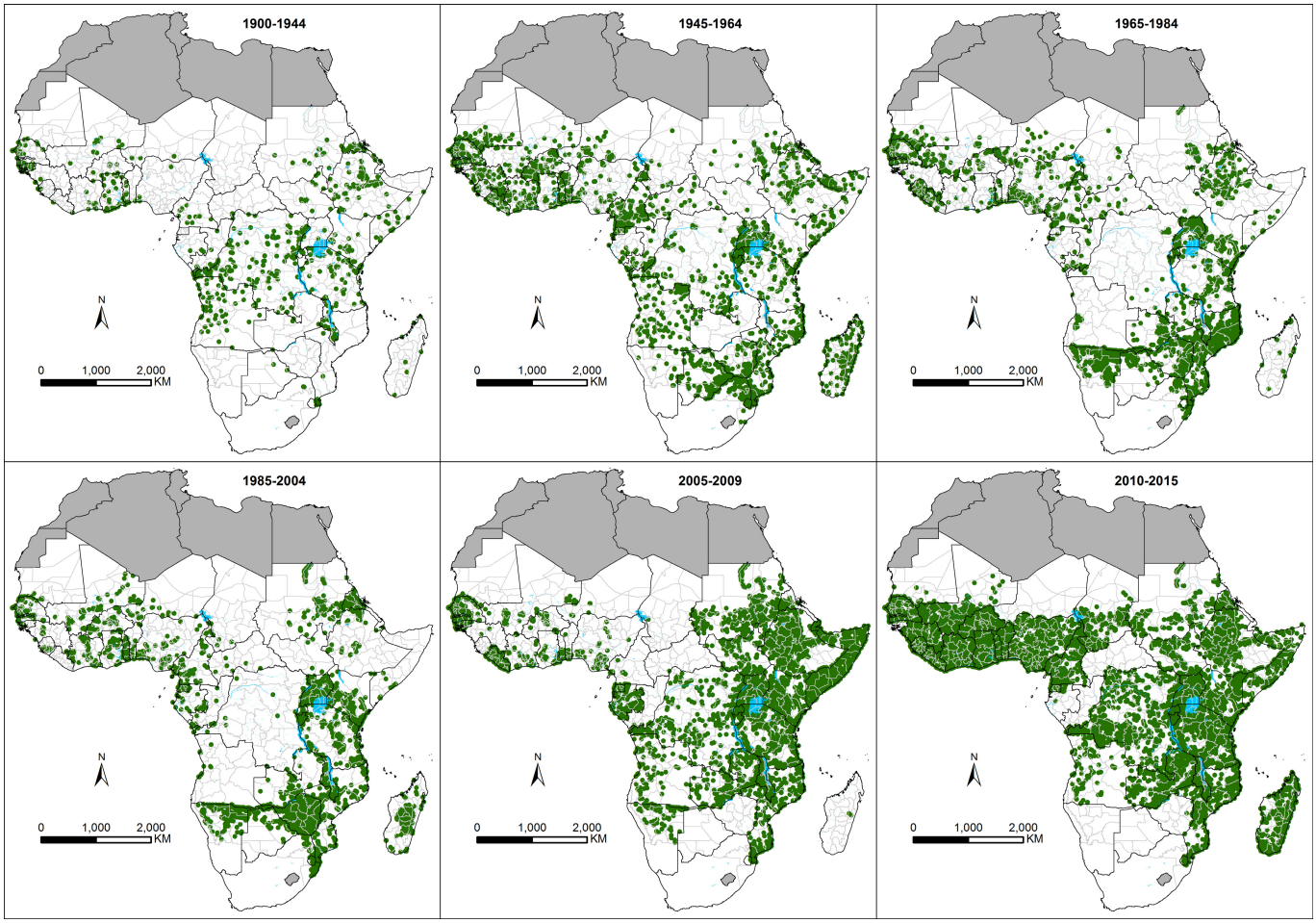
Ethics statement. As the secondary use of aggregate survey data, our research centre considered the work to be non-human research for which individual informed consent was not applicable.

Code availability. The WINBUGs code for both the negative binomial and Poisson models are freely accessible at http://dx.doi.org/10.7910/DVN/Z29FR0. No restrictions apply to their use.

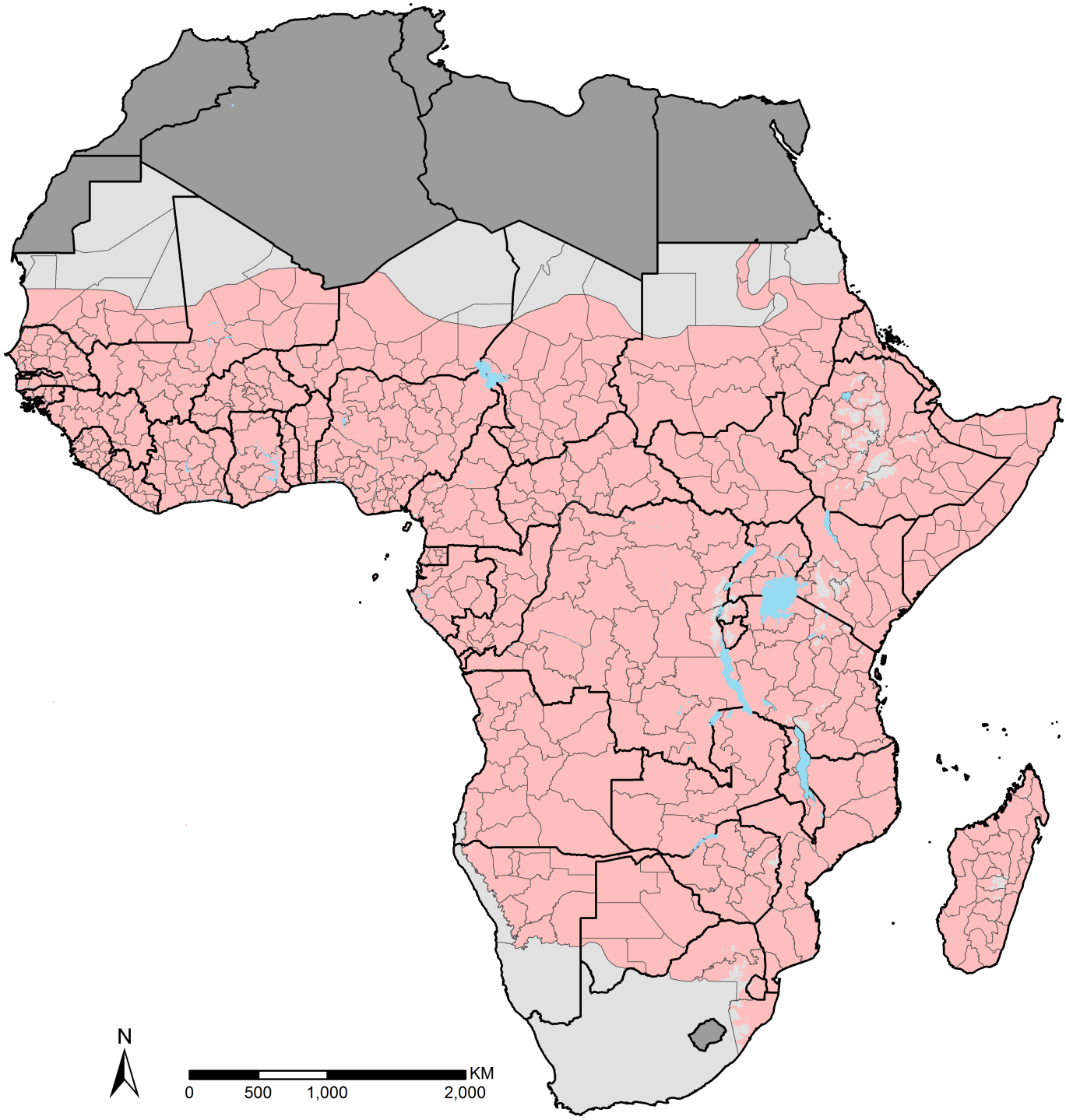
Data availability. The full database of survey data that support the findings of this study are available from the Harvard Dataverse (http://dx.doi.org/10.7910/DVN/Z29FR0) under a CC-BY 4.0 license. Source Data are available in the online version of the paper.



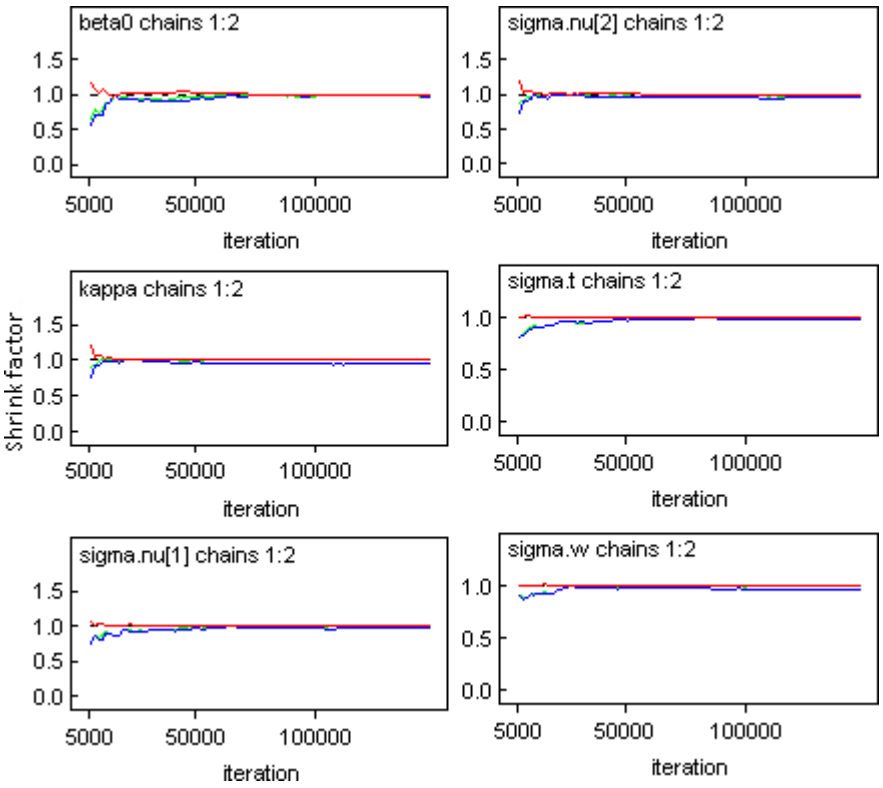
Extended Data Figure 1 | Availability of survey data over time. The temporal distribution of survey data per interval selected for analysis (number of surveys shown on top of bars).



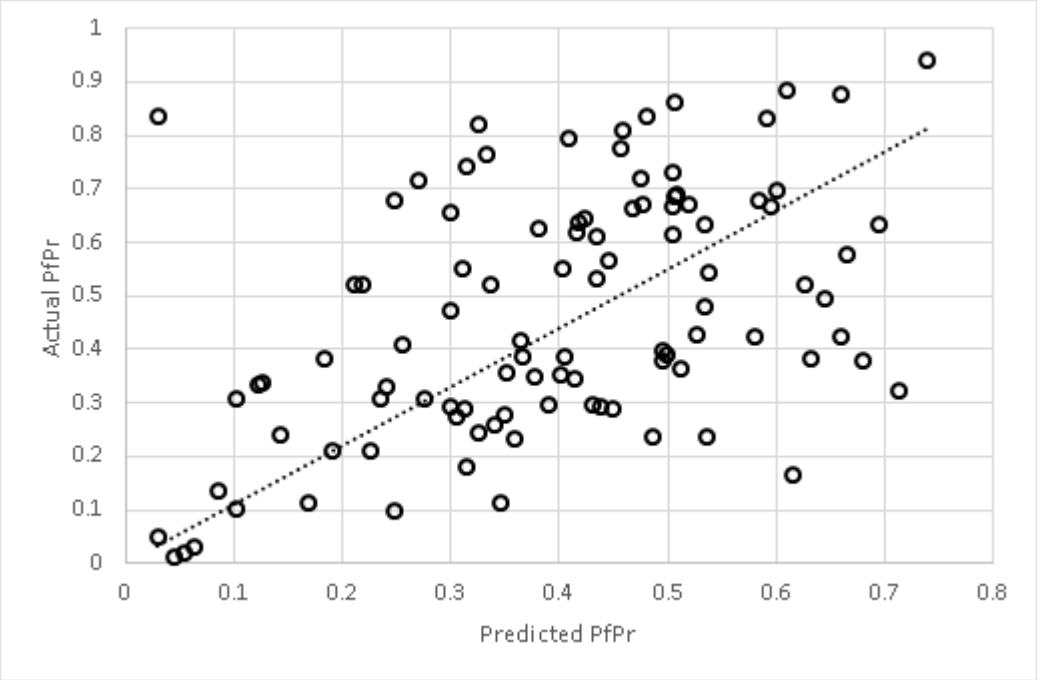
Extended Data Figure 2 | Spatial distribution of survey data. Location of 50,424 *P. falciparum* parasite surveys undertaken at 39,033 locations by time interval from 1900–1944 to 2010–2015.



Extended Data Figure 3 | The spatial range of *P. falciparum* in Africa between 1900 and 1950. Light grey, absence of natural *P. falciparum* transmission; pink, natural extent of transmission; dark grey, countries not included in the analysis.



Extended Data Figure 4 | Model convergence: Gelman–Rubin–Brooks plots demonstrating convergence during MCMC simulation for key model parameters. Black line, ratio of within-chain variability to between-chain variability; dark grey line, within-chain variability (pooled); light grey line, between-chain variability (average).



Extended Data Figure 5 | Model validation. Predicted *Pf*PR$_{2-10}$ versus observed *Pf*PR$_{2-10}$ for 100 randomly selected data points. Ninety-nine per cent of data points are within 95% credible interval (CI); Spearman rank correlation 0.46, $P < 0.001$ (two-sided test).

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Corresponding author(s): Robert W Snow

☐ Initial submission ☐ Revised version ☒ Final submission

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Experimental design

1. Sample size

Describe how sample size was determined.

Data was obtained from 1000s of community random sample studies over the last 115 years designed for different purposes and with different sample sizes. The data from these studies are provided at Harvard Dataverse with the publication of this paper - see data availability statement - showing original sample sizes. The Bayesian statistical methods used for analysis adjust for sample size variations in the estimation of parasite prevalence. This described in methods section and SI

2. Data exclusions

Describe any data exclusions.

Data were excluded if not from randomly sampled community surveys, did not have geo-coordinates or were done only among febrile cases, or those attending clinics or among special groups such refugees. Geographically data was excluded for the small off shore Island states of Sao Tome and Principe, Cape Verde, Annobin and Bioko islands of Equatorial Guinea, Zanzibar and Pemba of the United Republic of Tanzania and the archipelagos of Comores, Mauritus, Reunion and Mayotte.

3. Replication

Describe whether the experimental findings were reliably reproduced.

The Bayesian methods code and data are provided with the manuscript and should be easily reproducible

4. Randomization

Describe how samples/organisms/participants were allocated into experimental groups.

Not Applicable. Please refer to data exclusion criteria to explain data selected for this study

5. Blinding

Describe whether the investigators were blinded to group allocation during data collection and/or analysis.

Not applicable

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For all figures and tables that use statistical methods, confirm that the following items are present in relevant figure legends (or in the Methods section if additional space is needed).

n/a	Confirmed
☐	☒ The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement (animals, litters, cultures, etc.)
☐	☒ A description of how samples were collected, noting whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
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☒	☐ Clearly defined error bars

See the web collection on statistics for biologists for further resources and guidance.

Software

Policy information about availability of computer code

7. Software

Describe the software used to analyze the data in this study.

WinBUGS for Bayesian inference, Excel for data assembly, ArcGIS for map display

For manuscripts utilizing custom algorithms or software that are central to the paper but not yet described in the published literature, software must be made available to editors and reviewers upon request. We strongly encourage code deposition in a community repository (e.g. GitHub). *Nature Methods* guidance for providing algorithms and software for publication provides further information on this topic.

Materials and reagents

Policy information about availability of materials

8. Materials availability

Indicate whether there are restrictions on availability of unique materials or if these materials are only available for distribution by a for-profit company.

None

9. Antibodies

Describe the antibodies used and how they were validated for use in the system under study (i.e. assay and species).

Not Applicable

10. Eukaryotic cell lines

a. State the source of each eukaryotic cell line used.

Not Applicable

b. Describe the method of cell line authentication used.

Not Applicable

c. Report whether the cell lines were tested for mycoplasma contamination.

Not Applicable

d. If any of the cell lines used are listed in the database of commonly misidentified cell lines maintained by ICLAC, provide a scientific rationale for their use.

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11. Description of research animals

Provide details on animals and/or animal-derived materials used in the study.

Not Applicable

Policy information about studies involving human research participants

12. Description of human research participants

Describe the covariate-relevant population characteristics of the human research participants.	Data on malaria infection among surveyed populations were used in aggregate form and were anonymised