



## Quantifying the Impact of Human Mobility on Malaria

Amy Wesolowski *et al.*  
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# Quantifying the Impact of Human Mobility on Malaria

Amy Wesolowski,<sup>1,2</sup> Nathan Eagle,<sup>3,4</sup> Andrew J. Tatem,<sup>5,6,7</sup> David L. Smith,<sup>6,8</sup> Abdisalan M. Noor,<sup>9,10</sup> Robert W. Snow,<sup>9,10</sup> Caroline O. Buckee<sup>4,11\*</sup>

Human movements contribute to the transmission of malaria on spatial scales that exceed the limits of mosquito dispersal. Identifying the sources and sinks of imported infections due to human travel and locating high-risk sites of parasite importation could greatly improve malaria control programs. Here, we use spatially explicit mobile phone data and malaria prevalence information from Kenya to identify the dynamics of human carriers that drive parasite importation between regions. Our analysis identifies importation routes that contribute to malaria epidemiology on regional spatial scales.

Local “hot spots” of malaria prevalence, resulting from complex interactions between the malaria parasite *Plasmodium falciparum* and its human and mosquito hosts, provide specific targets for the strategic deployment of malaria interventions (1–4). Movements of infected humans can increase the dispersal of parasites beyond what would be possible for mosquitoes alone (5, 6), and national malaria control programs must ac-

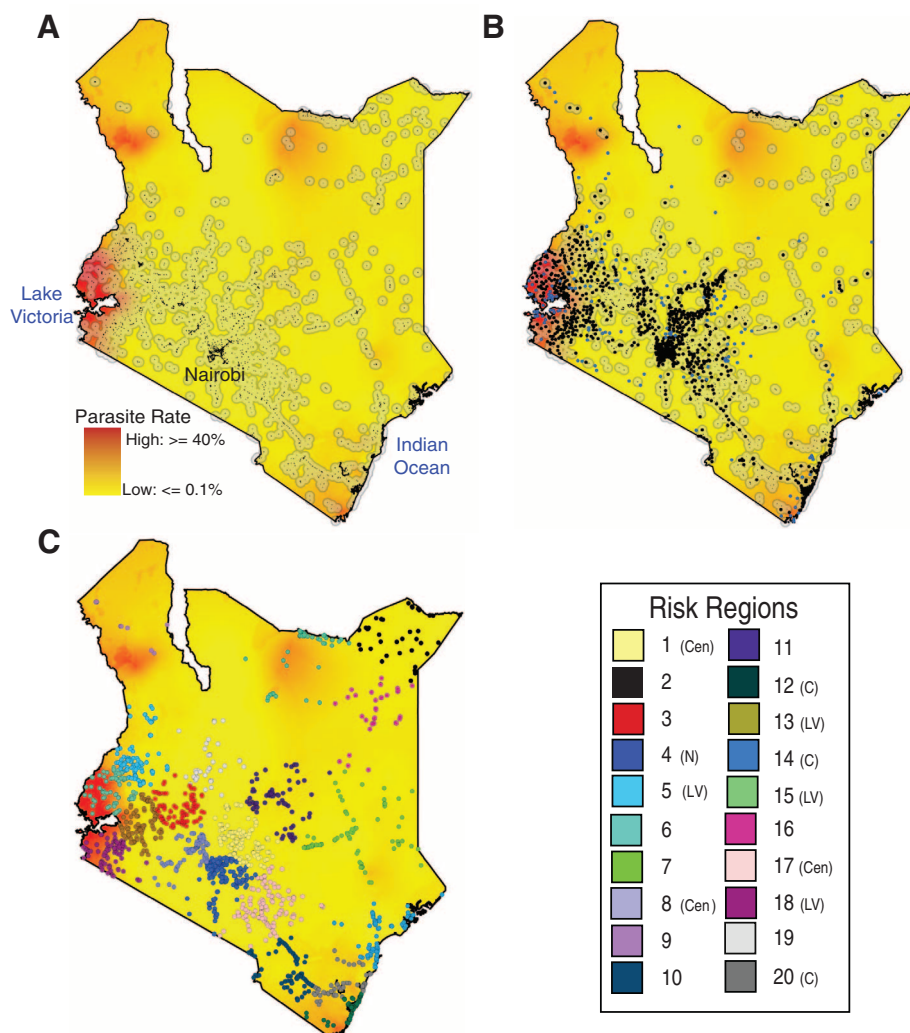
count for this human travel-mediated spread of parasites because frequent introduction of imported parasites could undermine local control or elimination strategies (5, 7–9). Mapping the routes of parasite dispersal by human carriers will allow for additional targeted control by identifying both the regions where imported infections originate and where they may contribute substantially to transmission. International migrants can contrib-

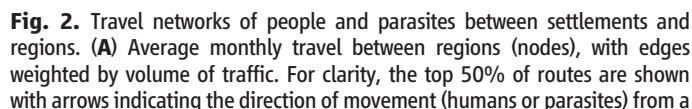
ute to continental parasite dispersal across Africa, and census surveys have provided insights into these routes of importation (6). The vast majority of travelers that will affect malaria parasite dispersal are those moving within a country between regions of variable malaria receptivity on a daily or weekly basis, however.

<sup>1</sup>Department of Engineering and Public Policy, Carnegie Mellon University, Pittsburgh, PA 15221, USA. <sup>2</sup>Department of Statistics, Carnegie Mellon University, Pittsburgh, PA 15221, USA. <sup>3</sup>College of Computer and Information Science, Northeastern University, Boston, MA 02115, USA. <sup>4</sup>Department of Epidemiology, Harvard School of Public Health, Boston, MA 02115, USA. <sup>5</sup>Emerging Pathogens Institute, University of Florida, Gainesville, FL 32610, USA. <sup>6</sup>Fogarty International Center, National Institutes of Health, Bethesda, MD 20892, USA. <sup>7</sup>Department of Geography, University of Florida, Gainesville, FL 32610, USA. <sup>8</sup>Department of Epidemiology and Malaria Research Institute, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD 21205, USA. <sup>9</sup>Malaria Public Health and Epidemiology Group, Centre of Geographic Medicine, KEMRI–Wellcome Trust–University of Oxford Collaborative Programme, Nairobi, Kenya. <sup>10</sup>Centre for Tropical Medicine, Nuffield Department of Clinical Medicine, University of Oxford, Oxford OX3 7LJ, UK. <sup>11</sup>Center for Communicable Disease Dynamics, Harvard School of Public Health, Boston, MA 02115, USA.

\*To whom correspondence should be addressed. E-mail: cbuckee@hsph.harvard.edu

**Fig. 1.** The distribution of settlements, cell towers, and malaria risk in Kenya. **(A)** Malaria prevalence in Kenya in 2009 (from  $PfPR_{2-10} < 0.1\%$  in yellow to  $PfPR_{2-10} > 40\%$  in red) and the locations of settlements used in the analysis (settlement centers are shown in black, and mapped with a 10-km extent around the perimeter of the settlement in gray). **(B)** The location of mobile phone towers (black or blue dots) and the extended settlement boundaries. Towers that fall within a settlement are shown in black, and those excluded from the analysis are shown in blue. **(C)** Regions used for visual mapping of transmission routes. Each settlement was allocated to 1 of 20 regions by a clustering algorithm (14) on the basis of homogeneous malaria risk and geography, as shown. Regions near Lake Victoria (LV), in Nairobi (Nairobi), the central areas (Cen), and along the coast (C) are labeled accordingly.





The travel network (Fig. 2A and fig. S2A) is dominated by the Kenyan capital Nairobi, which forms a hub for human movements to and from all regions of the country. Although the highest

**A**

**B**

Source

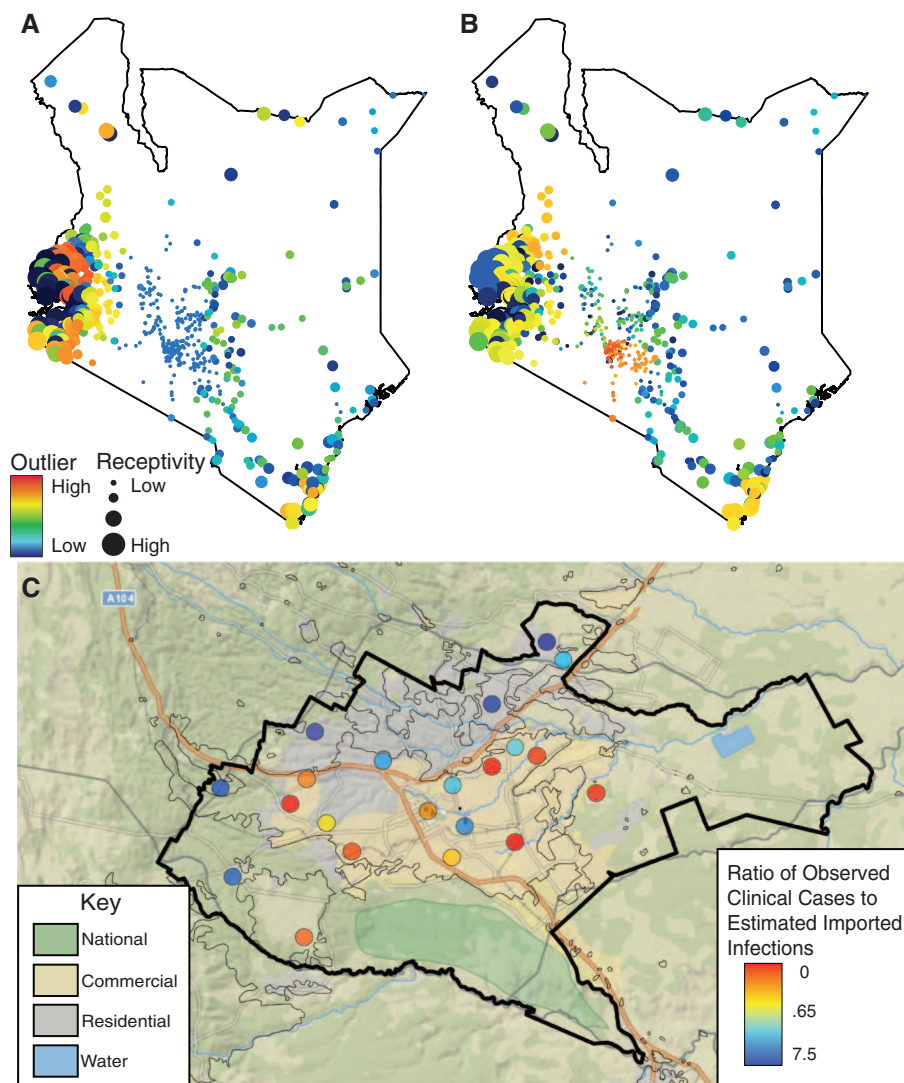
Sink

volume of travel occurs between Nairobi and the central regions of the country, substantial movement also occurs between the central region and Lake Victoria (for values, see tables S1 and S2).

mitters” in previous frameworks (1) (fig. S2C and tables S5 and S6). For this analysis, we assume that receptivity to transmission is reflected by the prevalence of infection, although this simplification does not account for current control measures, which we discuss below. The structures of these networks were remarkably stable over the course of the year (see figs. S3 to S5), so, although seasonal changes in transmission might cause our estimates of parasite movement to be generally high, the routes and relative volumes will remain unaffected.

Parasite movement networks represent only a subset of the human mobility network underlying them, because of the spatial heterogeneity in malaria risk across the country. The human travel network is denser than the parasite networks, as expected, with more edges and a higher mean degree per settlement, as well as greater connectivity (see table S7). Returning residents contribute to some movements of parasites between





**Fig. 4.** Local analysis of source-sink anomalies. (A) Source outliers and (B) sink outliers. Settlements are colored by their outlier rank (from low values in blue to high values in red) and sized according to  $R_c$ , an indicator of receptivity. (C) The ratio of estimated localized importation to malaria cases at clinics around Nairobi. A topographic map of the city was from National Geographic, and the Economic and Social Research Institute's geographic information system highlights the national park, commercial, and residential areas.

regions within the Lake Victoria and coastal areas (Fig. 2B), but Nairobi imports the largest fraction of infections in this way, with infected residents returning after journeys to the coast, Lake Victoria, and low-endemicity regions in central Kenya. Visitors contribute to transmission anywhere that is receptive to transmission (14) (Fig. 2C), but may have less impact in the capital, for example, where vectors are scarce. Hence, the visitor network is dominated by importation around Lake Victoria and shows relatively low importation rates between the lake and the coast, the two main foci of transmission. Visitors carrying parasites within regions are therefore likely to be a much more important consideration for control programs than interregional visitors, which suggests that the Lake Victoria and coastal regions may be considered as weakly connected, but relatively

independent, entities for the purposes of malaria elimination.

To examine directional and net movements of people and parasites between settlements, we analyzed asymmetries between “source” and “sink” settlements. Here, we rank settlements based on their contribution as net emitters (sources) and net receivers (sinks) of people and parasites (human travel in Fig. 3A, parasite movement in Fig. 3B) (14). The difference between each settlement's source and sink rank distinguishes those that are primarily sources of people or parasites versus those that are primarily sinks. Sources and sinks of human travel are almost entirely overlapping and reflect patterns of population density and regular travel (Fig. 3A). In contrast, the parasite routes show directional movement between source settlements in the Lake Victoria region

and parasite sinks on the periphery of this focus of transmission and in the Nairobi area (Fig. 3B) (14). The capital city and its surroundings are thus a major destination for both humans and parasites, but most of the parasite importation that can contribute to onward transmission occurs on the periphery of the highly endemic Lake Victoria region. Therefore, even though malaria prevalence is low in these regions and so amenable to control, elimination programs must account for imported infections to be successful.

The high spatial resolution of our mobility data allowed us to pinpoint particular settlements that are expected to receive or transmit an unexpectedly high volume of parasites compared with surrounding regions. The result of an analysis of outlying settlements identified by means of an anomaly detection algorithm is shown (Fig. 4, A and B) (14). Here, the size of the circle represents  $R_c$ , the basic reproductive number of the parasite under control (16). This measure provides insights into how important outliers are likely to be for transmission, because importation can only contribute to transmission if local conditions and vector populations allow it. Combining local estimates of importation with information about locally heterogeneous transmission—including vector behavior, ecology, and population distributions on a fine scale—will play an important role in future regional elimination efforts. Again, the settlements on the edge of Lake Victoria are major sources of parasites, and the neighboring settlements farther inland are most vulnerable to importation. Returning residents played an important role in importing parasites to major parasite sinks, with residents from the top 10% of outlying settlements taking, on average, 29 trips during the year, compared with 20 trips by individuals from the remaining 90% of settlements (medians 10.4 versus 7.6, respectively, Mann-Whitney  $U$  test,  $P < 0.0001$ ). These sinks also received substantial numbers of visitors from higher malaria endemicity settlements (24% of visitors) compared with settlements that were not considered sinks (12% of visitors). In contrast, individuals from the top 10% of major parasite source settlements did not travel more frequently, but 62% of journeys made were to settlements with lower malaria endemicity compared with 0.08% of journeys made from the remaining 90% of settlements ( $P < 0.0001$ ) (tables S8 and S9).

In Nairobi, the density of cell towers enabled further localization of these estimates and a comparison with cross-sectional clinical surveys of malaria incidence carried out in 2010 (17). Frequent malaria epidemics occurred in the capital at the beginning of the 20th century but declined markedly after substantial control efforts, rapid population growth, and urbanization. The current potential for local transmission within the city is controversial, with studies showing substantial infection prevalence and ongoing treatment of presumed clinical cases, despite the scarcity of suitable mosquito vectors (18–20). The ratio of monthly clinical cases to our predicted monthly imported

cases from mobile phone data at the location of each hospital survey is shown (Fig. 4C) (14) (table S10 and fig. S6). Areas in the highly urbanized center of the city, where transmission is unlikely, show a very large ratio of estimated imported-to-clinical cases. In contrast, hospitals on the periphery of the city have a higher ratio of clinical cases to estimates from the mobile phone data. The patterns suggest some local transmission may be occurring in these residential and less developed areas and could increase if migration into the areas surrounding the city is not accompanied by improved public health infrastructure and surveillance programs. Poor malaria monitoring in clinics around the city is currently hindering the accurate assessment of malaria transmission (17). Although caution must be exercised in the interpretation of comparisons between clinical and mobile phone estimates, this approach provides a starting point for the identification of transmission foci in low-risk urban settings and the local implementation of surveillance programs.

There are limitations to this approach (10), because we can only measure mobility among phone owners in areas where there are cell towers (21) (see supplementary materials for discussion), we cannot capture cross-border migration, and our importation calculations are constrained by the available, nonseasonal malaria prevalence estimates. Nevertheless, this analysis has made it possible to assess the degree of connectivity among different regions of Kenya—the resulting estimates can be used to estimate costs for regional elimination strategies, identify “source” regions where reducing transmission would provide benefit to surrounding areas, evaluate patterns of importation and endemicity in low-intensity

areas such as Nairobi, and pinpoint likely importation hot spots. On an extremely local scale, driven primarily by vector biology and habitat and local variability in household structures, hot spots of transmission can be targeted by indoor residual spraying, vector habitat removal, insecticides, drug administration, and bed-net use. Control-program activities targeting the large volumes of human traffic between regions that we have identified here will be completely different from those that concentrate on local transmission hot spots, focusing on communicating risks to travelers to alter their behaviors, restricting travel patterns, and/or conducting routine surveillance in high-risk areas.

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#### Supplementary Materials

[www.sciencemag.org/cgi/content/full/338/6104/267/DC1](http://www.sciencemag.org/cgi/content/full/338/6104/267/DC1)  
Materials and Methods  
Supplementary Text  
Figs. S1 to S6  
Tables S1 to S10  
References (22–28)

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## Preference by Association: How Memory Mechanisms in the Hippocampus Bias Decisions

G. Elliott Wimmer and Daphna Shohamy\*

Every day people make new choices between alternatives that they have never directly experienced. Yet, such decisions are often made rapidly and confidently. Here, we show that the hippocampus, traditionally known for its role in building long-term declarative memories, enables the spread of value across memories, thereby guiding decisions between new choice options. Using functional brain imaging in humans, we discovered that giving people monetary rewards led to activation of a preestablished network of memories, spreading the positive value of reward to nonrewarded items stored in memory. Later, people were biased to choose these nonrewarded items. This decision bias was predicted by activity in the hippocampus, reactivation of associated memories, and connectivity between memory and reward regions in the brain. These findings explain how choices among new alternatives emerge automatically from the associative mechanisms by which the brain builds memories. Further, our findings demonstrate a previously unknown role for the hippocampus in value-based decisions.

**D**ecisions are sometimes guided by direct past experience: If a choice led to a good outcome in the past, people are likely

to make that same choice again. This process is known to depend on reward learning mechanisms in the striatum (1, 2). But frequently in

life, we have to decide between options we have never considered before. It has been suggested that such decisions could be guided by associative memory (3–5); however, surprisingly little is known about how this process happens.

We investigated the mechanism by which neural circuits for memory modulate value and guide decisions about new choice options. Our central hypothesis was that the hippocampus enables the positive value of reward to spread across associated memories, thereby increasing the value of items that were never rewarded. Specifically, we hypothesized that receiving reward can lead to two simultaneous and interactive processes: (i) the direct learning of stimulus-reward associations in the striatum and (ii) the spread of reward to associated items stored in memory via the hippocampus.

Our hypothesis is grounded in two essential features of how the hippocampus builds memories. First, the hippocampus encodes relationships

Department of Psychology, Columbia University, New York, NY 10027, USA.

\*To whom correspondence should be addressed. E-mail: shohamy@psych.columbia.edu