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# Outbreak of a new measles B3 variant in the Roma/Sinti population with transmission in the nosocomial setting, Italy, November 2015 to April 2016

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**A measles outbreak occurred from November 2015 to April 2016 in two northern Italian regions, affecting the Roma/Sinti ethnic population and nosocomial setting. Overall, 67 cases were reported. Median age of 43 cases in three Roma/Sinti camps was four years, nosocomial cases were mainly adults. The outbreak was caused by a new measles virus B3.1 variant. Immunisation resources and strategies should be directed at groups with gaps in vaccine coverage, e.g. Roma/Sinti and healthcare workers.**

Despite a national goal to eliminate measles by 2015, Italy is one of 18 European Region Member States where endemic transmission of measles has not been interrupted [1]. We report here a recent measles outbreak in northern Italy (Figure 1) affecting the Roma/Sinti ethnic population and the nosocomial setting.

## Outbreak description

Measles cases were reported to the Italian national integrated measles and rubella surveillance system coordinated by the Istituto Superiore di Sanità in Rome.

## Milan clusters (Lombardy region, northern Italy)

The outbreak originated in a nomadic settlement in Milan and then spread to two other settlements in the city. The three settlements house persons of mainly Roma/Sinti ethnic origin but also migrants of various nationalities. A large degree of social interaction occurs between the three camps, as they are inhabited by related family groups.

In total, 43 cases were reported with dates of rash onset from 7 November 2015 to 18 February 2016 (weeks 45/2015 to 7/2016). Figure 2 shows the number of reported measles cases by date of symptom onset. Forty of 43 cases were Roma/Sinti while three were migrants, including one individual from North Africa and two from South Asia. Median age of the 43 cases was four years (range: 5 months–29 years) and 39 were 15 years or younger. Thirty-nine of 43 cases were unvaccinated. Of the four vaccinated cases, three had received post-exposure vaccination in connection with the outbreak.

Twenty-seven cases were laboratory-confirmed and 16 were probable cases [2]. Twelve cases reported complications, including six cases of pneumonia, four cases of diarrhoea and two cases of otitis media. Eighteen cases were admitted to hospital and 14 were seen in the emergency department but not admitted.

## Parma and Piacenza clusters (Emilia Romagna region, northern Italy)

The outbreak spread from Lombardy to Emilia Romagna in the cities of Parma (17 cases from 13 January to 13 April) and Piacenza (seven cases from 6 March to 3 April 2016). Here, transmission occurred mainly in the nosocomial setting.

The Parma cluster originated from an unvaccinated adult, employed near a Roma camp in Milan, who was visiting Parma. While there, the patient developed fever in early January 2016 and was hospitalised four days later. Measles was diagnosed after two more

**FIGURE 1**

Areas involved in a measles outbreak affecting the Roma/Sinti population with transmission in the nosocomial setting, Italy, November 2015–April 2016



days, following rash onset. While hospitalised, the case transmitted the infection to an unvaccinated adult sibling. Two healthcare workers (HCW) in the same hospital and two persons who visited the hospital during the outbreak period also developed measles, although no direct contact with any of the other identified cases was reported. One of the HCW transmitted the infection to another HCW. Other cases related to this cluster included four HCW working in different healthcare settings in the community and six persons with no apparent contact with any of the above cases. All 17 cases were linked virologically. Median age was 37 years (range: 22–49 years). None were vaccinated. One case was complicated by keratoconjunctivitis.

The Piacenza cluster originated from a hospital worker with no direct patient contact, who was hospitalised with high fever over 14 days starting in the first week of March, but in whom a diagnosis of measles was made only retrospectively, after hospital discharge, when four unvaccinated HCW in the same hospital developed measles. Two HCW transmitted the infection to a relative outside the hospital setting. Median age of these seven cases was 40 years (range: 35–68 years). A high rate of complications occurred among cases: three developed pneumonia, two of them with acute respiratory insufficiency, three developed diarrhoea, two keratoconjunctivitis, one pericarditis, one

thrombocytopenia and one Guillain-Barré syndrome. In addition, five of seven cases developed increased liver enzymes. All HCW were admitted to hospital.

### Genotyping and phylogenetic analysis

We performed a genetic analysis of measles viruses isolated from 14 of 43 cases from the three Roma camps (respectively, two, 11 and one strain from camps 1, 2 and 3) and 22 of 24 cases from the clusters in Emilia Romagna (respectively, 17 and five cases from the Parma and Piacenza clusters). Phylogenetic analysis based on the available partial nucleoprotein gene sequences showed that all sequences were identical to each other and belonged to genotype B3.1 (Figure 3) [3,4].

BLAST analysis of the World Health Organization's measles nucleotide surveillance (MeaNS) database [5,6] shows that the strain identified represented a new variant of this genotype, first reported in August 2015 (week 32/2015) in Como (Lombardy) and Rimini (Emilia-Romagna), in two cases for whom no epidemiological link was found.

Since August 2015 and up to April 2016, besides the outbreak cases, the new B3 variant has also been identified in other cases with no epidemiological link to the outbreak. In total, it has been identified in 17 non-Roma cases in various cities in Lombardy and four cases in Emilia-Romagna in cities other than Parma and Piacenza (Figure 3).

All sequences are available in the MeaNS database (sequence ID numbers: 86275-86278, 86280-86281, 86283, 86285-86323, 89315-89325) [5,6].

Outside of Italy, identical sequences have been identified in Spain, Germany, and the United Kingdom in August to September 2015 (before the described outbreak, weeks 35–39/2015) and in Romania in January 2016 (week 3/2016).

### Control measures

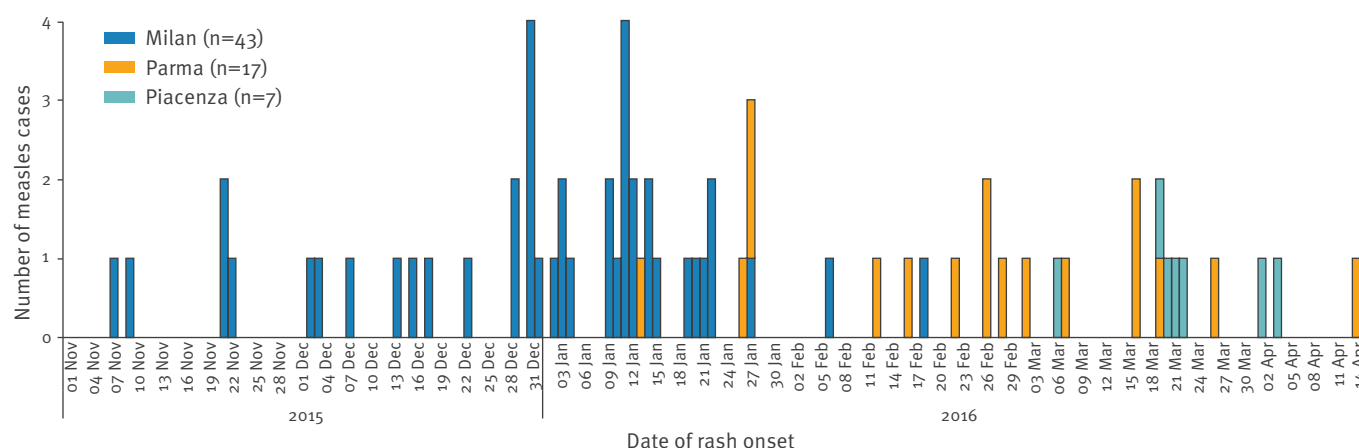
Staff of the local health authorities in Milan visited the three settlements on several occasions, together with cultural mediators, to identify contacts and search for additional cases. All persons 18 years or younger were invited to the vaccination centre for post-exposure prophylaxis. At the time of the visits, 54, 165 and 27 persons, respectively, were present in the three camps, of whom 52 (38 aged ≤18 years) received vaccination.

In Emilia Romagna, contacts were invited to be vaccinated. Overall 17 contacts were vaccinated (15 were older than 18 years).

In both regions, local health authorities informed local hospitals, emergency departments, family physicians/paediatricians and the local media of the outbreaks, and urged physicians to investigate all suspected measles cases, to isolate them if infectious and to notify

**FIGURE 2**

Number of reported measles cases by date of symptom onset, outbreak involving the Roma/Sinti population in Milan and spreading to Parma and Piacenza, Italy, November 2015–April 2016 (n = 67)



cases immediately. In Emilia Romagna, occupational physicians were reminded to implement the regional recommendation to verify measles immunity status of all unvaccinated hospital staff and staff of community healthcare services, and to offer vaccination to all those found to be susceptible. Results on uptake are pending at the time of publication.

## Discussion

This outbreak highlights the immunity gaps existing in Roma/Sinti population groups and HCW in Italy. Both are considered to be hard-to-reach populations and outbreaks have also been reported in Europe and other industrialised countries [7–9].

The Roma people are a heterogeneous nomadic ethnic group who live in many countries worldwide but primarily in Europe. An estimated 140,000 Roma live in Italy (ca 3,400 in Milan). It is difficult to monitor uptake of measles-mumps-rubella vaccine among the Roma, but it is known to be low [9]. In this outbreak, a high level of susceptibility was found among children (median age of cases: 4 years), confirming a low vaccination uptake. Barriers to vaccination among the Roma have been previously described [9].

In Milan, the outbreak in the Roma settlement did not spread extensively in the community and no nosocomial cases were reported, despite the high number of cases that sought medical treatment. On the other hand, in Emilia Romagna, transmission occurred mainly in the nosocomial setting, suggesting that vaccination coverage is low among HCW in this region. In both regions, measles vaccination uptake in 2015 was above the national average but below the target of 95% for both doses (first-dose coverage in two-year old children 89.5% and 88.3%, respectively, in Lombardy and Emilia Romagna; second-dose coverage in six year-old children 91% in both regions) [10].

Despite a thorough investigation, no source of exposure was found for some cases in Parma and Piacenza and phylogenetic analysis was essential to confirm connections among cases. It is likely that not all cases were detected by the surveillance system, the sensitivity of which should be strengthened by raising awareness of measles among physicians and increasing the rate of suspected cases being tested. However, it is known that many measles cases do not seek health care and this may also have affected sensitivity. Some cases were initially misdiagnosed, probably because many clinicians are no longer familiar with the clinical presentation of measles. They need to be reminded to consider measles in the differential diagnosis of any febrile rash illness, especially in unvaccinated persons, and to immediately report suspected case to public health authorities so that control measures can be implemented rapidly.

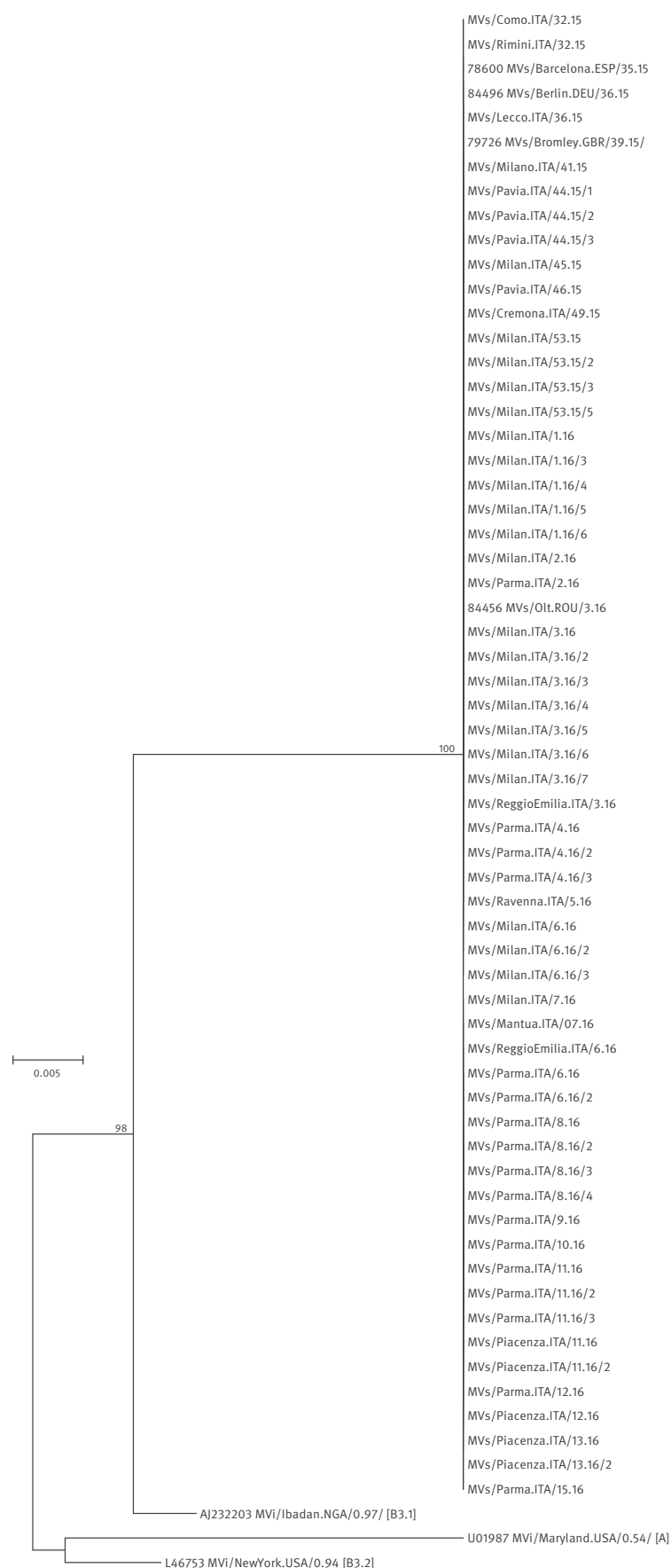
Three cases were detected among migrants living in one of the Roma settlements in Milan. It is possible that other undetected measles cases may have occurred among migrants living temporarily in the settlement. However, this is unlikely, as local health authorities, together with cultural mediators, visited the camp repeatedly during the outbreak and did not detect other cases. At the time of the outbreak, only a very small minority of persons living in one of the camps were migrants. These were persons of southern Asian and northern African origin who have access to medical care and immunisations in Italy.

MeaNS data from the last 12 months show that measles virus genotype B3 was endemic in several countries worldwide [6]. The appearance of a new B3 variant in Italy is probably due to importation, however, the route of introduction remains unknown.

To achieve measles elimination, besides strengthening surveillance and improving vaccination coverage in the general population, immunisation resources and

**FIGURE 3**

Neighbour-joining tree for nucleotide sequences of a new measles virus B3.1 variant causing an outbreak involving the Roma/Sinti population and the nosocomial setting, Italy, November 2015–April 2016 (n = 36)



The selected strains were aligned using BioEdit. Bayesian information criterion was used to determine the model of nucleotide substitution that best fit the data using the selection tool available in MEGA6. The model that best fit the data was the Kimura 2-parameter model.

tailored strategies should be directed at Roma communities and HCW. It has been suggested that greater vaccination uptake may be achieved among Roma children by inviting complete households for vaccination and by involving cultural mediators [11]. Also, control measures must be strengthened, including rapid case identification with implementation of airborne precautions in healthcare facilities to avoid nosocomial transmission [12].

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## Conflict of interest

None declared.

## Authors' contributions

A Filia coordinated surveillance activities and the epidemiological investigation and wrote the manuscript. A Amendola, S Bianchi, and E Tanzi performed genotyping and phylogenetic analysis of measles virus sequences isolated from cases in the Lombardy region and interpreted results. M Faccini, S Senatore, coordinated outbreak investigation and control activities in Milan (Lombardy). M Del Manso analysed data from the national measles surveillance system. A Ciampelli, MT Filippini carried out outbreak investigation activities in the nomadic camps. BM Borriani, MG Pascucci coordinated the epidemiological investigation in the Emilia Romagna region. T Lazzarotto, G Piccirilli performed genotyping and phylogenetic analysis of measles virus sequences isolated from cases in the Emilia Romagna region and interpreted the results. M Baggieri, F Magurano analysed measles virus sequences from the Lombardy and Emilia Romagna regions and performed phylogenetic analysis. F Magurano coordinated the virological investigation of the outbreak and supervised phylogenetic analyses. All authors reviewed and approved the final manuscript.

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# Global distribution and environmental suitability for chikungunya virus, 1952 to 2015

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Chikungunya fever is an acute febrile illness caused by the chikungunya virus (CHIKV), which is transmitted to humans by *Aedes* mosquitoes. Although chikungunya fever is rarely fatal, patients can experience debilitating symptoms that last from months to years. Here we comprehensively assess the global distribution of chikungunya and produce high-resolution maps, using an established modelling framework that combines a comprehensive occurrence database with bespoke environmental correlates, including up-to-date *Aedes* distribution maps. This enables estimation of the current total population-at-risk of CHIKV transmission and identification of areas where the virus may spread to in the future. We identified 94 countries with good evidence for current CHIKV presence and a set of countries in the New and Old World with potential for future CHIKV establishment, demonstrated by high environmental suitability for transmission and in some cases previous sporadic reports. *Aedes aegypti* presence was identified as one of the major contributing factors to CHIKV transmission but significant geographical heterogeneity exists. We estimated 1.3 billion people are living in areas at-risk of CHIKV transmission. These maps provide a baseline for identifying areas where prevention and control efforts should be prioritised and can be used to guide estimation of the global burden of CHIKV.

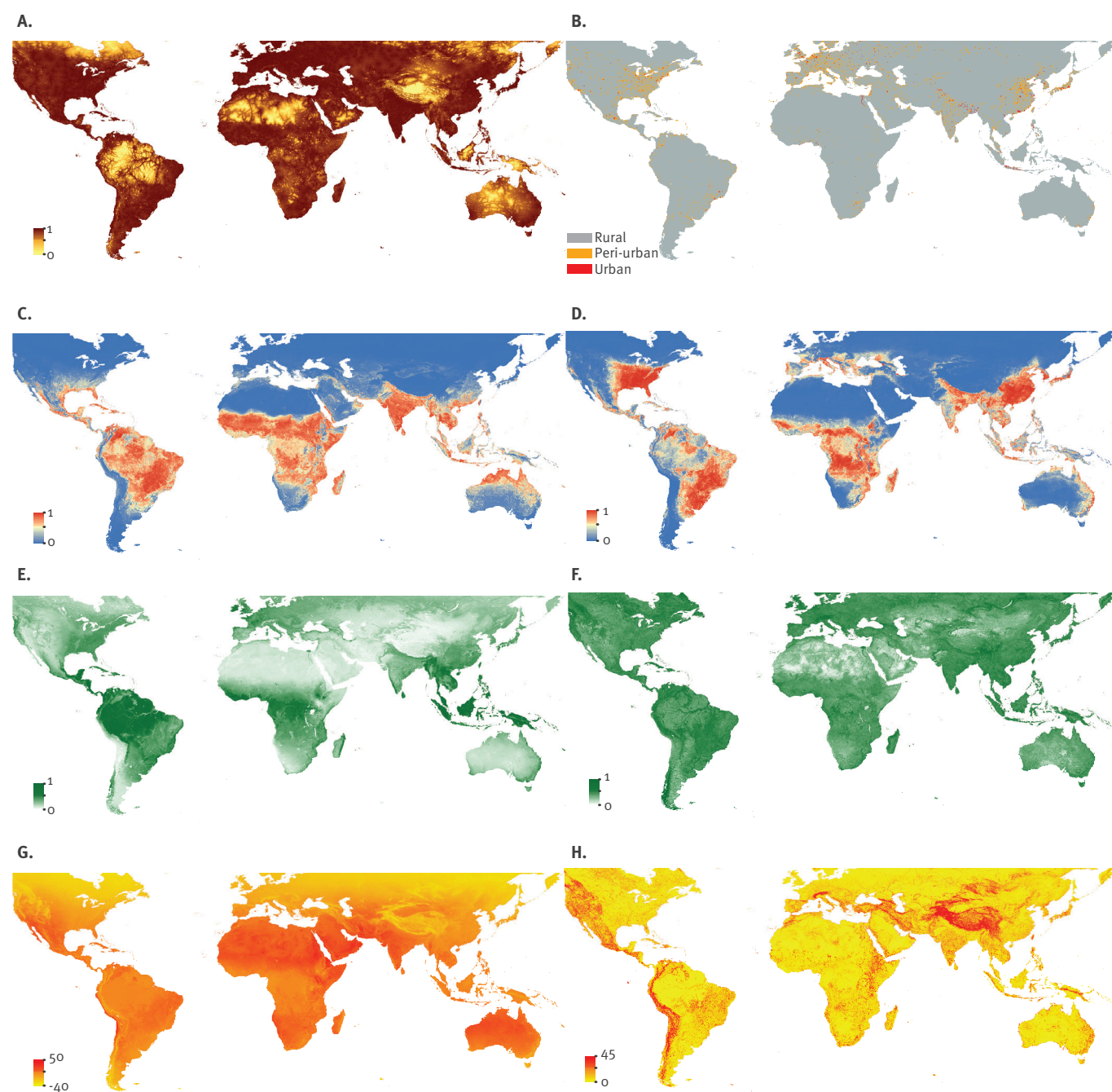
## Introduction

Recent emergence and re-emergence of chikungunya virus (CHIKV) in several regions globally underscores the importance of implementing and strengthening surveillance systems for rapid and accurate case identification [1]. CHIKV causes an acute febrile illness with severe arthralgia [2]. It is a mosquito-borne pathogen of the genus *Alphavirus* and transmitted to and between humans by *Aedes* mosquitoes with four circulating genotypes [3,4]. In the eastern part of Africa CHIKV is sustained in an enzootic, sylvatic cycle that involve arboreal mosquito vectors and non-human primates [5]. Clinical manifestations of the disease usually include fever, rash and arthralgia, which are similar to dengue symptoms, potentially leading to misdiagnosis and underreporting in the absence of specific laboratory diagnostic testing [6]. CHIKV and dengue also share the same primary vector *Ae. aegypti* and occasional co-infection has been reported [7]. Although it has been shown that also *Ae. albopictus* can transmit the virus between humans such outbreaks have been more localised [8]. Mortality from CHIKV infection is rare, infected persons can experience prolonged joint pain and arthritis over a period of months to years. There are also no antiviral agents or treatments for chikungunya fever (CHIK), but candidate vaccines are under development [9].

In 2013, the first autochthonous transmission of CHIKV in the western hemisphere was reported on Saint

**FIGURE 1**

Environmental covariates used in this study to predict the global environmental suitability of chikungunya virus transmission

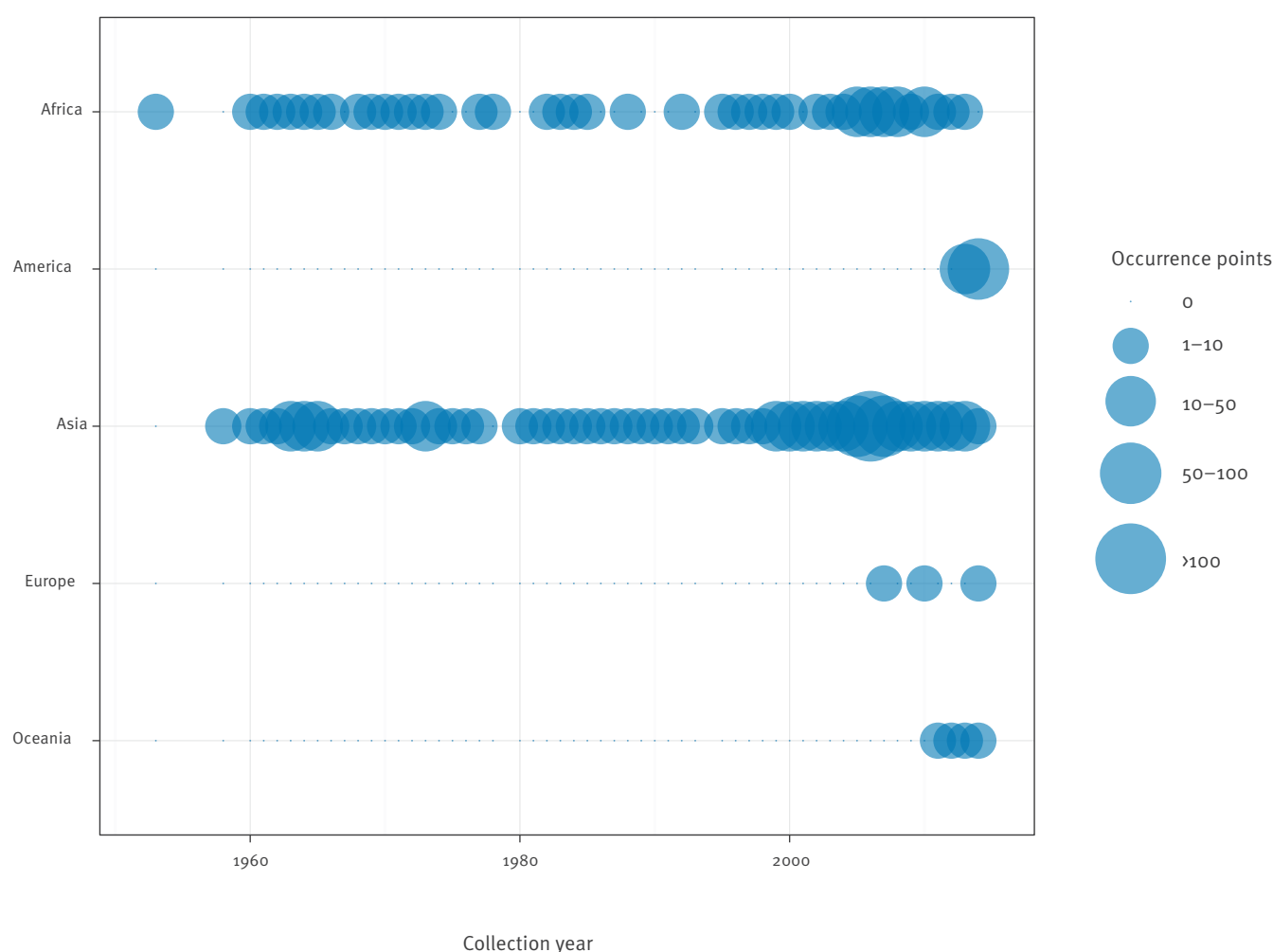


EVI: enhanced vegetation index; LST: land surface temperature.

A. Urban accessibility, with brown representing high relative accessibility to urban areas and yellow representing rural isolated areas; B. Urban, peri-urban and rural areas; C. *Aedes aegypti* suitability, with red representing high environmental suitability for the vector and blue representing low suitability; D. *Ae. albopictus* suitability, with red representing high environmental suitability for the vector and blue representing low suitability; E. EVI mean values, whereby dark green represents areas with year round vegetation growth and light green represents areas with low moisture levels; F. EVI range; G. LST mean values, whereby orange represents high temperatures and yellow lower relative temperatures; H. LST range, with orange representing high variation throughout an average year and yellow representing little annual variation.

**FIGURE 2**

Distribution per continent of chikungunya virus transmission occurrence points from peer-reviewed evidence and health organisations, 1952–May 2015



Martin Island in the Caribbean. The outbreak subsequently spread to several Caribbean islands and the wider Americas (including many countries in Central and northern South America), reaching over one million suspected and confirmed cases by December 2014 [10] due to the presence of a large naïve population and competent vectors. Despite an increasing global spatial distribution and clinical significance, detailed spatial information of the global geographical extent of CHIKV remains sparse. Previous work has mostly focused on assessment of CHIKV presence at a country level [1]. A comprehensive assessment of CHIKV transmission records globally is critical to identify knowledge gaps and regions where preventive and targeted control efforts should be prioritised. Additionally, this information can be used to guide estimation of the global clinical burden of CHIKV, which is currently unknown. Studies on the prediction of future trends and possibility of transmission in unaffected regions would also benefit from a thorough evaluation of the contemporary distribution and mapping of the extent of CHIKV on a fine spatial scale.

To assess the global distribution of CHIKV, we adopt established disease mapping approaches [11] previously used for dengue [12,13], Zika [14], and the leishmaniases [15]. We developed a comprehensive database of unique locations where CHIKV transmission has been reported and an evidence consensus score – a summary statistic of evidence for CHIKV presence or absence using a weighted scoring system [13]. We then couple these data with a comprehensive set of environmental correlates and contemporary *Aedes* distribution maps in a species distribution model to generate high-resolution risk maps (5km x 5km), representing environmental suitability for CHIKV transmission and to derive population-at-risk estimates. These maps and estimates can be refined as new data on disease and vector range expansion become available.

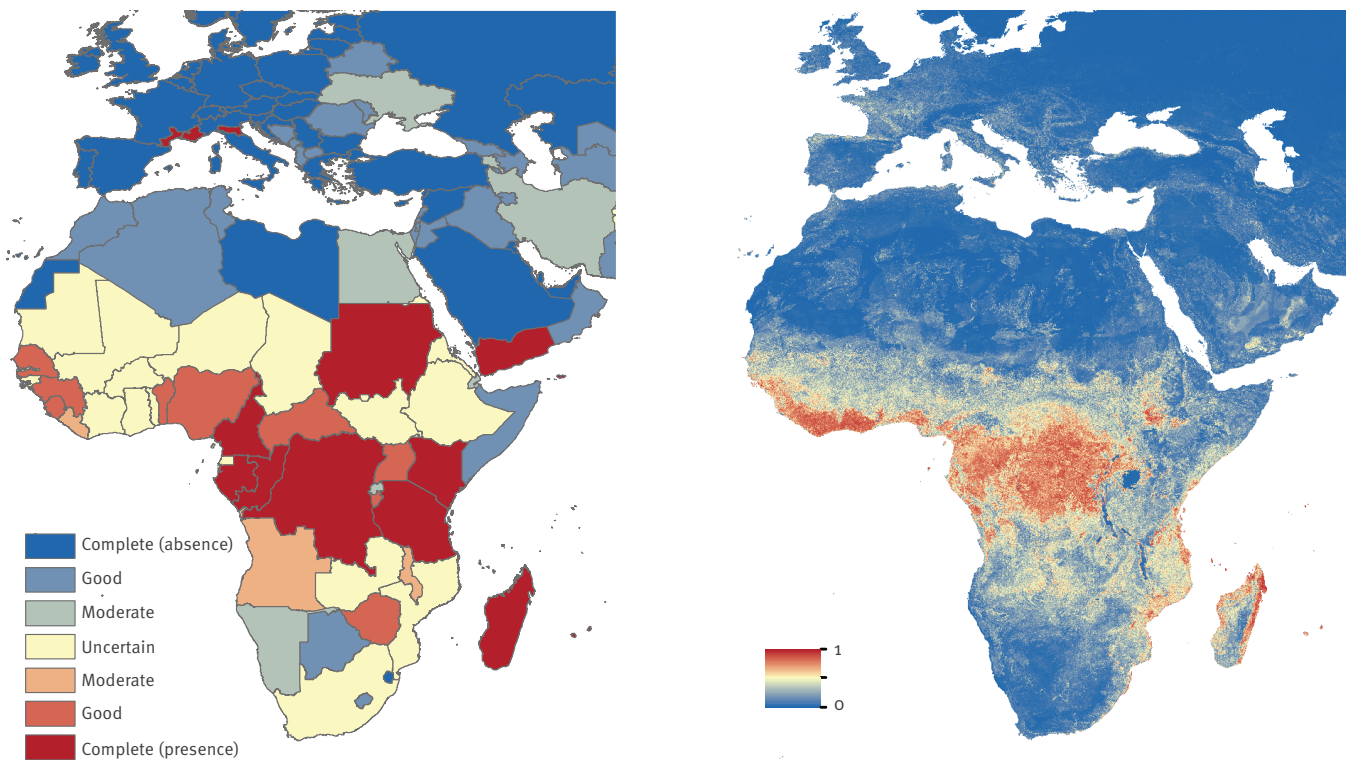
## Methods

### Occurrence records

Data on CHIKV transmission events were obtained from peer-reviewed literature, health organisations, and supplementary evidence such as, data on vector presence. A comprehensive review of peer-reviewed

**FIGURE 3**

Evidence consensus (a) and predicted environmental suitability (b) for chikungunya virus in Africa and Europe, as of May 2015



The colour legend for the evidence consensus ranges from red to blue, representing complete consensus on presence to complete consensus on absence. Indeterminate consensus is in yellow. Evidence consensus is presented at Admin1 level for Italy and France, and Admin0 level for all other countries. The predicted environmental suitability map is presented in similar colours with 1 and 0 representing most and least suitability for chikungunya virus, respectively.

literature was initially conducted on 4 April 2014 in PubMed using the search term 'chikungunya'. The data were subsequently updated every month until 7 May 2015. A total of 2,120 articles were identified. Abstracts of all articles were read to identify those that possibly included geographical information of clinical cases, epidemiological records or outbreaks of CHIKV. Information was extracted on: (i) date of occurrence, (ii) type of event (autochthonous vs imported cases), (iii) number of cases, and (iv) the geographical location (latitude, longitude and administrative level) following a standard protocol [16]. If people were diagnosed after returning from travel but the location of infection could be identified unambiguously due to information on the travel history, the data were retained. Of the 2,120 articles, 339 were used, resulting in 463 unique occurrence points.

We also consulted health organisation webpages, including the World Health Organization (WHO), Pan American Health Organization (PAHO), Global Infectious Disease and Epidemiology Network (GIDEON) and the United States (US) Centers for Disease Control and Prevention (CDC). Data on recent and ongoing outbreaks in the Americas were obtained from weekly PAHO epidemiological reports. Since PAHO is a WHO

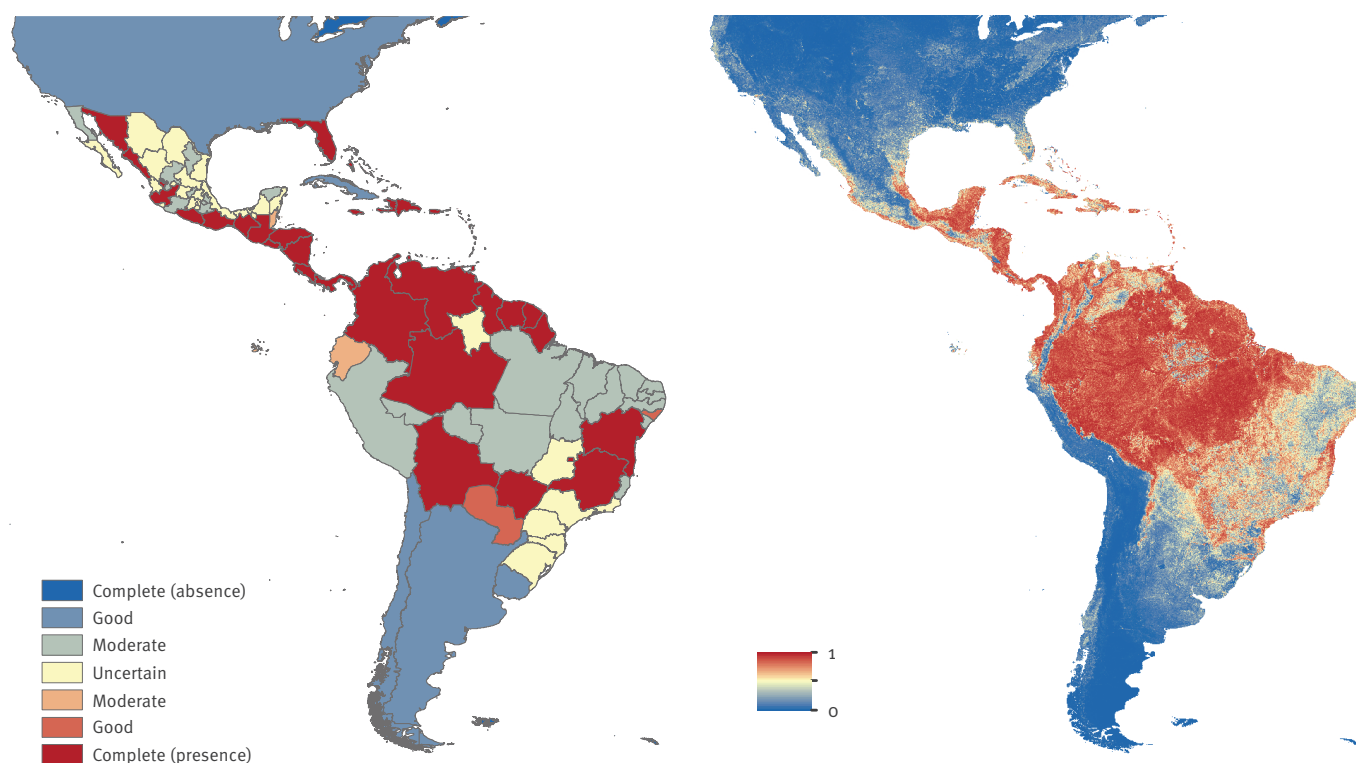
affiliate organisation, PAHO and WHO data were combined to represent a single health organisation data source resulting in 65 unique occurrence points.

Finally, 600 occurrence points based on reports of CHIKV outbreaks were obtained from the online surveillance system, HealthMap (<http://www.healthmap.org/en>).

Each location was then classified as precise (e.g. a town) or an administrative unit and the latter were linked to the appropriate polygon information from the Global Administrative Unit Layers dataset (<http://www.gadm.org>). Each occurrence point was attributed to a specific location (latitude and longitude) based on the level of detail presented in the data source. We used Google Maps (<https://www.google.co.uk/maps>) to support the geopositioning. The database subsequently underwent temporal and spatial standardisation using a standard protocol described elsewhere [16]. We removed any duplicate records so that the resulting database contained only one occurrence record per spatial entity. Further, any records reported from the above mentioned data sources that fell outside land borders (e.g. due to wrong geopositioning of the original source) were removed. Occurrence records

**FIGURE 4**

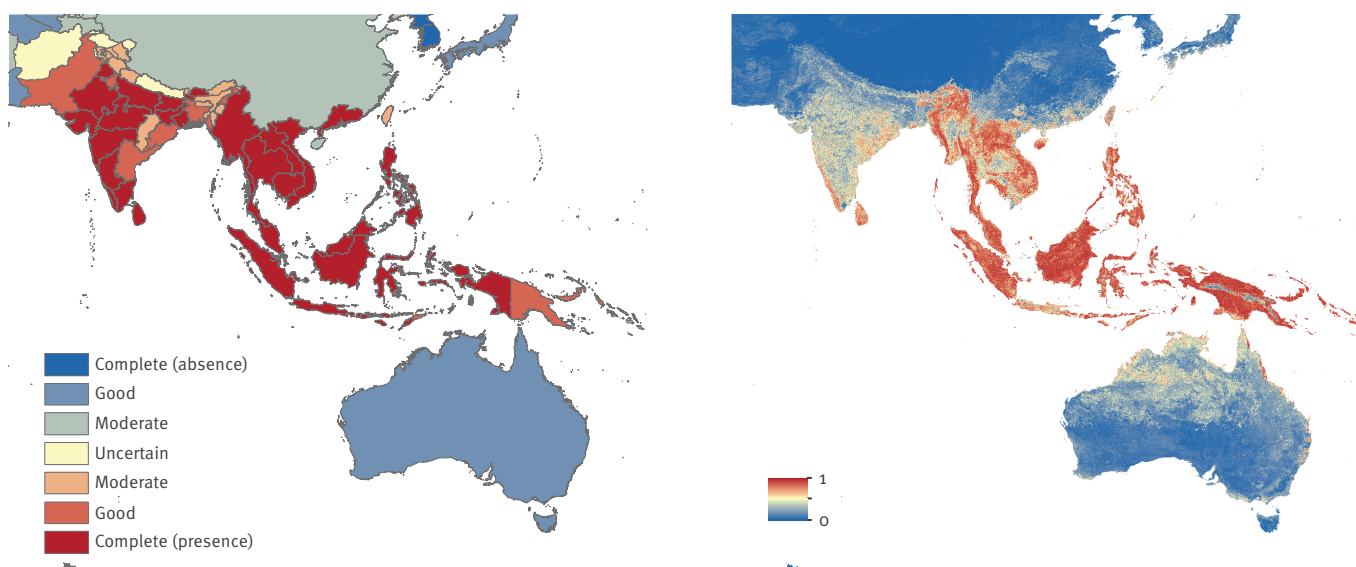
Evidence consensus (a) and predicted environmental suitability (b) for chikungunya virus in the Americas, as of May 2015



The colour legend for the evidence consensus ranges from red to blue representing complete consensus on presence to complete consensus on absence. Indeterminate consensus is in yellow. Evidence consensus is presented at Admin1 level for United States, Mexico, Argentina and Brazil, and Admin0 level for all other countries. The predicted environmental suitability map is presented in similar colours with 1 and 0 representing most and least suitability for chikungunya virus, respectively.

**FIGURE 5**

Evidence consensus (a) and predicted environmental suitability (b) for chikungunya virus in Asia and Oceania, as of May 2015



The colour legend for the evidence consensus ranges from red to blue representing complete consensus on presence to complete consensus on absence. Indeterminate consensus is in yellow. Evidence consensus is presented at Admin1 level for India and China, and country level for all other countries. The predicted environmental suitability map is presented in similar colours with 1 and 0 representing most and least suitability for chikungunya virus, respectively.

TABLE 1

Summary of evidence used for chikungunya virus transmission consensus scoring

Evidence category	Score
Health organisation status	
Number of health organisations <sup>a</sup> reporting epidemics or local transmission of chikungunya virus	
3	+6/-6
2	+3/-3
Peer reviewed evidence	
Date of chikungunya virus transmission occurrence	
2005–2015	3
1997–2004	2
Pre-1997	1
Diagnostic procedure	
Polymerase chain reaction (PCR)	3
IgM/IgG-based enzyme-linked immunosorbent assay (ELISA) and haemagglutination inhibition	2
No specified test	1
Combination score <sup>b</sup>	
3 types <sup>b</sup>	3
2 types <sup>b</sup>	2
1 types <sup>b</sup>	1
Case data or health expenditure	
Case data: most recent outbreak	
0–7 years	9
7–14 years	6
14–21 years	3
28–35 years	-3
≥35 years	-6
If no case data: health expenditure in 2014	
HE <100 USD + sporadic cases	6
HE <100 USD + sporadic cases	3
100 USD ≤ HE <500 USD	-3
HE ≥500 USD	-9
Supplementary evidence <sup>c</sup>	
Number of evidence types <sup>c</sup>	
4 types <sup>c</sup>	6
3 types <sup>c</sup>	4
2 types <sup>c</sup>	2

<sup>a</sup> The three health organisations considered included (i) the United States Centers for Disease Control and Prevention (CDC), (ii) the Global Infectious Disease and Epidemiology Network (GIDEON) and (iii) the World Health Organization (WHO) and Pan American Health Organization (PAHO) taken together.

<sup>b</sup> The types of evidences include (i) reports from health organisations, (ii) the date of chikungunya virus transmission occurrence, and (iii) the diagnostic procedure.

<sup>c</sup> The types of evidences include (i) mosquito presence, (ii) dengue presence, (iii) travel advisories, and (iv) HealthMap.

were then cross-checked manually and for each occurrence point, the probability of occurrence for both vectors was extracted (Figure 1c and d) and records deemed implausible (probability of occurrence for both vectors individually fell below 70%) [17] were removed. The cut-off at 70% was chosen based on a qualitative interpretation of the *Ae. aegypti* and *Ae. albopictus* probability of occurrence maps, where >90% of the geographical spaces with non-zero probabilities were recorded between vector presence 70% to 100%.

### Evidence consensus

The evidence consensus is a summary statistic representing confidence in the presence or absence of CHIKV for a given political region. We calculated evidence consensus scores at a national level for all countries apart from Argentina, Brazil, China, France, India, Italy, Mexico, and the US, where sufficient detailed epidemiological information was available to distinguish within-country epidemiological, environmental, and economic variability. Smaller countries were not considered for subnational assessment.

**TABLE 2**

Relative contribution of each covariate in explaining the global environmental suitability of chikungunya virus and 95% confidence interval, 1952–May 2015

Variable	Mean (95% confidence interval)
<i>Aedes aegypti</i>	46.16 (43.45–48.74)
EVI mean and range	33.37 (29.71–36.94)
Urban accessibility	7.42 (6.52–8.50)
<i>Ae. albopictus</i>	6.70 (5.78–7.60)
LST mean and range	5.98 (4.83–6.90)
Urbanisation	0.30 (0.15–0.70)

EVI: enhanced vegetation index; LST: land surface temperature.

Each evidence category was scored independently and category weights applied to reflect the level of detail each category provides: health organisation status (max. score 6), peer-reviewed evidence (max. 9), case data (max. 9) and supplementary evidence (max. 6). For example, if all three health organisations considered (i.e. PAHO/WHO, GIDEON and CDC) agreed on presence/absence of CHIKV, the country was assigned a score of +6/-6. If two health organisations agreed on presence/absence, the country was assigned a score of +3/-3. For each country, we identified peer-reviewed evidence to confirm CHIKV presence based on seroprevalence studies, polymerase chain reaction (PCR) confirmation, case reports and local outbreaks. Each published study was scored independently based on date of disease occurrence and the average was taken and added to a score of accuracy, which was assigned based on diagnostic procedure. The case data category was scored based on the most recent outbreak year and pre-defined inter-epidemic period intervals to account for herd immunity [18,19]. In the absence of case data, healthcare expenditure (HE) was used as a proxy to indicate confidence in a country's ability to detect and report CHIKV cases and to supplement this category [13]. In addition, countries for which HE data were unavailable were assigned low HE, and overseas territories were assigned the same HE as their parent nations.

For areas with conflicting reports from the input sources and recent geographical expansion (e.g. Mexico and Brazil), database records were cross-checked against expert knowledge and recent national reports. Supplementary evidence was extracted from peer-reviewed literature on mosquito presence, dengue presence, travel advisories (<http://www.nathnac.org/travel>), and the HealthMap database (<http://www.healthmap.org>) and scored based on number of evidence types. The HealthMap database contains data extracted from online news sources and alerts from official public health sources (e.g. ministries of public health) and was available from 2006 to 2014. We also used data on the global evidence consensus on dengue transmission from Brady et al. [13] to supplement

data on the presence of other arboviruses for each country. See Table 1 for a summary and Brady et al. [13] for additional details on the scoring process.

The scores from all previously described evidence categories were summed, divided by the maximum potential score, 30 and multiplied by 100. The evidence consensus was then mapped based on seven equidistant categories: complete evidence for presence ( $>71.42$  to  $\leq 100$ ) or absence ( $>100$  to  $\leq 71.42$ ), good evidence for presence ( $>42.84$  to  $\leq 71.42$ ) or absence ( $>71.42$  to  $\leq 42.84$ ), moderate evidence for presence ( $>14.27$  to  $\leq 42.84$ ) or absence ( $>42.84$  to  $\leq 14.27$ ), and indeterminate evidence ( $>14.27$  to  $\leq 14.27$ ).

## Environmental risk maps

A boosted regression tree (BRT) modelling approach was applied to derive probabilistic global environmental risk maps for CHIKV. BRT models combine the strengths of regression trees with boosting and are frequently used in species distribution modelling (SDM) [20] with the overall aim to identify areas that exhibit similar conditions to areas where the disease has been reported. These statistical models heavily depend on the input occurrence database. We included the following globally available environmental and socioeconomic covariates in our models: (i) probabilistic global niche maps for *Ae. aegypti* and *Ae. albopictus* [17]; (ii) land surface temperature (LST); (iii) a categorical differentiation in urban, peri-urban and rural environments, as well as their accessibility; and (iv) enhanced vegetation index (EVI).

### *Aedes aegypti* and *Aedes albopictus* suitability

We constructed probabilistic global *Aedes* risk maps based on the most comprehensive globally representative occurrence dataset for both *Ae. aegypti* and *Ae. albopictus* species and a set of contemporary environmental covariates explaining their global distribution (Figure 1c and d) [17,21].

### Land surface temperature (LST)

Temperature is one of the most important determinants of *Aedes* survival [22]. We used MODIS daytime LST after processing through a gap-filling algorithm to account for variation in temperature globally [23] (Figure 1g and h). Additionally, transmission of CHIKV is dependent on the persistent presence of the disease vectors, *Ae. aegypti* and *Ae. albopictus* mosquitoes. Both adult female longevity and length of first gonotrophic cycle are temperature dependent. We used the combined temperature suitability of population persistence for both species to identify areas where by proxy we expect the geographical limits of transmission of CHIKV to be located [13,15]. The binary outputs of this model were used as a mask to exclude the placement of 10,000 pseudo-absences inside their physiologically plausible range.

### Enhanced vegetation index (EVI)

Water availability is another important factor for CHIKV transmission, as it has been shown, for example, that mosquitoes may cluster around houses if containers with standing water due to precipitation are present [24]. EVI measures vegetation canopy greenness and can be used as a proxy for soil surface-level moisture that are associated with the presence of vector larval development sites [25]. We used range and mean values of MODIS EVI after processing through a gap-filling algorithm described in Weiss et al. [23] (Figure 1e and f).

### Urbanisation

To account for differences in urban, peri-urban and rural environments we built a categorical variable by supplementing the projected 2010 Global Rural Urban Mapping Project (GRUMP) urban rural categories with land-cover classes using night-time light satellite imagery and population density based on the most up-to-date national censuses available at the smallest available administrative unit [26]. A gridded surface of 5 km x 5 km cells was generated with each pixel representing urban, peri-urban, or rural areas (Figure 1b).

### Urban accessibility

Human movement defines the spatial scale of transmission of most communicable diseases. To account for areas that are highly accessible, we used a friction surface that shows pixel level travel time through the respective region of interest [27] (Figure 1a).

We then applied an ensemble BRT using 180 sub-models to derive probabilistic estimates and underlying uncertainty at a 5 km x 5 km resolution. We also estimated populations-at-risk of CHIKV transmission. For each occurrence record we identified the maximum risk probability of transmission within a buffer of 10 km around the occurrence record to derive a threshold value of probability of occurrence that should be included in the population-at-risk estimate. We then converted the continuous surface of transmission risk into a binary at-risk/not-at-risk classification. Finally, we extracted the population living in areas of potential transmission using a global population surface.

## Results

The data abstraction yielded 528 occurrence points from peer-reviewed evidence and health organisations. The distribution of occurrence points by region is shown in Figure 2. Individually, WHO (+PAHO), CDC and GIDEON implicated 60, 96, and 70 countries with reported CHIKV transmission, respectively. However, based on evidence from all available sources, we identified 94 countries with good or better evidence consensus on CHIKV presence from which 47% were in Asia and Africa and 44% in the Americas. Of the 94 countries, at total of 24, 41, 20, two and seven were located in Africa, the Americas, Asia, Europe and Oceania, respectively.

High probability of *Ae. aegypti* occurrence was the strongest predictor for environmental suitability with a mean contribution of 46% (Table 2). Based on our environmental suitability predictions, we estimate that 1.3 billion people are living in areas of potential CHIKV transmission.

Global consensus on CHIKV presence or absence based on the evidence consensus scoring system and predictions of environmental suitability for CHIKV are further presented in for Africa and Europe, the Americas, and Asia and Oceania, respectively. The evidence ranges from complete presence to complete absence.

### Africa and Europe

Most of the African countries with reliable evidence on presence and absence were located in Central and North Africa, respectively. The strong evidence for CHIKV presence in Central Africa was aided by in-country seroprevalence studies such as in Cameroon and Gabon and confirmed imported cases to non-endemic regions [28,29]. There was insufficient data to determine presence or absence for some countries in Africa such as Ghana, Ethiopia, and Mozambique, due to a general lack of data and poor surveillance. South Africa, for example, had a reported outbreak in 1977 [30], but there have been no new reports of outbreaks or seroprevalence studies. Most countries in Central and parts of West Africa with strong evidence on CHIKV presence also demonstrated high environmental suitability for CHIKV (Figure 3b). We estimated that over 240 million people live in areas at risk for CHIKV transmission in Africa.

Autochthonous transmission of CHIKV in Europe has been reported in Ravenna, northern Italy in 2007 and in the south-eastern French city of Fréjus in 2010 and Montpellier, southern France in 2014 (Figure 3a). Prior to these cases, CHIKV in Europe was limited to travel-related cases. Genomic micro-evolution of CHIKV in Reunion facilitated transmission by *Ae. albopictus* [31] in strains belonging to the so called Indian Ocean lineage (IOL) which increased the likelihood of CHIKV outbreaks in Europe. We predicted moderate environmental suitability for parts of France, Spain, Germany and Italy (Figure 3b), which is in agreement with previous projections of increased risk of CHIKV for France, northern Italy and the Pannonian Basin [32].

### Americas

Systematic reporting of confirmed cases to PAHO provided well-documented evidence for CHIKV occurrence in the Americas. As of 13 March 2015, 45 countries had reported local transmission of CHIKV, while the evidence consensus identified 90% of countries and island states with good or better consensus (Figure 4a). Laboratory confirmation of autochthonous cases made a major contribution in defining the South and Central America's higher consensus cluster on presence. The evidence strength varied across states for Brazil and Mexico. Brazil had strong evidence on presence in

south-central states; indeterminate evidence for several southern states and Amazonas state in the north; and moderate evidence in central and northern states. In contrast, the environmental suitability map predicted that coastal Brazil and areas in the Amazon were highly suitable for CHIKV transmission. Peru and states in Brazil where evidence currently suggest absence, demonstrated a moderate to high suitability for CHIKV (Figure 4b).

Our models identified *Ae. aegypti* as the strongest predictor of CHIKV suitability, which is the primary vector implicated in the outbreak in most of the Caribbean and the Americas [33,34]. Based on the environmental suitability predictions, we estimated that over 260 million people live in areas at risk for CHIKV transmission in the Americas.

### Asia and Oceania

CHIKV presence is well documented for most countries and islands in south and south-east Asia. Persistent vector populations and naïve populations contributed to notable pre-2000 CHIKV outbreaks in India, Myanmar/Burma, Philippines, Sri Lanka, Thailand and Vietnam [35,36]. In the last decade, outbreaks have been reported in India, Indian Ocean islands, Indonesia, Sri Lanka, and Thailand, following a 41-year period of quiescence [37,38]. Good to better consensus on CHIKV presence was reported for 80% of Asian countries and clusters of complete consensus were observed ranging from Myanmar/Burma to Vietnam for south Asia and Indonesia to Papua New Guinea for south-east Asian islands (Figure 5a). Complete consensus on CHIKV presence was noted for Guangdong province, China, where an outbreak was reported in 2010 [39]. With the exception of Chhattisgarh in Central India and smaller states in the north-east and north-west, most Indian states had good consensus on CHIKV presence. There was insufficient data on presence or absence of CHIKV in Nepal. Despite reports of large outbreaks in neighbouring India and indication of possible local transmission, there are no published national seroprevalence surveys for Nepal and CHIKV cases are possibly misdiagnosed as typhoid or other bacterial or viral fevers [40].

The environmental suitability map strongly complemented the evidence consensus with high risk of CHIKV transmission predicted for most south-east Asia islands and parts of south-east Asia, and moderate risk predicted for India (Figure 5b). Predictions of suitability for CHIKV transmission in China were patchy, with high risk predicted for regions where local CHIKV transmission has been observed. High prevalence has been noted for other arboviral diseases in south-east coastal regions, and northern and inland regions [41,42], signalling suitability and risk for CHIKV transmission. We estimated that over 270 million people live in areas at risk for CHIKV in Asia.

## Discussion

We present the global distribution and predict environmental suitability for CHIKV transmission. In this analysis we combined extensive evidence available at multiple geographical scales and integrated them into a well-established disease mapping approach. We identified areas particularly in Africa where CHIKV status is uncertain (e.g. Ethiopia, Mozambique) although there is high environmental suitability, presence of disease vectors and reported outbreaks in neighbouring countries such as Tanzania. We also predicted high environmental suitability for some regions but observed moderate evidence for absence of local transmission, such as in the Amazon basin. The former could be attributable to inadequate surveillance or misdiagnosis, while the latter could be explained by the recent emergence of CHIKV and the endemicity of dengue in these regions. Our comprehensive assessment of these evidence gaps enables the identification of areas where surveillance, seroprevalence studies, and vector control measures are increasingly important [43].

Despite the growing economic and public health impact of CHIKV outbreaks, there have not been any thorough assessments of knowledge gaps or high-resolution distribution maps of the disease. This is the first comprehensive assessment of the current spatial extent and environmental suitability of CHIKV transmission globally. We considered extensive available evidence from 1952 when CHIKV was first identified in Tanzania. We observed the following: (i) CHIKV evidence is dynamic, re-emerging and due to its high attack rate likely to cause outbreaks with significant timelags; (ii) misclassification and delay in reporting may influence the perception of current vs. former presence; (iii) while there is strong evidence for some areas (e.g. Asia), data on CHIKV occurrence in many parts of Africa remain sparse.

Similar to dengue [44], CHIK appears to be an under-recognised problem in Africa. Our environmental suitability maps indicate moderate to strong risk for CHIKV transmission in most of West, Central and East African countries. However, there is insufficient data to determine presence or absence for several countries in West Africa and parts of East Africa. Seroprevalence surveys are needed for countries with indeterminate status.

Additionally, co-occurrence and comorbidity of CHIKV and other arboviruses have been reported, which stresses the importance for better diagnostic tests. Furthermore, since dengue and CHIKV share the same primary vectors, similar estimates for population-at-risk would be expected. Population at risk of dengue virus transmission has been estimated at 3.97 billion people [13], which is far more than the 1.3 billion predicted for CHIKV. However, the estimates for dengue were derived on a national level, while our estimates are at a 5 km x 5 km spatial scale.

Improvements, such as the identification of the primary vector for each occurrence record can be made as new data become available. Particularly in Africa and Micronesia it is hypothesised that other mosquito vectors (*Ae. africanus*, *Ae. luteocephalus*, *Ae. furcifer-taylori* and *Ae. hensilli*) are responsible for transmitting CHIKV between humans [45]. Data on their geographical distribution however remains sparse and could not be integrated in the current analysis. Furthermore, since the maps are derived using long-term average covariate layers, they identify areas that are at risk of transmission at any time during the year but do not consider explicitly how seasonality or human mobility affects transmission; these factors influence the short and long-term dynamics of CHIKV transmission. In addition, there are a number of other factors that may influence the probability of infection that relate to social aspects, such as, housing quality, poverty, susceptibility of the population, and the use of air-conditioning. Such factors were not included in our study since no reliable data are available on a global scale.

Furthermore, there are some challenges in developing a global model to predict environmental suitability for CHIKV transmission. For instance, although there have been reports of major outbreaks of CHIKV in India, the environmental suitability models predicted moderate environmental suitability for CHIKV in these regions. The spatial variability observed for India is similar to the *Ae. albopictus* distribution map [17]. This suggests that our modelling approach strongly mirrors *Ae. albopictus* distribution in India, which has been implicated in recent outbreaks, but fails to capture the distribution of other covariates. Similarly, in Europe where there have been documented outbreaks of CHIKV in France, and Italy, risk appears to be low in our prediction. Modelling the likely distribution of CHIKV based on *Ae. albopictus* which is well distributed in southern Europe could help refine our approach. However, as transmission of CHIKV is relatively low in Europe, our results indicate the likely lower relative global contribution of *Ae. albopictus* compared with *Ae. aegypti*. Additionally, our maps indicate high transmission risk in small island states that have experienced large outbreaks in the last decade. However, herd immunity was not incorporated into the environmental risk models, implying that small countries or islands with (>50% attack rates) might have a lower risk of CHIKV outbreaks despite environmental suitability. These observations highlight the need for region or country-specific modelling, which would be most beneficial for local public health policy decisions. These maps can however be used as baseline layers for future projections of CHIKV and compared with global maps of dengue [12].

Despite these limitations, the usefulness of these maps cannot be understated, especially given the CHIKV's currently expanding range in the Americas and elsewhere. By synchronising the evidence consensus, environmental suitability prediction maps and population

at-risk estimates, areas most in need of CHIKV surveillance resources were identified. It is equally important that these maps are updated as soon as new information becomes available, such as recent reports of local CHIKV transmission in states of Brazil, and Peru and this will be achieved by incorporating the results of this study into the Atlas of Baseline Risk Assessment for Infectious Diseases (ABRAID, at [www.abraid.ox.ac.uk](http://www.abraid.ox.ac.uk)).

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## Conflict of interest

None declared.

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## Authors' contributions

Elaine Nsoesie and Moritz Kraemer performed data collection and analysis, and drafted the manuscript. Catherine Moyes reviewed data for accuracy. Michael Johansson served as the expert reviewer. Nick Golding, Oliver Brady and David Pigott advised on methods. John Brownstein and Simon Hay provided resources. All authors contributed to the writing and editing of the manuscript.

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# European Commission publishes evaluation of the 2nd Health Programme 2008–2013

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On 10 May 2016, the European Commission published an evaluation report [1] on the main outcomes of the European Commission 2<sup>nd</sup> Health Programme (2008–2013) [2].

The evaluation considered programme management, dissemination practices, impact, and synergies with other programmes and services and it was based on 13 case studies including five projects, five joint actions and three tenders. It found that the Programme delivered outputs with a clear link to European Union (EU), as well as national, health policy priorities which for example in areas such as rare diseases, cardiovascular diseases and safety of organs for donation, had added value to the EU. The evaluation also found that (i) the dissemination of action outputs varied, (ii) reaching key stakeholders was not systematically ensured and (iii) underlined that transforming outputs into results and tangible impacts could be improved.

The overall aim of the Programme was to ‘complement, support and add value to Member States’ policies and contribute to increased solidarity and prosperity in the EU by protecting and promoting human health and safety and improving public health [1].’ The Programme financed actions aiming to achieve objectives under three main headings:

- improving citizens’ health security and protecting them from health threats and emergencies, such as pandemics or natural disasters;
- promoting health and reducing health inequalities across Europe, whether relating to lifestyle, such as access to opportunities for physical activity, or to healthcare, such as access to the necessary medical intervention; and
- generating health information and health knowledge and disseminating them to relevant parties, from the general public to policymakers and health professionals.

The findings and recommendations from the evaluation will be taken into account to improve the implementation of the 3<sup>rd</sup> Health Programme (2014–2020) [3]. Read more about the 3<sup>rd</sup> Programme [here](#).

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