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Dengue, Chikungunya and West Nile Virus in North Africa: Documented Transmission and Limits of Detection

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A B S T R A C T

Evidence on dengue, chikungunya and West Nile virus (WNV) in North Africa has been synthesised one virus at a time or at continental scale. This narrative review with a structured search brings all three together for Morocco, Algeria, Tunisia, Libya, Egypt and Sudan, and asks whether few reported cases reflect under-detection or low transmission. PubMed/MEDLINE, Scopus and Google Scholar were searched from 1940 to 3 October 2026, with WHO reports and citation chaining; each source was assigned one of five evidence levels and claims worded to match. WNV transmission is documented in Egypt, where the virus was isolated from human sera in 1951, and in Algeria, Tunisia, Morocco and Libya, with recurrent neuroinvasive outbreaks in Tunisia (1997–2023) and Algeria (1994). The reported excess of North African seroprevalence over Southern Europe (15.4% against 2.7%) rests largely on assays without neutralization confirmation; where both were measured, confirmed values were lower (6.7% against 9.8% in Algiers; 10% against 25% in Tunisian horses in 2023). Dengue and chikungunya transmission is documented in Sudan, including all four dengue serotypes in Kassala in 2023–24, and in Egypt, in Upper Egypt and on the Red Sea and Sinai coasts. Neither has been reported as locally acquired in the Maghreb or Libya, although *Aedes albopictus* has been present there for 8 to 16 years and transmits chikungunya virus in the laboratory. Reported case counts cannot distinguish low transmission from non-detection. The study most needed is a neutralization-confirmed serosurvey of these two viruses in resident Maghreb populations.

حمى الضنك والشيكونغونيا وفيروس غرب النيل في شمال أفريقيا: الانتقال الموثَّق وحدود الكشف

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الكلمات المفتاحية:
الفيروسات المنقولة بالمفصليات
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الانتشار المصلي
فاشيات الأمراض
الترصد الوبائي
شمال أفريقيا

الملخص

جُمِعت الأدلة المتعلقة بحمى الضنك والشيكونغونيا وفيروس غرب النيل في شمال أفريقيا فيروسًا تلو الآخر، أو على نطاق القارة. تجمع هذه المراجعة السردية المنظّمة الفيروسات الثلاثة في المغرب والجزائر وتونس وليبيا ومصر والسودان، وتتساءل: هل تعكس قلة الحالات المُبلَّغ عنها قصورًا في الكشف أم انخفاضًا في الانتقال؟ بُحث في قواعد بيانات PubMed/MEDLINE وScopus وGoogle Scholar للمدة من عام 1940 حتى 3 أكتوبر 2026، إلى جانب تقارير منظمة الصحة العالمية وتتبّع المراجع، وصُنِّف كل مصدر في أحد خمسة مستويات للدليل. انتقال فيروس غرب النيل موثَّق في مصر، حيث عُزل الفيروس من أمصال بشرية عام 1951، وكذلك في الجزائر وتونس والمغرب وليبيا، مع فاشيات متكررة للمرض العصبي الغازي في تونس (1997–2023) والجزائر (1994). أما تفوّق الانتشار المصلي في شمال أفريقيا على نظيره في جنوب أوروبا (15.4% مقابل 2.7%) فيستند في معظمه إلى مقايسات لم تُؤكَّد باختبار التعادل؛ وحيثما قيس الاثنان معًا جاءت القيم المؤكَّدة أدنى: 6.7% مقابل 9.8% في الجزائر العاصمة، و10% مقابل 25% لدى الخيول في تونس عام 2023. وانتقال حمى الضنك والشيكونغونيا موثَّق في السودان، بما في ذلك سريان الأنماط المصلية الأربعة لفيروس الضنك في كسلا خلال 2023–2024، وفي مصر، في صعيد مصر وعلى ساحلي البحر الأحمر وسيناء. ولم يُبلَّغ عن إصابة محلية المنشأ بأيٍ منهما في المغرب أو الجزائر أو تونس أو ليبيا، مع أن بعوضة Aedes albopictus موجودة هناك منذ 8 إلى 16 عامًا وتنقل فيروس الشيكونغونيا مخبريًا. وأعداد الحالات المُبلَّغ عنها لا تكفي للتمييز بين انخفاض الانتقال وغياب الكشف. والدراسة الأشد حاجةً هي مسح مصلي لهذين الفيروسين مؤكَّد باختبار التعادل بين سكان المغرب العربي المقيمين.

1. Introduction

Dengue reached a record of more than 14 million reported cases in 2024, with 9,508 deaths and a global case-fatality rate of 0.07% [1]. Between January and September 2025, more than 445,000 suspected and confirmed cases of chikungunya were reported from 40 countries

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and territories, and the World Health Organization (WHO) warned that gaps in surveillance and diagnostic capacity may themselves contribute to spread into new areas [2]. West Nile virus (WNV) transmission in Europe peaked in 2018, when the season exceeded the total for the seven preceding years combined [3].

The southern shore of the Mediterranean shares a sea, at least one invasive vector and, along the coast, a Mediterranean climate with the northern countries where recent transmission has occurred. European data show how quickly outbreaks can follow vector establishment. Between 1990 and 2024, the interval from the first documented establishment of *Aedes albopictus* in a European Union region to the first autochthonous dengue or chikungunya outbreak there fell from 25 years to under 5, and each 1 °C rise in mean summer temperature carried a hazard ratio of 1.55 (95% CI 1.30–1.85) after adjustment for health-care expenditure, imported cases and land use [4]. The same group has since projected a northward expansion of that risk [5]. The 2007 chikungunya outbreak in Italy showed that a single viraemic introduction into an *Ae. albopictus*-infested, immunologically naive population could produce a large outbreak in a temperate setting [6]. Whether North Africa is following the same course is unresolved.

The evidence for North Africa has been reviewed in parts: dengue [7] and chikungunya [8] across the Middle East and North Africa; dengue in Egypt alone [9]; WNV in the Eastern Mediterranean region [10], in Africa [11,12] and in the Mediterranean Basin [13]; arboviral emergence risk in Morocco alone [14]; and sandfly-borne phleboviruses [15]. The closest in scope is the review of mosquito vectors and mosquito-borne diseases in North Africa by Nebbak and colleagues [16], which covers all six countries and lists 26 vector species and seven mosquito-borne diseases. It is organized by vector and by disease, not by strength of evidence, so a serological signal from an unconfirmed assay sits beside a laboratory-confirmed outbreak. It also predates the literature published since 2022, which includes the Sudanese outbreaks of 2022 and 2023–24, the Tunisian outbreak of 2023, the Algerian mosquito screening and the Egyptian vector surveys drawn on here. The other reviews each take one virus, one vector group, one country, or an area wider than North Africa. None covers the three viruses together across these six countries, and none sorts the evidence for each country by what it can and cannot show.

The question matters because two observations are often read as evidence of unrecognized transmission: that North Africa has the highest pooled WNV seroprevalence but the fewest reported human cases in the Mediterranean Basin [13], and that dengue and chikungunya have not been reported in the Maghreb despite the arrival of a competent vector. The evidence behind both is mixed: serosurveys with assays of differing specificity, single-country outbreak reports, laboratory competence experiments and models. Each supports a different kind of conclusion, yet they are often cited as if they supported the same one.

This review organizes the published evidence on dengue, chikungunya and WNV in Morocco, Algeria, Tunisia, Libya, Egypt and Sudan by virus and by type of evidence, marks the limits of each type of evidence, and assesses whether the small number of reported cases in the region is better explained by under-detection or by low transmission. It is written for the public health laboratories, arbovirus surveillance programmes and infectious disease clinicians of the six countries, and for the entomologists, veterinary services and travel medicine practitioners whose data the argument depends on. The search and the evidence-level scheme are described in Section 2. Section 3 presents the evidence by virus, then by vector and by the constraints on detection, and Section 4 weighs the two competing explanations before turning to limitations and research gaps.

2. Materials and Methods

2.1. Design and scope

This is a narrative review with a structured search. It borrows the aim of mapping heterogeneous evidence set out by Arksey and O'Malley [17], but no protocol registration, record counts or flow diagram are available for it, so conformity with PRISMA-ScR [18] is not claimed. The sources range from outbreak reports and serosurveys to entomological surveys, laboratory competence experiments, modelling studies and surveillance bulletins. Their estimates are not commensurable, so no pooled estimate was calculated and no formal risk-of-bias instrument was applied. Pooled estimates published by others are reported as such and attributed. North Africa follows the United Nations geoscheme for Northern Africa, which includes Sudan. Sudan has no Mediterranean coast; it is included because it has had the region's largest documented dengue and chikungunya epidemics, and because it forms with Egypt's Red Sea coast the eastern part of the region. Western Sahara also belongs to that grouping but is not treated separately here, because no source retrieved for this review reports arbovirus or vector data specific to the territory; the few records that exist are reported within Morocco. The Maghreb refers here to Morocco, Algeria and Tunisia.

2.2. Search

PubMed/MEDLINE, Scopus and Google Scholar were searched iteratively between July and October 2026, without language restriction, for publications from 1940 to 3 October 2026, the date of the final search. The lower bound is the year in which the first isolate of WNV, obtained in Uganda in 1937, was described in print [19]. Search strings combined pathogen terms (“West Nile virus”, “dengue”, “chikungunya”, “arbovirus”, “phlebovirus”, “Usutu”, “Sindbis”) with geographic terms (“North Africa”, “Maghreb”, “Morocco”, “Algeria”, “Tunisia”, “Libya”, “Egypt”, “Sudan”) and

thematic terms ("seroprevalence", "outbreak", "surveillance", "vector", "Aedes", "Culex"). WHO Disease Outbreak News, the WHO Weekly Epidemiological Record and national observatory bulletins were consulted as grey literature, and the reference lists of retrieved reviews were searched by hand. The search was not designed to be exhaustive.

Targeted searches were also run for Libyan *Aedes* and arbovirus records, Maghreb dengue and chikungunya serosurveys, arbovirus detection in North African mosquitoes, *Ae. albopictus* density and seasonality in Algeria and Morocco, climatic determinants of WNV timing within North Africa, and phylogenetic analyses connecting Egyptian or Sudanese DENV and CHIKV to Maghreb sequences. Bibliographic details for all cited articles were checked against PubMed.

2.3. Eligibility

Sources were eligible if they reported primary or secondary data on the detection of dengue virus (DENV), chikungunya virus (CHIKV) or WNV in humans, animals, vectors or the environment in at least one of the six countries, or regional and global analyses from which North African data could be extracted. Studies of other arboviruses (phleboviruses, Usutu virus, Sindbis virus) were used only where they bear on flavivirus serological cross-reactivity or on the differential diagnosis of neurological illness. Travel-associated cases were included where they indicated unrecognised local transmission. Non-arboviral causes of acute febrile illness were treated on the same restricted basis: sources on filoviruses were used only where they bear on diagnostic breadth or on the differential diagnosis of undifferentiated febrile illness, and were retrieved by targeted searching rather than by the strategy set out in section 2.2. Figures for the 2026 Bundibugyo virus epidemic were taken from WHO Disease Outbreak News and peer-reviewed reports consulted on 6 November 2026, after the final search date, and are cited with the dates to which they refer.

Five grounds led to exclusion. Regional and global analyses that gave no country-level or subregional figure for any of the six countries were excluded for lack of extractable North African data. Sources that stated no assay or design were excluded because no evidence level could be assigned to them; conference abstracts and surveillance summaries fell on this ground unless the assay was named. When one event was reported more than once, the fullest report was kept and the others cited only if they added data (the Tunisian epidemics of 1997 and 2003 and the Sudanese epidemic of 2018–19 each appear in several sources). Commentary without data, such as editorials and letters restating figures published elsewhere, was excluded, as were other-arbovirus studies outside the restriction described above. Two further constraints follow from the design, not from an exclusion criterion: estimates were not pooled, and no formal risk-of-bias instrument was applied, for the reasons given in section 2.1.

2.4. Ethical approval

Not required. This review draws only on published literature and surveillance reports already in the public domain.

2.5. Evidence levels

Each source was assigned to one of five evidence levels according to what was measured and how it was confirmed (Table 1). Levels were assigned from each source's stated design and assays. A level describes the strongest inference a source can support and is not a judgement of its quality. Wording in the Results follows the level: "documented" for levels A and B, "reported" for level C, "shown in the laboratory" or "present" for level D, and "inferred" or "projected" for level E. Where an estimate came from a single study or a single synthesis, this is stated in the text. No statistical software was used, because no quantitative synthesis was performed.

Table 1. Evidence levels used in this review

Level	Evidence type	What it supports	Wording used
A	Laboratory-confirmed human infection or disease (RT-PCR, sequencing, virus isolation, or neutralization-confirmed IgM or seroconversion)	Transmission has occurred	Documented
B	Neutralization-confirmed antibodies in humans or sentinel animals	Past exposure of a population (cumulative, not incidence)	Documented (exposure)
C	Antibodies detected by ELISA or haemagglutination inhibition without neutralization confirmation	Possible exposure; inflated by cross-reacting viruses	Reported
D	Vector presence, natural infection of mosquitoes, or experimental competence	Capacity for transmission	Shown in the laboratory; present; detected
E	Extrapolation or model inference from other regions or from age-structured serology	A hypothesis	Inferred; projected

3. Results

3.1. The evidence base by country

Table 2 sets out the strongest evidence found for each virus and vector in each country.

Table 2. Strongest evidence by country and virus. Letters in parentheses are evidence levels (Table 1)

Country	West Nile virus	Dengue and chikungunya	*Aedes* vectors
Morocco	Human exposure confirmed by neutralization (B); equine outbreaks in 1996, 2003 and 2010 with one human case (A)	None reported	*Ae. albopictus* since 2015 (D); transmits CHIKV in the laboratory (D)
Algeria	Outbreak in 1994 and later cases (A); neutralization-confirmed human exposure (B)	No human case reported; CHIKV RNA reported in three mosquito pools collected 2018–2019, in *Culex* and *Anopheles* abdomens only, one of three confirmed at Ct 35 and none sequenced (D, unconfirmed) [20]	*Ae. albopictus* since 2010 (D)
Tunisia	Epidemics in 1997, 2003, 2012, 2018 and 2023, with sequencing (A); equine infections confirmed across 2018–2023 (B)	None reported	*Ae. albopictus* since 2018, in several regions (D); transmits CHIKV in the laboratory (D)
Libya	Neutralization-confirmed exposure in horses and dogs (B); one human ELISA dataset (C) [21]	None found	No primary record of either invasive species found; one local larval survey without *Aedes* (D)
Egypt	Virus isolated from human sera in 1951 (A) [22]; endemic since at least the 1950s; neutralization-confirmed exposure in Nile Delta horses (B)	Outbreaks in Upper Egypt in 2015 and 2016 (A); dengue outbreak and traveller-detected transmission on the Red Sea and Sinai coasts, 2021–2024 (A); no autochthonous chikungunya found	*Ae. aegypti* re-established since 2018 [23]; on the Red Sea coast, naturally infected with DENV-1, -2 and -3 (D); at case locations in Upper Egypt (D)
Sudan	Antibody prevalence 2.2–47% in a previous systematic review (C; primary studies not appraised) [10]; no WNV IgM in 152 febrile patients in Darfur, 2018 (C) [24]	Dengue IgG in 67% of febrile outpatients tested in 2012–13 (C; subset neutralized) [25]; chikungunya epidemic 2018–19; dengue and chikungunya in Tandalti, 2022; all four dengue serotypes in Kassala, 2023–24 (A)	*Ae. aegypti* in most of the country; *Ae. albopictus* first reported in 2022, in Kordofan, Sennar and White Nile states (D)

Two patterns in Table 2 shape the rest of the review. The level of evidence differs by virus: WNV has level A or B evidence in five of the six countries, whereas dengue and chikungunya have level A evidence only in Sudan and Egypt. For Morocco, Algeria and Tunisia the evidence for these two viruses remains confined to vector presence and laboratory competence (level D) and to extrapolation from Europe (level E). The single Algerian report of chikungunya virus RNA in mosquitoes [20] does not change this, for reasons given in section 3.3.3.

Coverage is also uneven. Libya has the thinnest evidence base, with WNV data from animals and a single human ELISA dataset [21], and no primary record of either invasive *Aedes* species. For Sudan, the WNV evidence was taken from a previous systematic review and was not appraised at the level of the primary studies.

Figure 1 shows the same assignments as a matrix. Read by column, WNV is the only virus for which laboratory-confirmed human infection or neutralization-confirmed exposure exists across the region. Read by row, the region splits in two: Egypt and Sudan have confirmed dengue or chikungunya transmission, whereas in the Maghreb the evidence for those two viruses stops at vector presence and laboratory competence. Libya is the only country with no retrieved source for either virus. The rest of the Results examines what lies behind that pattern.

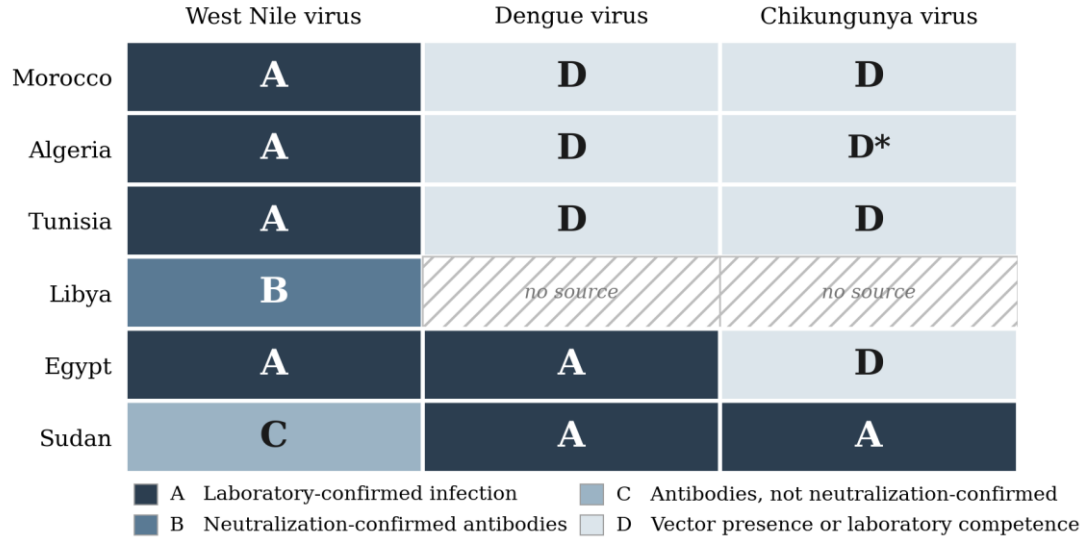


Fig. 1. Strongest evidence level retrieved for each country and virus. Shading darkens with the strength of the inference the evidence can support. Assignments follow Table 2; levels are defined in Table 1.

3.2. West Nile virus

3.2.1. Endemic history and human outbreaks

WNV was isolated in Uganda in 1937 and described in 1940 [19], but its ecology was worked out in the Nile Delta in the early 1950s, where surveys found antibody prevalence approaching 60% and the bird–*Culex* cycle was described [26,27]. In 1951 the virus itself was isolated from the sera of patients in Egypt and shown to be apparently identical to WNV [22]. That isolation is the earliest level A evidence for the region and shows human infection in Egypt directly, without relying on antibody prevalence. High antibody prevalence in Egypt is best read as the product of decades of endemicity, not of a recent introduction.

In the Maghreb, human neuroinvasive disease has been documented repeatedly (Table 3). Tunisia had epidemics in 1997, 2003, 2012 and 2018. Sequencing of the 2012 outbreak showed lineage 1 strains related to Tunisian isolates from 1997 and Israeli isolates from 1998, alongside a second cluster related to Kunjin virus [28], and the 2018 outbreak again involved lineage 1 [29]. Algeria recorded a meningoencephalitis outbreak in the Tinerkouk oasis of the south-western Sahara in 1994, with about 50 suspected cases and eight deaths [30,31], a fatal neuroinvasive case in Jijel in 2012, and two clinical cases in 2013–2014 in Timimoune and Guelma [31]. In Morocco the outbreaks were equine (1996, 2003, 2010), with a single human case in 1996 [32,33]. No human outbreak has been reported in Libya.

Table 3. Documented West Nile virus outbreaks and clinical events in North Africa

Country, year	Event	Human cases (deaths)	Confirmation and limitation	Ref.
Egypt, 1950–1954	Outbreaks and serosurveys, Nile Delta; virus isolated from human sera, 1951	Not enumerated	Virus isolation from patients established human infection directly [22]. Antibody prevalence up to about 60%; bird–*Culex* cycle described. Historical serology, not comparable with current assays.	[22,26]
Algeria, 1994	Meningoencephalitis outbreak, Tinerkouk oasis	About 50 suspected (8)	Clinical diagnosis with serology (83% of sera positive). Suspected-case count approximate.	[30,31]
Algeria, 2012–2014	Fatal neuroinvasive case (Jijel); cases in Timimoune and Guelma	3 (1)	Clinical reports. Small number of cases.	[31]
Morocco, 1996	Equine outbreak, Kenitra region; one human case	1 human; 94 equine (42 deaths)	Lineage 1a.	[32,33]
Morocco, 2003	Equine outbreak, Kenitra	0 human; 9 equine (5 deaths)	Full genome; lineage 1a.	[32]
Morocco, 2010	Equine outbreak, central and north-west	0 human; 17 equine (8 deaths)	Epizootic confirmed.	[33]
Tunisia, 1997	Epidemic, central coastal region	111–173 (8)	Lineage 1a. Counts differ between sources.	[28]
Tunisia, 2003	Epidemic	21–112 (3–9)	Lineage 1a. Counts differ widely between sources.	[28]
Tunisia, 2012	Sahel epidemic (Monastir)	86 (12)	Two strain groups; human, mosquito and bird isolates closely related.	[28]
Tunisia, 2018	Neuroinvasive outbreak	Not fully enumerated	Lineage 1 by sequencing.	[29]
Tunisia, 2023	Outbreak affecting humans and horses	Not enumerated in the retrieved source	Of 20 horses tested in November 2023, 5 were ELISA-positive (25%) and 2 were confirmed by microneutralization (10%); no viral RNA was detected and no equine clinical cases were reported. Small, non-random equine sample from a single institution; follow-up in December 2023 and January 2024 showed no seroconversion.	[34]
Libya	No human outbreak reported	None	Neutralization-confirmed antibodies in horses and dogs, 2016–2019.	[35]

The Tunisian survey covering 2011–2014 examined humans, mosquitoes and birds together; 7 of 79 cerebrospinal fluid samples from suspected cases (8.8%) were PCR-positive, all from the Monastir region [28]. Enzootic circulation of lineage 1 was detected in sentinel chickens in northern Tunisia between epidemics [36], and a retrospective screen of 25 suspected equine samples collected across 2018 to 2023 found 11 positives (44%), all lineage 1a and forming a distinct group within the Mediterranean subtype of that clade [37]. The pattern in Tunisia and Algeria is consistent with a virus that circulates locally and produces human epidemics at intervals, rather than with repeated introduction alone. That reading rests on molecular data for Tunisia (level A) but on a small number of clinical reports for Algeria.

Two limitations apply to Table 3. Case numbers for several epidemics differ between sources, so the Tunisian epidemics of 1997 and 2003 are reported as ranges. All counts are of clinically recognised neuroinvasive disease, which by definition excludes infections that were mild or asymptomatic.

3.2.2. Serological evidence

Table 4 lists serosurveys by host, assay and evidence level.

Table 4. Serological evidence of West Nile virus circulation in North Africa

Country	Host (n; year)	Assay	Result	Level	Ref.
Egypt	Humans; national estimate	Pooled	24.0%	Pooled	[13]
Egypt	Equids (n = 1,100; 2020–2022)	c-ELISA	26.6% IgG; higher in horses than donkeys (OR 1.65)	C	[38]
Egypt	Horses (n = 930; Nile Delta; 2018)	ELISA + PRNT	16.8% (156 of 930)	B	[39]
Algeria	Humans (n = 164; Algiers region, 2010)	ELISA + PRNT	9.8% IgG; 6.7% neutralization-confirmed	B	[40]
Algeria	Humans (n = 1,964; multi-province, 2017–2018)	Serocatalytic survey	16.7% overall	C/E	[41]
Algeria	Equids (north-east, 2014)	ELISA + VNT	17.4%	B	[31]
Algeria	Equids and poultry (n = 389)	ELISA + MNT/MIA	52.4% flavivirus IgG; 30.8% WNV-confirmed	B	[20]
Algeria	Horses (n = 289; nine provinces across three climatic zones; 2021–2023)	ELISA + VNT	23.2% flavivirus IgG; 17.0% WNV-confirmed; 0.7% Usutu-confirmed; no tick-borne encephalitis virus	B	[42]
Tunisia	Blood donors (n = 844)	ELISA + VNT	17.5% IgG; 31.1% of positives neutralization-confirmed	B	[13,43]
Tunisia	Hospital patients without recorded clinical data (n = 353; 2017–2020)	ELISA, IFA, RT-PCR	28.8% IgM; 19.2% IgG; 9.2% PCR-positive	A	[44]
Tunisia	Horses (n = 20; Tunis; November 2023 outbreak)	ELISA + MNT	25% ELISA-positive (5 of 20); 10% microneutralization-confirmed (2 of 20); no viral RNA	B	[34]
Sudan	Febrile patients (n = 152; Darfur; Sep–Dec 2018)	IgM ELISA	No WNV IgM detected	C	[24]
Sudan	General population	Various	2.2–47%	C	[10]
Morocco	Healthy adults (n = 499; Meknes, Rabat, Kenitra; 2011)	Virus neutralization	18.8% in Kenitra, 12% in Rabat	B	[45]
Morocco	Horses (n = 840; four regions; 2011)	ELISA + VNT	31% overall (261 of 840); highest in the Atlantic Gharb and pre-Rif region	B	[33]
Libya	Humans (n = 400; Tripoli, Aug–Oct 2013)	ELISA	2.75% (11 of 400)	C	[21]
Libya	Horses (n = 574) and dogs (n = 63); 2016–2019	c-ELISA + VNT	ELISA 13.2% (horses), 30.2% (dogs); VNT-confirmed 10.8% and 27%	B	[35]
Regional	Humans (n = 7,930; 2010–2023)	Random-effects meta-analysis	15.4% (95% CI 6.7–24.2)	Pooled	[13]

ELISA, enzyme-linked immunosorbent assay; c-ELISA, competitive ELISA; IFA, immunofluorescence assay; MIA, microsphere immunoassay; MNT, microneutralization test; PRNT, plaque-reduction neutralization test; VNT, virus neutralization test. Pooled estimates combine assays.

The most consistent feature of Table 4 is the difference that neutralization makes. In the Algiers region, 9.8% of 164 people were IgG-positive by ELISA and 6.7% were confirmed by neutralization; the authors noted that specific IgG fell after plaque-reduction confirmation and that antigenically related flaviviruses should be investigated [40]. Among 844 Tunisian blood donors, 17.5% were IgG-positive and 31.1% of the positives were confirmed by neutralization [13,43], which implies a confirmed prevalence of about 5% (0.175 × 0.311) if the proportion confirmed applies to all positives. Across the Mediterranean Basin, neutralizing antibodies were found in about half of 731 IgG-positive people tested [13]. Unconfirmed ELISA values from the region may therefore overstate exposure to WNV, by a factor of about 1.5 to 3 in the human series where both measures are available. Cross-reacting flaviviruses are a plausible cause: Usutu virus has been detected in Tunisian mosquitoes and in horses alongside WNV [46], and the same virus has since been confirmed by neutralization in Algerian chickens and equids [20].

Animal data are more often confirmed by neutralization, but the loss on confirmation varies widely between series and no single value characterizes them. In Libya the loss was small: 13.2% by ELISA against 10.8% by neutralization in horses, and 30.2% against 27% in dogs [35]. In the Algerian survey of 289 horses across nine provinces it was about a quarter, 23.2% by ELISA against 17.0% confirmed for WNV, with 0.7% confirmed for Usutu virus and none for tick-borne encephalitis virus [42]. In the 20 Tunisian horses tested during the 2023 outbreak it was large: 5 of 20 were ELISA-positive (25%) but only 2 were confirmed by microneutralization (10%), so three of the five ELISA positives were not confirmed [34]. The small Libyan loss is thus a feature of one series, not of animal data in general, and the range across these three series spans a loss of roughly one-fifth to three-fifths. Even after confirmation, values remain substantial: 31% of 840 Moroccan horses, with the highest values in the Atlantic Gharb and pre-Rif region [33], and 16.8% of 930 horses in the Nile Delta [39]. Age group was the only risk factor that reached significance in the Libyan study [35]. These data show enzootic circulation in places where human data are sparse. They do not convert into human incidence, and equine sampling frames were not designed to represent human exposure.

Sampling frames also limit the human data. The Moroccan values of 18.8% in Kenitra and 12% in Rabat came from 499 healthy adults, with the highest value in the Kenitra outbreak zone [45], and are not

a national estimate. The Libyan figure comes from 400 people who attended the Tripoli Reference Laboratory between August and October 2013 to obtain a health certificate, of whom 11 (2.75%) were IgG-positive by ELISA [21]. That is a convenience sample of one city in one season, tested without neutralization confirmation, and it is the only human serological dataset available for the country.

3.2.3. Seroprevalence and reported cases

The regional meta-analysis reported that North Africa had the highest pooled human seroprevalence in the Mediterranean Basin (15.4%; 95% CI 6.7–24.2, compared with 2.7% in Southern Europe) and fewer reported human cases, and it suggested potential under-diagnosis and cryptic transmission [13]. Country values summarized in that review range from 2.8% in Libya and 4.4% in Morocco to 21.5% in Tunisia and 24.0% in Egypt [13]. The comparison cannot be taken at face value, for three reasons.

First, most of the pooled estimate derives from assays without neutralization confirmation, which Table 4 suggests overstate exposure. Across the whole review, neutralization data covered 731 IgG-positive people, about 7% of the roughly 10,000 IgG-positive people implied by the overall prevalence of 6.5% among 160,775 tested (computed from [13]). Second, cumulative antibody prevalence and reported cases measure different things: the highest country values come from Egypt, where endemicity has been recognised for seventy years, and from Tunisia, where epidemics have occurred. Third, most WNV infections cause mild illness or none. In the household survey after the 1999 New York outbreak, roughly 140 infections were estimated for each case of encephalitis or meningitis [47]. A large ratio of infections to reported neuroinvasive cases is therefore expected even where case detection is complete.

The expected ratio for North Africa is not known, and the regional incidence of neuroinvasive disease has not been measured, so the size of the shortfall in reported cases cannot be calculated from the available sources. Only one study has modelled human serology in the region, and it points towards outbreaks that went unrecognized. A 2026 analysis of a cross-sectional human survey in Algeria (seroprevalence 16.7%, with age, region and residence type identified as risk factors) fitted serocatalytic models and found that patterns in two southern provinces were consistent with low-level endemic transmission over decades, whereas patterns in three northern provinces were consistent with single outbreaks within the past 10 to 15 years, possibly in Oran in 2010 and in Jijel and Tizi Ouzou near the time of data collection in 2017 and 2018 [41]. This is an inference from age-structured serology, not a confirmed outbreak record, and the assay and confirmation strategy of that survey were not appraised here. Even so, it raises the possibility that some northern outbreaks are missing from Table 3.

The data support a narrower conclusion than cryptic transmission at scale. Clinically apparent WNV disease occurs in the region and is recognised when it appears as a cluster (Table 3). Where laboratory testing has been applied to suspected cases in Tunisia, the yield has been modest: WNV RNA was found in 7 of 79 cerebrospinal fluid samples from suspected cases collected between 2011 and 2014, all from the Monastir region [28], and three human cases were confirmed through national meningitis surveillance during the 2021–22 entomological study [48]. Under-diagnosis is likely; its magnitude is unmeasured.

3.2.4. Vectors, co-circulating flaviviruses and climate

Tunisian surveillance has detected WNV, Usutu virus and Sindbis virus in mosquito pools, and antibodies to WNV, Usutu virus and tick-borne encephalitis virus in horses: of 369 horses surveyed, 146 were flavivirus-seropositive by competitive ELISA, and of 104 tested further, 74 were positive for WNV, 8 for Usutu virus and 2 for tick-borne encephalitis virus [46]. A Tunisian investigation combining human and entomological surveillance, with mosquito sampling from November 2021 to October 2022, detected WNV RNA in 21 mosquito pools (10.19%) belonging to *Culex pipiens*, *Cx. perexiguus*, *Aedes caspius* and *Ae. detritus*; three human cases were identified through national meningitis surveillance, and all sequences belonged to sublineage 1a [48]. RNA in mosquito pools shows infection, or a recent blood meal from a viraemic host; it does not by itself show that a species is a competent vector. In Morocco, WNV RNA in *Cx. pipiens* and antibodies in horses were found in parallel [49], and the authors of the Moroccan reports point to wetlands on migratory bird routes near Kenitra [32,45].

In Europe the 2018 transmission season started early [50], and short-term temperature and precipitation affect the incidence of neuroinvasive disease [51]. A continental risk map for Africa exists [52]. One Mediterranean model is more directly relevant: using about 270 human and equine cases notified in Italy, Greece, Portugal, Morocco and Tunisia between 2008 and 2012, together with altitude, land-surface temperature, vegetation indices and modelled mosquito seasonality, Conte and colleagues produced monthly maps of areas suitable for West Nile disease and placed Tunisia, Libya and Egypt among the most suitable between May and July, with suitability persisting on the coasts of Morocco, Tunisia, Libya and Egypt into December [53]. That analysis is ecological and was fitted largely to European and Moroccan-Tunisian case data. Within North Africa itself, the Algerian equine survey found that confirmed WNV seroprevalence varied with climatic zone and altitude as well as with age and breed [42], which establishes that exposure is spatially structured by climate. No retrieved source, however, relates measured temperature or precipitation to the *timing* of WNV cases or equine outbreaks within the region: the Algerian result is a spatial

gradient in cumulative exposure, not a time series, and cannot be used to anticipate when a season will begin.

3.3. Dengue and chikungunya

3.3.1. Sudan

Dengue in Sudan is documented by serosurveys and by outbreak and molecular reports. A systematic review of the Middle East and North Africa identified 105 human prevalence measures in 13 of 24 countries and 81 outbreaks in nine countries between 1941 and 2015, with seroprevalence studies concentrated in the Red Sea region and Pakistan, a median of 25% (range 0–62%), and neutralization testing used in only 5% of seroprevalence studies [7]. Among febrile outpatients in eastern and central Sudan in December 2012 and January 2013, 302 of 448 IgM-negative sera (67%) were IgG-positive (89% in Port Sudan, 61% in Kassala, 57% in North Kordofan), and 31 of 32 ELISA-positive sera neutralized dengue viruses [25]. A community survey in Kassala State in 2016–17 found IgG in 42% of 157 sera tested, a house index of 32.8% for *Ae. aegypti*, and DENV RNA in 1 of 329 adult mosquitoes [54]. DENV-4 was detected in Sudan in 2023 [55].

The chikungunya epidemic of 2018–19 is the best-documented event in the region (Table 5). Red Sea State reported its first suspected cases on 31 May 2018 and Kassala State on 8 August; by 2 October, 13,978 cases had been reported from seven states, 95% of them in Kassala State [56], and 20,110 cases had been reported by November [57]. Across the nine months of the epidemic, Kassala and Red Sea States reported 48,763 cases in total, and Bower and colleagues describe it as the largest chikungunya epidemic in Africa and the Middle East to date, affecting an estimated 487,600 people [58].

That figure is not an independent measurement. The authors cite seroprevalence surveys from other outbreak settings indicating that surveillance systems capture only 2–15% of cases, adopt 10% as a conservative value, and divide the 48,763 reported cases by it [58]. Tenfold under-ascertainment is thus an input to the estimate, not a finding, and the figure says nothing independent about the size of the gap in Sudan. What the authors do supply independently are reasons to expect substantial under-reporting in this particular outbreak: a sentinel system designed to raise an alert rather than to count every case, with one cited study reporting 67 symptomatic cases for each case captured by such a system; reluctance to attend hospital on grounds of cost; limited diagnostic capacity; no data collection in private clinics; and the wide range of illness severity [58]. The direction of the bias is well supported for Sudan in 2018–19. Its magnitude rests on an assumption imported from other settings.

The hospital cohort reported by Bower and colleagues enrolled 102 adults and 40 children with chikungunya-like illness at Kassala Teaching Hospital in October 2018. Of these 142 patients, 120 (84.5%) were RT-PCR positive; nine of the 120 (7.5%) had concurrent dengue virus infection, 34 of 118 tested (28.8%) had a positive rapid test for *Plasmodium falciparum*, six (5.0%) had hemorrhagic symptoms including two children with life-threatening bleeding, and one died with acute renal injury. All sequences fell in a single clade of the Indian Ocean lineage of the East/Central/South African genotype. Two to four months after illness, 63% of the adults available for follow-up still had arthralgia and 11% remained moderately disabled [58]. These proportions describe febrile hospital patients at one time point and cannot be extended to the population, but persistent arthralgia on this scale means that acute case counts understate the burden of such an epidemic. The authors attribute continuing vulnerability to widespread *Ae. aegypti* and household water storage [58], and WHO noted *Ae. aegypti* in most parts of the country [56]. An entomological survey in Kassala State recorded immature *Aedes* stages in some localities [59]. *Ae. albopictus* was first reported in Sudan in 2022, from a larval survey in North and South Kordofan, Sennar and White Nile states, where three specimens were identified by sequencing [60].

Three further datasets extend the Sudanese record beyond the 2018–19 epidemic. In Tandalti, White Nile State, an outbreak of undifferentiated febrile illness reported through 32 health clinics between early October and mid-December 2022 yielded 63 laboratory-confirmed cases among 773 suspected cases (8.2%): 49 chikungunya (77.8% of those confirmed), 11 dengue (17.5%) and 3 co-infections (4.8%), confirmed by IgM capture ELISA with real-time RT-PCR [61]. This was the first reported outbreak of either virus in that state, in an area with high *Ae. aegypti* density. In Kassala between September 2023 and February 2024, 276 of 548 febrile patients (50.4%) were confirmed as dengue by rapid testing, and RT-PCR serotyping of 266 samples found DENV-3 in 50%, DENV-2 in 39.8%, DENV-4 in 3%, and DENV-1 only in combination with DENV-3 (2.4%), with DENV-2 and DENV-3 co-detected in a further 4.9% [62]. All four serotypes therefore circulated simultaneously in a single outbreak, which carries a recognized risk of severe disease on secondary infection. Earlier, in Darfur between September and December 2018, IgM ELISA on 152 febrile patients found dengue in 15.1% and chikungunya in 3.3%, alongside yellow fever in 44.7% with 11 molecularly confirmed yellow fever cases; no WNV, Rift Valley fever or Crimean-Congo hemorrhagic fever exposure was detected [24]. These are IgM results from a selected febrile group, not population prevalences, and the high yellow fever fraction in a vaccinated population complicates interpretation, but together the three datasets show dengue and chikungunya activity in Sudan extending west to Darfur and recurring through 2022 and 2023–24.

3.3.2. Egypt

The earliest recent evidence comes from Upper Egypt, not from the

coasts. In October 2015, 253 patients were admitted to Dayrout Fever Hospital in Assiut Governorate with acute febrile illness; 118 serum samples were collected and 28 were positive for dengue, confirmed as DENV-1 by ELISA and PCR at the Central Public Health Laboratories and NAMRU-3, with *Ae. aegypti* larvae and adults found at the affected sites [63]. A second outbreak in Upper Egypt in 2016 was investigated with an RT-LAMP assay, which was positive in 31 of 51 clinically suspected patients (60.8%) and in 16 of 36 pools of field-collected *Ae. aegypti* (44.4%), with DENV-1 the most prevalent serotype in both [64]. That study also reported combinations of two, three or four serotypes in most positive samples; mixed-serotype results from an isothermal amplification assay need confirmation by sequencing before they can be taken as evidence of co-circulation on that scale. Both outbreaks followed the re-establishment of *Ae. aegypti* in Egypt, which was documented in 2018 after the species had been considered absent for decades [23].

Dengue transmission on the Red Sea and Sinai coasts is documented by five further sources (Table 5). An outbreak of DENV-2 in El Quseir in 2021–22 was confirmed in humans and in mosquitoes [65]; German surveillance recorded an increase in dengue among travellers returning from Egypt in 2023 [66]; sentinel travellers uncovered an outbreak in Sharm El-Sheikh in May 2024 [67]; sequencing of DENV-2 from travellers returning to Israel from the Sinai Peninsula showed phylogenetic links to South Asian strains [68]; and DENV RNA was detected in field-collected *Ae. aegypti* from a Red Sea governorate in 2023–24, with serotypes 1, 2 and 3 all present [69]. That last study also reported a seasonal pattern that runs against the expectation imported from temperate settings: across five districts, viral detection peaked in December 2023 and no infected pool was found between June and September [69]. This rests on a single season in one governorate and needs confirmation before it is relied on, but if dengue transmission on the Red Sea coast proves to be a winter rather than a summer phenomenon, surveillance and vector control timed to the European season would look in the wrong months. Four of these five sources detect transmission indirectly, through returning travellers or field-collected mosquitoes, which shows that local transmission occurred but gives no denominator. Taken with the Upper Egyptian outbreaks, the evidence documents multi-serotype dengue transmission in Upper Egypt and on the Red Sea and Sinai coasts without establishing its incidence. The Nile Delta and the Mediterranean coast remain unmeasured: no retrieved source reports dengue testing of febrile patients or vector infection in either.

No autochthonous chikungunya has been reported in Egypt. Isolated travel-associated cases with chikungunya IgM have been described after travel to Egypt, but IgM alone is cross-reactive and a single case report cannot establish local transmission.

Table 5. Dengue and chikungunya events with documented North African involvement

Year	Location	Event	Evidence and limitation	Ref.
1941–2015	Middle East and North Africa, including Sudan and Egypt	81 dengue outbreaks in nine countries	Systematic review; regional, not restricted to North Africa.	[7]
2012–13	Sudan (Port Sudan, Kassala, North Kordofan)	Dengue IgG in 67% (302 of 448) of IgM-negative sera from febrile outpatients	31 of 32 ELISA-positive sera neutralised dengue viruses. Clinic attenders, not a population sample.	[25]
2016–17	Sudan (Kassala State)	Dengue IgG in 42% of 157 sera; *Ae. aegypti* house index 32.8%	ELISA only; 1 of 329 adult mosquitoes DENV-positive; small sample.	[54]
2018–19	Sudan (seven states; Kassala and Red Sea most affected)	Chikungunya epidemic, described as the largest in Africa to date	13,978 cases reported by 2 October 2018; 20,110 by November; about 487,600 people estimated affected. Indian Ocean lineage of the ECSA genotype. Dengue co-infection in 9 of 120 PCR-positive hospital patients (7.5%); 63% of adults followed up still had arthralgia at 2–4 months.	[56–58]
2018	Sudan (Darfur)	Dengue and chikungunya IgM in febrile patients	IgM ELISA on 152 patients: dengue 15.1%, chikungunya 3.3%; no WNV detected. Selected febrile group; IgM alone; high concurrent yellow fever positivity complicates interpretation.	[24]
2022	Sudan (Tandalti, White Nile State)	First dengue and chikungunya outbreak in the state	63 of 773 suspected cases confirmed (8.2%): 49 chikungunya, 11 dengue, 3 co-infections. IgM capture ELISA with RT-PCR confirmation; October to mid-December 2022.	[61]
2023	Sudan	DENV-4 detected	Molecular confirmation.	[55]
2023–24	Sudan (Kassala)	Dengue outbreak with all four serotypes circulating	276 of 548 febrile patients confirmed (50.4%); RT-PCR on 266 samples found DENV-3 (50%), DENV-2 (39.8%), DENV-4 (3%) and DENV-1 only with DENV-3 (2.4%). Hospital attenders, not a population sample.	[62]
2015	Egypt (Dayrout, Assiut; Upper Egypt)	DENV-1 outbreak	253 suspected cases admitted; 28 of 118 samples positive by ELISA and PCR; *Ae. aegypti* larvae and adults at affected sites.	[63]
2016	Egypt (Upper Egypt)	Dengue outbreak with vector infection	RT-LAMP positive in 31 of 51 suspected patients and 16 of 36 *Ae. aegypti* pools; DENV-1 predominant. Reported mixed serotypes not confirmed by sequencing.	[64]
2021–22	Egypt (El Quseir, Red Sea)	DENV-2 outbreak	Confirmed in humans and mosquitoes.	[65]
2023	Egypt (travellers returning to Germany)	Increase in dengue among travellers from Egypt	Surveillance signal; no denominator.	[66]

Year	Location	Event	Evidence and limitation	Ref.
2024	Egypt (Sharm El-Sheikh, Sinai)	Outbreak uncovered through sentinel travellers	Traveller cases with sequencing; DENV-2 related to South Asian strains. Local incidence unknown.	[67,68]
2023–24	Egypt (Red Sea governorate)	DENV RNA in field-collected *Ae. aegypti*; serotypes 1, 2 and 3	Entomological RT-PCR; human incidence not measured.	[69]
Collected 2018–2019	Algeria (El Kala, El Taref)	CHIKV RNA reported in three mosquito pools, alongside Banna, Sindbis and Usutu viruses	Multiplex screening of 180 pools from 1,658 mosquitoes. Positive pools were *Cx. pipiens*, *Cx. perexiguus* and *An. labranchiae*; virus was found in abdomens only, with no head or thorax positive. One of the three pools was confirmed by RT-PCR, at Ct 35; the other two were not confirmed, and none was sequenced. The authors interpret the result as a blood meal taken on an infected host and rule out *Culex* and *Anopheles* as vectors. No human case.	[20]
To 3 October 2026	Morocco, Algeria, Tunisia, Libya	No locally acquired dengue or chikungunya reported	Sensitivity of detection unknown; no recent serosurvey of resident populations found.	[7,8]

All level A evidence for dengue and chikungunya comes from the eastern part of the region, and the events recur: Sudan has documented events in 2018–19, 2022 and 2023–24, and Egypt in 2015, 2016 and from 2021 onwards. The Egyptian evidence now covers Upper Egypt as well as the Red Sea and Sinai coasts, but much of it is indirect and none of it establishes incidence; the Nile Delta and Mediterranean coast are untested. The Algerian detection of chikungunya virus in mosquitoes is examined separately in section 3.3.3.

3.3.3. Maghreb and Libya

No locally acquired dengue or chikungunya has been reported in Morocco, Algeria, Tunisia or Libya in the sources available to 3 October 2026. The systematic review of dengue in the Middle East and North Africa noted that study coverage across the region was insufficient to draw conclusions about *Aedes* or DENV presence in several countries [7], and the review of urban chikungunya concluded that urban populations in the region were largely immunologically naive [8]. That conclusion rests on sparse serological data, and no recent serosurvey of dengue or chikungunya immunity in the resident Maghreb population was identified. Sudan, by contrast, has several dengue serosurveys (Table 5).

One entomological finding is often cited as evidence that this absence is only apparent, and it needs to be stated precisely. A study combining a high-throughput multiplex platform for mosquitoes with serological testing of animal hosts screened 1,658 mosquitoes in 180 pools, collected with CO_2-baited CDC light traps in July and October 2018 and October 2019, and reported chikungunya, Banna, Sindbis and Usutu viruses in Algeria [20]. Chikungunya virus was reported from three pools, all collected at El Kala in El Taref province: urban *Culex pipiens*, rural *Cx. perexiguus* and rural *Anopheles labranchiae*. Four details matter. The virus was found in abdomens only, and no head or thorax from any of the three pools was positive, which indicates an ingested blood meal rather than a disseminated infection. Confirmatory RT-PCR was positive for only one of the three pools, at a cycle threshold of 35, close to the limit of detection; the other two pools were not confirmed. No chikungunya sequence was obtained, so the virus cannot be placed relative to the Indian Ocean lineage strains that circulated in Sudan or to strains circulating elsewhere. And the authors themselves read the finding as a blood meal taken on an infected host, stating that the involvement of *Culex* and *Anopheles* in transmission can be ruled out [20].

On the authors' own interpretation, then, this is a possible trace of a chikungunya-infected vertebrate host at El Kala, detected once and weakly, and not evidence of virus circulating in mosquitoes as vectors. It deserves follow-up, because an infected host implies an infection somewhere, and the study used traps that are not designed to catch *Ae. albopictus* (no specimens of that species were collected, and the authors recommend BG-Sentinel traps for future work). But it does not establish autochthonous transmission or lift the Maghreb above vector presence and laboratory competence, and until it is confirmed and sequenced it should not be cited as though it did.

3.4. Vectors and competence

Ae. albopictus was first documented in Algeria in 2010 [70], in Morocco (Rabat) in 2015 [71] and in Tunisia (greater Tunis) in 2018 [72]; intermittent nationwide sampling in Tunisia between 2013 and 2023 collected 35 species against 49 previously reported in the literature and recorded the spread of *Ae. albopictus* to several regions [73]. These are dates of first documentation, not of arrival or establishment: arrival probably preceded detection, whereas a first detection does not by itself show a breeding population that survives winters. In Algeria the species has since spread well beyond the point of first detection: a molecular survey of specimens from five districts reported that it had colonized the whole of the north of the country as far as the semi-arid zone, and that Algerian sequences fell in a single cytochrome oxidase haplotype shared with Morocco, Madagascar, Mauritius and populations on three other continents [74]. That establishes range expansion and a common invasion source, but twenty specimens from five districts give no measure of abundance, and no retrieved source describes the density or seasonality of the species in Algeria or Morocco. Yet transmission depends on density, not on presence alone. The species is spreading across the African continent [75], and global distribution models place parts of the North African coast within the suitable range of

both *Aedes* species [76–78], which is a level E inference.

Table 6 summarizes vector records and competence. A field-collected *Ae. albopictus* strain from Morocco transmitted chikungunya virus efficiently and transmitted dengue, Zika and yellow fever viruses less so, with virus in saliva by day 3 after infection for chikungunya virus, day 14 for dengue and yellow fever viruses and day 21 for Zika virus [79]. A Tunisian population had a chikungunya transmission efficiency of 25% at 21 days, compared with 8.7% for Zika virus and 8.3% for dengue virus [80]. These are laboratory results from single populations and specific virus strains, and both groups noted that the outcome depends on the pairing of mosquito population and virus genotype [79,80]. A single amino-acid change in chikungunya virus alters its use of *Ae. albopictus* [81], so competence measured with one strain does not transfer automatically to the strains likely to be imported. They show what local mosquitoes can transmit, not what they have transmitted.

Table 6. Invasive *Aedes* records, natural infection and vector competence in North Africa

Country	Vector record	Competence or infection evidence	Limitation	Ref.
Algeria	*Ae. albopictus* first documented 2010; reported by 2025 to have colonised northern Algeria to the semi-arid zone	CHIKV reported in abdomens of *Cx. pipiens*, *Cx. perexiguus* and *An. labranchiae* at El Kala, collected 2018–2019; Banna, Sindbis and Usutu viruses also detected	Range from 20 specimens in five districts; no density or seasonality data. Virus in abdomens only, no head or thorax positive; one of three pools confirmed at Ct 35; no sequencing; authors read it as a blood meal. No *Ae. albopictus* was caught: the CDC light traps used are not designed for that species.	[20,70,74]
Morocco	*Ae. albopictus* first recorded, Rabat, 2015	Field-collected strain transmitted CHIKV efficiently (saliva positive by day 3); dengue, Zika and yellow fever viruses less so	One population; laboratory infection.	[71,79]
Tunisia	*Ae. albopictus* first detected in greater Tunis, 2018; spread to several regions by 2023	CHIKV transmission efficiency 25% at 21 days; Zika virus 8.7%; dengue virus 8.3%	One population; single strains; laboratory infection.	[72,73,80]
Libya	No primary record of *Ae. albopictus* or *Ae. aegypti* found; a 2018 larval survey in Tarhuna found seven species, none *Aedes*	None	One local survey.	[82]
Egypt	*Ae. aegypti* re-established nationally from 2018; present on the Red Sea coast and at outbreak sites in Upper Egypt	Natural infection with DENV-1, -2 and -3 on the Red Sea coast; DENV in 16 of 36 pools in Upper Egypt, 2016 (neither is an experimental competence test)	Infection of mosquito pools shows exposure to virus, not competence. Upper Egyptian serotype combinations were not confirmed by sequencing.	[23,64,69]
Sudan	*Ae. aegypti* in most of the country, sustained by household water storage; *Ae. albopictus* first reported in 2022 (three sequenced specimens)	Vector setting of the 2018–19 chikungunya epidemic	*Ae. albopictus* record from one larval survey of four states.	[56,58,60]

Ae. aegypti is the documented vector setting of the Sudanese epidemic and the mosquito found infected with dengue virus both in Egypt's Red Sea governorate and at the Upper Egyptian outbreak sites [64,69]. Its re-establishment in Egypt after decades of presumed absence [23] is the change that makes the Egyptian outbreaks intelligible. The 2022 report of *Ae. albopictus* in Sudan [60] places that species at both the eastern and the western ends of the region. In Libya, a single local larval survey says little about the country as a whole. In Tunisia, *Ae. caspius* and *Ae. detritus* have been examined for a possible role in Zika virus transmission [83]. Table 6 shows a difference in the kind of evidence available: in Egypt and Sudan the mosquitoes carry virus; in Morocco and Tunisia the evidence is of presence and laboratory capacity without detected virus; and in Algeria the one report of arbovirus RNA relevant to these viruses was confined to *Culex* and *Anopheles* abdomens and was read by its authors as a blood meal [20], so it does not show virus carried by a vector.

3.5. Evidence on detection

Detection in the region is limited in four ways. The first is serological cross-reactivity. Neutralizing antibodies were found in about half of the IgG-positive people tested in the Mediterranean Basin [13], and among dengue seroprevalence studies in the Middle East and North Africa only 5% used neutralization testing [7].

The literature is also small. Of 200 WNV studies published in the Mediterranean Basin between 2010 and 2023, 153 came from Southern Europe, 33 from North Africa and 14 from Western Asia [13], and the North African datasets are isolated country-level publications without a shared regional source.

Testing practice is a third constraint. Where arboviral testing has been applied in Tunisia it has returned positives, although at modest rates: WNV RNA in 7 of 79 cerebrospinal fluid samples from suspected cases in 2011–14 [28], three human cases identified through national meningitis surveillance in 2021–22 [48], and, in a series of 353 hospital patients without recorded clinical data sampled between October 2017 and January 2020 across three hospitals, 28.8% IgM-positive, 19.2% IgG-positive and 9.2% positive by RT-PCR [44]. Toscana virus was found in 12.9% of neuromeningeal cases tested in another Tunisian series [15,84] and in 3.8% of tested cases in Algeria [15]. Eight sandfly-borne phleboviruses have been described in North Africa, with antibodies in 15.9% of healthy people [15]. Neurological illness in the region therefore has more than one viral explanation, and where testing is absent such cases

cannot be attributed to any of them. These positivity fractions come from patients selected for testing and are not population rates.

Last comes ascertainment during a declared epidemic. In Sudan's 2018–19 chikungunya epidemic, the reasons to expect under-reporting were specific and documented (a sentinel system built to raise alerts, not to count cases; cost barriers to attendance; limited diagnostics; no private-sector reporting; section 3.3.1), even though the frequently quoted estimate of 487,600 cases assumes the ascertainment fraction instead of measuring it. Together with the Algerian serocatalytic analysis of human serology (section 3.2.3), this is the evidence that bears most directly on events that went unreported. The Algerian mosquito finding does not belong here on present evidence (section 3.3.3); it would count only if confirmed and sequenced.

4. Discussion

4.1. What the evidence entitles each claim to say

Table 7 restates the main claims made about the region in the literature, with the wording each is entitled to under the evidence levels of Table 1.

Table 7. Main claims about arboviruses in North Africa, the wording the evidence supports, and what limits each claim

#	Claim	Wording	Basis	Main limitation
1	WNV circulates in Egypt, Algeria, Tunisia, Morocco and Libya.	Documented (A, B)	Outbreak investigation with sequencing (Tunisia); clinical and neutralization-confirmed evidence (Algeria, Morocco); neutralization-confirmed antibodies in animals (Libya, Egypt); historical outbreaks and serosurveys (Egypt).	Sudan is supported only by serosurveys summarised in a previous review [10].
2	Recurrent human neuroinvasive WNV disease occurs in Tunisia and Algeria.	Documented (A)	Four Tunisian epidemics (1997–2018); Algerian outbreak in 1994 and cases in 2012–2014.	Counts differ between sources; recognised disease only.
3	North African WNV seroprevalence exceeds that of Southern Europe.	Reported (C, pooled)	Pooled 15.4% compared with 2.7% [13].	Neutralization data cover about 7% of IgG-positive people basin-wide; cumulative; confirmed values are lower where measured (6.7% in Algiers; about 5% in Tunisian donors).
4	WNV disease is under-diagnosed in North Africa.	Inferred	Epidemics recognised as clusters; WNV RNA in 7 of 79 cerebrospinal fluid samples in 2011–14 [28] and three human cases through meningitis surveillance in 2021–22 [48]; one hospital series of 353 patients [44]; equine infections detected across 2018–2023; serocatalytic modelling in Algeria suggests unrecognised northern outbreaks [41].	Magnitude unmeasured; a large infection-to-case ratio is expected in any setting; the Algerian result is model-based.
5	Dengue transmission occurs in Upper Egypt and on Egypt's Red Sea and Sinai coasts.	Documented (A)	Dayrout outbreak, Assiut, 2015 (DENV-1, 28 of 118 samples) [63]; Upper Egypt 2016 (31 of 51 patients, 16 of 36 vector pools) [64]; El Quseir outbreak; traveller-based detection; DENV-1, -2 and -3 in *Ae. aegypti* [69].	Much of the coastal evidence is indirect; incidence unknown; the Nile Delta and Mediterranean coast are untested.
6	Dengue and chikungunya occur recurrently in Sudan.	Documented (A)	Chikungunya epidemic 2018–19, with 48,763 cases reported across nine months in Kassala and Red Sea States; dengue and chikungunya in Tandalti, 2022 (63 of 773 suspected cases) [61]; dengue in Kassala, 2023–24, with all four serotypes (276 of 548 febrile patients) [62].	The estimate of 487,600 people affected in 2018–19 is the reported count divided by an assumed 10% ascertainment, so it encodes under-reporting rather than measuring it [58].
7	*Ae. albopictus* is present in Algeria, Morocco and Tunisia, and in Sudan.	Present (D)	First documentation in 2010, 2015 and 2018 (Maghreb); 2022 in Sudan (three sequenced specimens); several regions of Tunisia by 2023; northern Algeria colonised to the semi-arid zone by 2025 [74].	First-documentation dates, not establishment; range described for Algeria from 20 specimens, but no density or seasonality data for Algeria or Morocco.
8	Local *Ae. albopictus* can transmit chikungunya virus, and less efficiently dengue virus, in the laboratory.	Shown in the laboratory (D)	Morocco and Tunisia.	Single populations and strains; laboratory infection.
9	Chikungunya virus has been detected in field-collected mosquitoes in Algeria.	Reported (D), unconfirmed	Multiplex screening of 180 pools from 1,658 mosquitoes collected at El Kala in 2018–2019; first report of CHIKV in the country [20].	Virus in abdomens only, with no head or thorax positive; one of three pools confirmed by RT-PCR at Ct 35 and the other two not confirmed; no sequencing; the authors interpret it as a blood meal and rule out *Culex* and *Anopheles* as vectors. No human case.
10	Locally acquired dengue and chikungunya have not been reported in Morocco, Algeria, Tunisia or Libya.	Absence of reports	No report found through 3 October 2026.	Sensitivity of detection unknown; no recent serological baseline; now qualified by claim 9.
11	The Maghreb is entering the window in which European regions had their first outbreaks.	Inferred (E)	European time-to-event analysis [4].	Derived from EU regions with importation as a covariate; importation into the Maghreb unmeasured; 8 to 16 years without a reported outbreak already exceed the recent European interval (under 5 years) but not the 1990s interval (25 years).

Claims about occurrence (rows 1, 2, 5, 6, 7 and 8) rest on levels A to D and can be stated without qualification beyond their stated

limitation. Row 9 is different in kind: it reports a single unconfirmed detection whose own authors interpret it as a blood meal, and it should be cited with those qualifications attached or not at all. Claims about magnitude and about the future (rows 3, 4 and 11) rest on levels C and E or on inference, and they are the claims repeated most often. Row 10 records an absence of reports, whose meaning depends on a detection sensitivity that has not been measured.

Two claims carry most of the weight of the view that the region's apparent quiet hides transmission: the excess of seroprevalence over Southern Europe (row 3) and the transferability of the European time-to-outbreak interval (row 11). Each rests on a single source, one systematic review of WNV in the Mediterranean [13] and one European time-to-event analysis [4], and neither has been tested against North African data.

4.2. Under-detection or low transmission

For WNV the question concerns magnitude, since transmission and recurrent human disease are documented. Under-diagnosis is likely: the Tunisian and Algerian epidemics were recognized when clusters of severe disease appeared, enzootic circulation continued between epidemics [36], 11 of 25 suspected equine samples collected across 2018 to 2023 were positive [37], laboratory testing of suspected cases in Tunisia has returned positives in both cerebrospinal fluid and hospital series [28,44,48], and serocatalytic modelling suggests northern Algerian outbreaks that were not recorded clinically [41]. The size of the gap cannot be stated, for the reasons given in section 3.2.3. The claim that North Africa combines the highest seroprevalence with the fewest reported cases in the Mediterranean is better read as a hypothesis generated by pooled crude data than as an established discrepancy.

For dengue and chikungunya in the Maghreb, two explanations fit the data and predict different findings. If transmission has occurred without being noticed, resident people without travel history should carry neutralizing antibodies and febrile patients tested for these viruses should be positive. If introductions have been rare or have not coincided with adequate vector density, both would be near zero. No source has measured either quantity in the resident population, so the absence of human reports cannot by itself discriminate between the two explanations. The Algerian mosquito finding [20] does not discriminate between them either. Because the virus was confined to abdomens and the authors read it as a blood meal, the result is equally compatible with an infected host present at El Kala and with a weak or spurious signal at the detection limit; it says nothing about whether virus is circulating in a vector population. The obvious next step is the same under either reading: a serosurvey of residents at El Kala.

4.3. The European interval and its transferability

The European interval invites a direct comparison. In the analysis by Farooq and colleagues the interval from first establishment of *Ae. albopictus* in an EU region to the first outbreak fell from 25 years to under 5, in models that included imported cases, health-care expenditure and land use [4]. In the Maghreb, first documentation dates from 2010, 2015 and 2018, so by 2026 sixteen, eleven and eight years had passed without a reported outbreak. If the recent European interval applied unchanged, all three countries would already be beyond it; the interval seen in the 1990s would not yet be exceeded in any of them (Fig. 2). Several explanations are possible: lower importation pressure, lower vector density, a climate that is less favourable in parts of the region, or transmission that was missed. Nor is time since first documentation a clean measure of time since establishment (section 3.4): earlier, undetected arrival would lengthen the true interval, and a first detection without a breeding population would shorten it. The available evidence does not rank these explanations, and the transferability of the European interval to the Maghreb has not been tested.

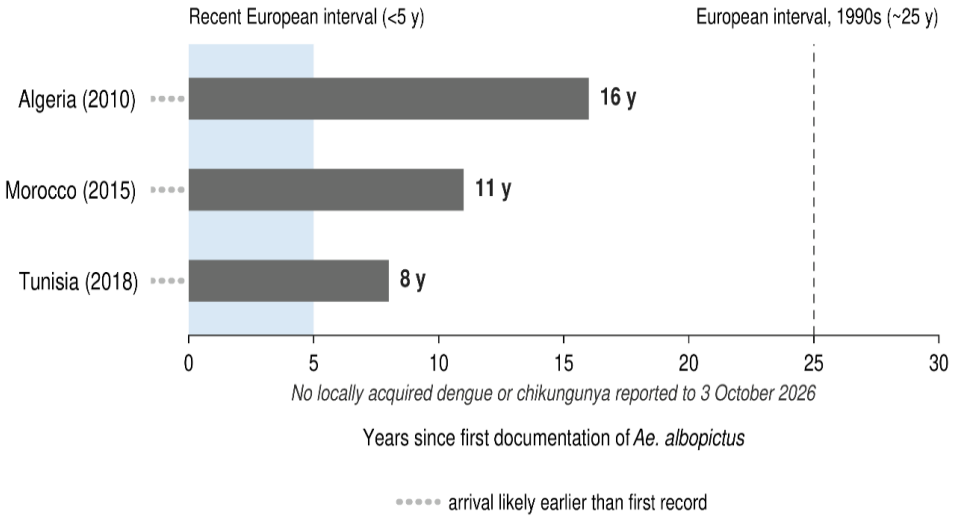


Fig. 2. Years elapsed without a reported locally acquired dengue or chikungunya outbreak since the first documentation of *Aedes albopictus* in Algeria (2010), Morocco (2015) and Tunisia (2018), counted to the evidence cut-off of 3 October 2026, set against the interval from establishment of the species to the first autochthonous outbreak in European Union regions (about 25 years in the 1990s, under 5 years recently).

The documented burden is unevenly distributed. Dengue and chikungunya transmission is documented at the eastern margin of the region, in Sudan and in Egypt, where it covers Upper Egypt as well as the Red Sea and Sinai coasts, and where *Ae. aegypti* is established and household water storage sustains it [23,58]. The Maghreb has an invasive vector and laboratory competence but no documented human transmission; the one entomological report there is an unconfirmed, abdomen-only detection in *Culex* and *Anopheles* that its authors attribute to a blood meal [20]. WNV transmission is documented across the region.

No source links virus strains between the eastern and western parts of the region. The Sinai DENV-2 sequences were related to South Asian strains [68], which suggests introduction from outside North Africa, and no phylogeographic study connects Sudanese or Egyptian dengue or chikungunya viruses to the Maghreb. The Algerian chikungunya detection was not sequenced, so it cannot be placed relative to the Sudanese Indian Ocean lineage strains, and in any case it came from mosquito abdomens rather than from a human or vertebrate case. Movement of virus between the two ends of the region by sea or overland travel is plausible but has not been examined.

4.4. Strengths and limitations

The main strength of this review is that it sorts the evidence for six countries and three viruses by what each source can support, so that a serological signal from an unconfirmed assay is not read alongside a laboratory-confirmed outbreak as though the two carried equal weight. Its limitations follow from the same design. Assays, populations and case definitions vary between studies, so figures in Tables 3 to 5 are not directly comparable. Several central figures depend on a single source: the pooled WNV seroprevalence and the fraction of positives confirmed by neutralization on one meta-analysis [13], the Sudanese epidemic estimate on one cohort report [58], the European time-to-outbreak interval on one analysis [4], and the Algerian chikungunya detection on one entomological study [20]. None of them has been reproduced with independent North African data. A second, independent pooled estimate for WNV in Africa has since been published, giving a frequency-weighted seroprevalence of 17.96% (95% CI 12.50–24.13) across 61 studies in 20 countries and a peak human value of 55% in Egypt [12]; it is continental rather than North African and uses a different pooling method, so it corroborates the order of magnitude without replicating the Mediterranean comparison.

Sudanese WNV evidence was taken from a previous systematic review rather than from the primary studies [10]. Some serosurvey figures in Table 4 were taken from the syntheses that report them and were not re-extracted from the primary studies, although the Libyan human figure has been traced to its primary source [21]. One frequently quoted Tunisian series, of 316 hospitalised suspected cases, was excluded because it could not be traced to a primary study: it appears in a regional synthesis without an attributable source report, and figures that cannot be checked against a primary record are not used here. Counts for some events, such as the Tunisian epidemics of 1997 and 2003, differ between sources and are given as ranges.

The search was iterative, drew on three databases, and could have missed local-language reports, theses and ministry bulletins, which are poorly indexed. Sources were classified by design and assay and were not scored with a formal risk-of-bias instrument. The evidence base is also concentrated in a small number of research groups,

which limits its independence: the Algerian sources cited here [20,31,40,41,74] come largely from one network centred on the Institut Pasteur d'Algérie and its collaborators, and the Tunisian entomological and competence sources [46,72,73,80,83] come largely from one group at the Institut Pasteur de Tunis. Agreement between such sources is weaker evidence than agreement between independent ones. The evidence cut-off is 3 October 2026, and the position for dengue and chikungunya in the Maghreb could change with a single locally acquired case.

4.5. Research gaps

The gaps below are specific measurements that would decide between the two readings in section 4.2. They are ordered by how much each would change the interpretation, and all could be done with methods already in use in the region.

First, a virus-specific, neutralization-confirmed serosurvey of dengue and chikungunya in resident Maghreb populations. No single study would be more informative. Every statement about whether dengue and chikungunya have circulated unnoticed in Morocco, Algeria or Tunisia currently rests on the absence of clinical reports, and no source has measured antibody prevalence in residents without travel history. El Kala, where the single chikungunya detection was made [20], is the obvious place to start, because a positive result there would connect that detection to human exposure and a negative result would constrain what the detection can have meant. Neutralization confirmation is not optional: the two human series in which both ELISA and neutralization were applied give a ratio of roughly 1.5 to 3 between unconfirmed and confirmed WNV prevalence (section 3.2.2), and although that figure is specific to WNV, it shows how far an unconfirmed assay can depart from a confirmed one in this setting.

Second, density and seasonality of *Ae. albopictus* in Algeria and Morocco. Range is now partly described for Algeria [74], but transmission is governed by abundance and by the length of the season during which abundance is high, and neither has been measured in either country. Longitudinal ovitrap or adult-trap series at a small number of sites, reported by month, would allow the European time-to-outbreak interval [4] to be tested against a comparable quantity instead of against a date of first documentation. Until that exists, the comparison in section 4.3 cannot be made rigorously.

Third, confirmation and sequencing of any future arbovirus detection in North African mosquitoes. The Algerian chikungunya result shows what is at stake: virus in abdomens only, one of three pools confirmed at Ct 35, and no sequence, which together leave the finding unable to support any conclusion about transmission [20]. Repeating the screening with confirmatory RT-PCR on every positive pool, separate testing of head and thorax, and sequencing of confirmed positives would establish whether there is anything there at all; and because the original sampling used CO_2-baited CDC light traps, which caught no *Ae. albopictus*, traps designed for that species should be used alongside them. Once published, an unconfirmed detection tends to be cited without its caveats.

Fourth, a phylogeographic analysis spanning the region. Serotype-level and sequence data now exist at both ends: DENV-1, -2 and -3 in Egyptian *Ae. aegypti* [69], DENV-2 from the Sinai related to South Asian strains [68], chikungunya virus from the Sudanese epidemic, and three sequenced *Ae. albopictus* specimens from Sudan [60]. No analysis places these in a common phylogeny with any Maghreb material. Whether the eastern and western parts of the region are epidemiologically connected or are separately exposed to introductions from outside is therefore unknown, and the two possibilities imply different surveillance priorities.

Fifth, the infection-to-reported-case ratio for WNV in North Africa. Section 3.2.3 shows that the shortfall in reported cases cannot be quantified because neither the regional incidence of neuroinvasive disease nor the local ratio of infections to recognized cases has been measured. A household or clinic-based study pairing confirmed incidence with confirmed seroprevalence in one well-characterized area (the Tunisian Sahel and the Moroccan Gharb are the best-documented candidates) would convert the most-repeated claim about the region from an inference into an estimate.

Sixth, replication of the two single-source claims that carry the interpretation. The excess of North African over Southern European seroprevalence rests on one meta-analysis [13] and the shrinking time-to-outbreak interval on one European analysis [4]. Neither has been reproduced with North African data, and both are cited as though settled.

Seventh, the country-specific deficits. Libya has one human serological dataset, from a convenience sample in one city in one season [21], and no primary record of either invasive *Aedes* species; a national larval and adult vector survey and a neutralization-confirmed human serosurvey would move the country from the weakest evidence base in the region to a usable one. For Sudan the WNV evidence has been read only through a previous systematic review [10] and the primary studies have not been appraised, while the dengue and chikungunya record now spans 2018–19, 2022 and 2023–24 [58,61,62] without any population-based incidence estimate for any of those events. For Egypt, transmission is documented in Upper Egypt and on the Red Sea and Sinai coasts but not measured in either, the Nile Delta and Mediterranean coast are untested, and the finding that dengue detection in vectors peaked in December and was absent between June and September [69] needs confirmation in a second season.

4.6. Diagnostic breadth beyond the arboviruses

Section 3.5 argued that undifferentiated febrile and neurological illness in this region has more than one viral explanation, and that cases which are not tested cannot be attributed to any of them. An event outside the region during the preparation of this review shows how that argument can fail in practice. Bundibugyo virus, an orthoebolavirus first identified in western Uganda in 2007 [85], re-emerged in Ituri Province, Democratic Republic of the Congo, in April 2026. A cluster of deaths investigated in Bunia on 30 April 2026 tested negative for Zaire ebolavirus on a species-specific assay, and the causative agent was identified as Bundibugyo virus only by whole-genome sequencing two weeks later [86]. WHO declared a public health emergency of international concern on 17 May 2026, and by 6 October 2026 the Democratic Republic of the Congo had recorded 8,728 confirmed cases and 4,205 confirmed deaths, a crude case-fatality ratio of 48.2% [89]; imported cases were subsequently confirmed in Kenya and Nigeria [90]. Two features bear on detection rather than on occurrence. An assay directed at the species that was expected returned a negative result for a divergent one, and Bundibugyo virus disease presents in most patients as a non-specific febrile and gastrointestinal illness, with haemorrhagic signs uncommon at presentation [87,88].

What this does not establish should be stated plainly. No filovirus has been detected in humans, animals, vectors or the environment in Morocco, Algeria, Tunisia, Libya, Egypt or Sudan, and this review makes no claim of filovirus circulation in North Africa. Filoviruses are not arthropod-borne, so the vector evidence assembled in section 3.4 has no bearing on them, and no evidence level under Table 1 can be assigned to them for any of the six countries. The historical outbreaks that gave Sudan virus its name occurred at Nzara, Maridi and Yambio, in territory that is now South Sudan [91], and therefore fall outside the region as defined here. The transferable point is methodological, and it restates for a non-arboviral pathogen what Table 1 asserts for the arboviruses: a negative result constrains only what the assay was designed to detect. Where acute febrile or neurological illness is investigated with a panel built on the pathogens a region expects, the composition of that panel sets a ceiling on what can be found, and the low reported arboviral burden of the Maghreb was established with panels of exactly that kind (section 3.5). This sharpens the case made in section 4.7 for widening diagnostic panels, and the case made in the third research gap of section 4.5 for confirming and sequencing unexpected results rather than reporting them alone.

4.7. Implications for practice

Two implications follow for practice while these questions remain open. The first concerns testing. Neurological illness in this region has several possible viral causes (WNV, Toscana and other sandfly-borne phleboviruses, Usutu virus), and the Tunisian and Algerian series show that testing such patients returns positives for more than one of them [15,44,84]. Arboviral testing of neurological presentations, with neutralization confirmation where serology is used, could add information quickly, because it uses patients who already reach hospital. The second concerns timing. Both the Mediterranean suitability model [53] and the Egyptian vector data [69] indicate that the transmission window in parts of the region does not coincide with the European summer season; vector control and surveillance scheduled on a European calendar may be mistimed, and the question should be checked against further seasons before any schedule is changed.

Some implications fall outside human medicine. Equine and avian surveillance has supplied most of the confirmed WNV evidence for Morocco, Libya and Algeria [33,35,42], which argues for a formal reporting link between veterinary and human public health services. For entomology, the region offers an unusual natural experiment: a competent invasive vector established for 8 to 16 years in three countries without a reported outbreak, which is informative about the conditions under which establishment does not lead to transmission. For climate and health research, North Africa remains largely absent from the datasets on which Mediterranean projections are built, so those projections are extrapolations into the region rather than descriptions of it.

5. Conclusions

WNV transmission is documented in Egypt, where the virus was isolated from human sera in 1951, and in Algeria, Tunisia, Morocco and Libya; recurrent human neuroinvasive outbreaks are documented in Tunisia and Algeria. Dengue and chikungunya transmission is documented in Sudan, recurrently in 2018–19, 2022 and 2023–24, and in Egypt, in Upper Egypt and on the Red Sea and Sinai coasts. Neither virus has been reported as locally acquired in Morocco, Algeria, Tunisia or Libya, although *Ae. albopictus* has been documented in the first three since 2010, 2015 and 2018 and local populations transmit chikungunya virus in the laboratory. One Algerian report of chikungunya virus in mosquito abdomens remains a single unconfirmed signal and does not change that position.

The often-cited contrast between high North African WNV seroprevalence and few reported cases rests largely on assays without neutralization confirmation and cannot be quantified from the available data. The European interval between vector establishment and first outbreak has not been tested in the Maghreb and cannot be applied to it without data on importation and vector density. Reported case counts are a lower bound of unknown tightness: in Sudan's 2018–19 chikungunya epidemic the reasons for under-reporting were documented, but the widely quoted tenfold figure is an assumption applied to the reported count rather than a

measurement of the gap. On their own, case counts from this region cannot show whether transmission is low or high; the measurements set out in section 4.5 can.

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Declarations

Author contributions: F.A.A.: Conceptualization, Methodology, Investigation (literature search and screening of West Nile virus sources), Data curation (Tables 1, 3, 4 and 7), Visualization (Fig. 1), Writing – original draft (Abstract, Introduction, Methods, sections 3.1, 3.2 and 3.5, Conclusions), Project administration. G.F.M.: Conceptualization, Methodology, Investigation (literature search and screening of dengue, chikungunya and vector sources), Data curation (Tables 2, 5 and 6), Visualization (Fig. 2), Writing – original draft (sections 3.3, 3.4 and 4), Validation (reference checking against PubMed). Both authors contributed equally to the work, and both reviewed, edited and approved the final manuscript.

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