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A historical perspective on arboviruses of public health interest in Southern Africa

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ABSTRACT

Arboviruses are an existing and expanding threat globally, with the potential for causing devastating health and socioeconomic impacts. Mitigating this threat necessitates a One Health approach that integrates vector surveillance, rapid disease detection, and innovative prevention and control measures. In Southern Africa, limited data on the epidemiology of arboviruses, their vectors, and their hosts prevent an effective response. We reviewed the current knowledge on arboviruses in Southern Africa and identified opportunities for further research. A literature search was conducted to identify studies published on arboviruses in 10 tropical and temperate countries of the Southern African Development Community (SADC) from 1900 onward. We identified 280 studies, half (51.1%) originating from South Africa, that described 31 arboviral species, their vectors, and their clinical effects on hosts reported in the region. Arboviral research flourished in the SADC in the mid-20th century but then declined, before reemerging in the last two decades. Recent research consists largely of case reports describing outbreaks. Historical vector surveillance and serosurveys from the mid-20th century suggest that arboviruses are plentiful across Southern Africa, but large gaps remain in the current understanding of arboviral distribution, transmission dynamics, and public health impact.

KEYWORDS

Arboviruses; vectors; public health; zoonoses; Southern Africa

Introduction

The effects of emerging and reemerging pathogenic infections can be catastrophic and are therefore of increasing global health concern. Prime recent examples include the global coronavirus pandemic and the Marburg virus outbreak in Tanzania [1]. Naturally, these infections result from cross-species viral transmission from animals to humans. Arthropod-borne viruses (arboviruses), which have their sylvatic cycle in vertebrates and are capable of transmission from animals to humans, are notable causes of many vector-borne human and veterinary diseases including zika (ZIKV), chikungunya (CHIKV), yellow fever (YFV), and dengue (DENV) [2,3]. Arboviruses' global spread has been attributed predominantly to increased proximity between sylvatic hosts and immunologically naive hosts, in the presence of competent hematophagous arthropod vectors [4]. This triad forms the basic conditions necessary to facilitate viral transmission and spillover events.

Although arboviruses frequently circulate in tropical, subtropical and temperate regions of the world, most arboviral research conducted in the African continent has been concentrated in Central, East, and West Africa, with relatively little research done in Southern Africa [5]. Anthropogenic perturbances and environmental fluctuations contribute to dynamic shifts in the geographic distribution of arboviral reservoirs and their vectors into novel areas [5,6]. Climate change has also led to recent changes in vector behavioral patterns [7]. Consequently, arboviral disease outbreaks have expanded and may continue to spread to new areas, with devastating results [8,9]. In 2022, to support the global response to arboviruses, the World Health Organisation (WHO) established the Global Arbovirus Initiative, which aims, in part, to address gaps in arboviral research [10]. This scoping review aims to aggregate and assess the knowledge achieved, and the gaps that remain in arboviral research in Southern Africa. Addressing these gaps will be critical to improving disease surveillance and emergency response systems to mitigate the detrimental health and economic effects of future arboviral outbreaks.

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Arboviruses

Arboviruses are obligate viruses that depend on hematophagous arthropod vectors for transmission between vertebrate hosts. The term arbovirus is not a taxonomic indicator but refers to a polyphyletic group of RNA viruses and one DNA virus, African Swine Fever virus [11]. RNA viruses lack a proof-reading RNA-dependent RNA polymerase, resulting in high mutation rates (averaging one mutation per genome replication round) that spread rapidly in large populations [12,13]. Consequently, arboviruses have vast genetic variability that has been associated with high adaptive and phenotypic plasticity and with the emergence of novel strains [14–16]. Additionally, a myriad of external factors, simply categorized as environmental and anthropogenic activities, have influenced and continue to contribute to the global distribution and transmission of arboviruses (Figure 1). Generally, arboviruses are host specific, but opportunistic virus species arise in response to environmental perturbance, adapt to new hosts, and become catalysts of both human and veterinary spill over events [8,18].

Factors such as urbanization, migration, and climate change have contributed to the expanded and overlapping distribution of arboviruses, their vectors, and hosts [4,6,9]. Particularly concerning are the Flaviviridae (DENV and ZIKV) and Togaviridae (CHIKV) families, which can cause deadly outbreaks in humans [19–22]. Clinical manifestations of these infections in humans range from asymptomatic cases to severe illness and even death. As mild symptoms resemble other common infections such as malaria and influenza, misdiagnosis becomes common and may lead to incorrect treatment [5,23]. Conversely, severe symptoms can manifest as febrile illness, neurological syndromes, debilitating arthralgia, and/or hemorrhagic fever, imposing a significant socioeconomic burden on affected communities and countries [24–26]. Developing nations in sub-Saharan Africa, where competent vector species are abundant, may bear a greater economic impact from arboviral disease outbreaks [27–29].

Transmission of arboviruses

Pathogenesis begins with transmission, a process that facilitates the perpetual preservation of a pathogen's genetic specificity and identity [30]. Several transmission mechanisms, broadly categorized as either non-biological or biological, exist for arboviruses. In the first category, an opportunistic species enters a susceptible host directly, leading to infection. The latter category requires the presence of both a competent vector and a susceptible host. Unsurprisingly, regardless of the mode of transmission, pathogens use any means necessary to ensure their survival. In general, arboviral transmission results in an array of interactions with direct pathologic outcomes that determine the overall eco-epidemiological impact of these viruses [2,31].

Non-biological transmission

Direct transmission is a widespread non-biological transmission mechanism amongst major arboviral groups as it is versatile and facilitates arboviral entry through innumerable circumstances (e.g. contamination of food and beverages; behaviors that cause injury, abrasions, and lesions) [3].

Another form of non-biological transmission is mechanical transmission, in which viral material is spread via contact with contaminated arthropod mouthparts. Mechanical transmission is assumed to have arisen independently and simultaneously with the evolution of hematophagy in arthropods [3]. Transmission efficiency depends on the arthropod vector's feeding behaviors and patterns. Compared to insects, acarines (ticks) are less efficient mechanical transmitters as they feed for long uninterrupted periods and tend to remain on the same host [3]. Effective mechanical transmission also requires high virus titers due to low blood volumes on mouthparts [3]. Mechanical transmission occurs with greatest efficiency when vector and host populations are densely concentrated [3].

Biological transmission

Successful biological transmission of pathogens hinges on the complicated network formed by the

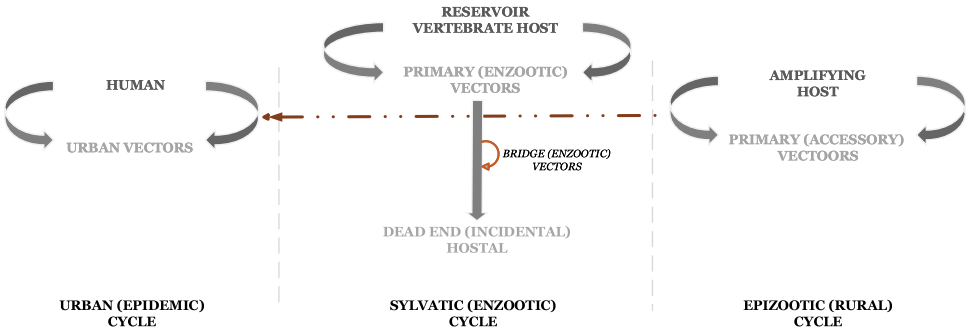

Figure 1. A schematic diagram of arboviral cycles. The schematic diagram adapted from Go et al. [17] illustrates the replicative cycles of arboviruses, how each cycle is maintained and the possible interaction between cycles.

triad of pathogen, vector, and host. Interactions are largely influenced by ecological factors that either promote or demote transmission. Pathogens must adapt to at least one of the two possible modes of biological transmission: vertical and horizontal [32]. Vertical transmission (also known as hereditary or transovarial transmission) transfers pathogenic agents from parent to progeny, whilst horizontal transmission refers to all other non-parental modes of transmission (e.g. sexual, vector-borne) between hosts and/or vectors [5,33]. These two modes can and often do occur in combination (mixed-mode transmission) to increase survivability, especially during adverse events [34]. Pathogens tend to use vertical transmission to persist in an environment when conditions for available hosts are unfavorable; in more optimal conditions, pathogens can adopt horizontal transmission, which allows for increased genetic variation by introducing mutations through recombination and reassortment [33]. For arboviruses, the pathogen-vector-host network is primarily maintained by horizontal transmission [5,33,35].

Hematophagy is a vital component of the arthropod gonotrophic cycle, as blood meals provide a source of protein. Arthropods may become infected with arboviruses when they ingest a blood meal from an infected vertebrate. Viral transmission to a feeding arthropod is more likely when the vertebrate has a higher blood concentration of virus and a longer duration of viremia [3]. The arbovirus develops in the arthropod, and viral transmission from the arthropod vector to a new, uninfected vertebrate may transpire during blood feeding. This becomes a naturally maintained cycle [35]. It is worth noting that arboviral transmission cycles are disparate because a vector's biology, host range, geographical spread, and dispersal patterns also affect the geographic distribution of arboviruses [11].

Three transmission cycles have been well described: enzootic sylvatic, epizootic, and urban cycles. In the enzootic sylvatic cycle, arboviruses amplify within and are transmitted to the sylvatic host by a primary vector. The virus can be harbored indefinitely in the sylvatic host, enabling the virus to persist at low concentrations within sylvatic populations. Reinfection occurs with little to no ill effect on the sylvatic host. In addition, in the presence of an enzootic and/or bridge vector (an arthropod that has a broad host range and facilitates transmission to nearby susceptible populations), arboviral transmission to a dead-end or incidental host is possible [6,17]. This incidental transmission is termed a spillover event. Humans are not central in this transmission cycle; they often are dead-end hosts, incapable of transmitting the virus back to its definitive or sylvatic host. However, when human populations are capable of re-infecting wild populations, a spillback event may occur [17]. In summary, humans could be infected during sylvatic amplification when they enter the forest for their activities, or directly by some vector species that could enter and feed in villages, even indoors. This has been demonstrated in Senegal, for example [36]). Moreover, the co-feeding of vectors on the same infected host may result in vector-to-vector transmission and has been at least described for West Nile virus (WNV) [2,37,38].

In the epizootic cycle, transmission occurs amongst domestic and non-domestic animals via a primary or accessory arthropod vector. Veterinary outbreaks occur when arboviruses are amplified within a domestic animal population with viral levels high enough to infect vectors. Human populations in proximity to these animals may be at risk of infection [11,17]. Furthermore, humans with high viremia levels can themselves become amplification hosts, serving as the viral source for mosquitoes that then become the main vector in an urban cycle. It is the urban cycle that temporarily exploits humans as amplification hosts during epidemics and thus has a global impact. Arboviruses like CHIKV, YFV, and DENV use this cycle, causing a varying range of clinical manifestations in humans that can result in high morbidity and mortality [11,39,40].

The greatest threat to pathogens is herd immunity. This occurs when a high proportion of community members develop protective antibodies against a disease, leading to a decline in the spread of the disease between individuals [41,42]. To circumvent this threat, arboviruses have devised three major strategies. In the first, arboviruses move to a novel area that has a small, immunologically naïve population with low immunity. The second strategy makes use of vertebrate species with shorter life spans and high fecundity rates (e.g. rodents, birds) to ensure an incessant supply of susceptible hosts [43]. The last strategy adopted is immune evasion. Through immune evasion, viruses hide in specific tissues and organs, remaining infectious for undetermined periods regardless of high concentrations of antibodies circulating within the vertebrate's blood [2,44]. Often, the first and second strategies are associated with acute, veterinary infections whilst immune evasion is characteristic of chronic infections in humans [30].

Arboviral vectors

Hematophagy in arthropods has independently arisen in four insect orders (Anoplura, Diptera, Hemiptera and Siphonaptera) and among ticks (order Ixodida) [3]. Vector feeding behavioral patterns have evolved to synchronize feeding activities with times optimal for successfully attaining a blood meal from a vertebrate host. Consequently, blood feeding behaviors vary. Moreover, vectors possess host preferences influenced by genetic and ecological factors. Notably, certain

vector species exhibit opportunistic behaviors, adjusting their host selection depending on the availability of vertebrates in a given ecological environment [40,45].

The primary arbovirus vectors are mosquito species from the *Culex* and *Aedes* genera [6]. *Culex* species are primarily zoophilic. In particular, they are the main ornithophilic mosquito species [46,47], and thus have been associated with veterinary arboviral disease outbreaks [45]. However, they do also act as bridge vectors for WNV causing human epidemics [48]. *Aedes* species exhibit variable degrees of feeding propensity and host specificity, with both zoophilic and anthropophilic tendencies, and are thus associated with most human outbreaks [45]. Globally, *Ae. aegypti* and *Ae. albopictus* are the dominant arboviral vectors in tropical and Mediterranean regions of the world [49]. *Aedes aegypti* exhibits particular phenotypic and behavioral plasticity that enables it to thrive in human populated settlements and modern urban environments. As a result, most human arboviral outbreaks in Africa, including Southern Africa, are caused by *Ae. aegypti* [50]. Notably, mosquito species have different vector competencies for transmitting viruses to humans [51].

Sylvatic hosts of arboviruses

In the natural habitats of non-human primates (NHP), arboreal mosquitoes transmit arboviruses from infected to naïve animals through a sylvatic transmission cycle. By the same modality, arbovirus infections transmitted by highly anthropophilic mosquitoes can spread rapidly amongst humans [40]. Sylvatic hosts have been well documented for DENV, ZIKV, CHIKV and YFV whilst hosts for other arboviruses are still yet to be determined [52]. Urbanization and deforestation are two critical factors that have increased habitat loss for wildlife and have led to greater co-occurrence between NHP and humans. These factors create interactions with biomedical and epidemiological consequences [53]. Among NHP of interest, *Chlorocebus pygerythrus* (vervet monkey) has an extensive geographical distribution in Africa, including the Southern regions of the continent, which encroaches into human territories [54]. Vervet monkeys have great biomedical importance as natural hosts of Simian Immunodeficiency Virus, a close relative of Human Immunodeficiency Virus (HIV) [53,55]. One of the first reports alluding to the possibility of vervet monkeys harboring arboviruses followed a viral endemic that erupted from lab animals in Germany [56]. The association between arboviruses and vervet monkeys aroused global interest, resulting in further characterization of members of the *Chlorocebus* genus from various African countries. Using Southern African vervet monkey biomaterials, McIntosh identified myriad viral species including Marburg, CHIKV and Bunyamwera virus (BUNV) [57]. McIntosh's discovery was, and still is, pivotal to epidemiological and viral research, as the findings show that NHP harbor many unidentified arbovirus species. The other known species of NHP from Southern Africa found infected with arboviruses (particularly CHIKV) is the Cape baboon (*Papio ursinus*) [52]. These baboons are widespread in the region and also live in close proximity to humans [57,58].

Birds have also been found to be sylvatic hosts of some zoonotic pathogens including arboviruses [4]. However, birds are less susceptible to pathogens because of their high body temperature, specificity to strains (among arboviruses, predominantly associated with WNV transmission) and excellent immune system [59,60]. Despite their lower susceptibility, birds are fundamental to the global spread of zoonotic pathogens by acting as natural hosts, reservoirs or amplifying hosts. Birds' various roles in arboviral transmission have yet to be thoroughly investigated, but in Southern Africa, they have been identified as sylvatic and/or amplifying hosts of Shuni (SHUV), Usutu (USUV), WNV, BUNV and Sindbis (SINV) viruses [5].

Impact of arboviruses in Southern Africa

A plethora of arboviruses of African origin currently circulate on the continent, yet the public health impacts remain undetermined in many respects (Table 1). Understanding arboviral transmission dynamics and improving surveillance for the timely detection of arboviral diseases are key components of an effective, integrated response [27]. Extensive reviews have been conducted on arboviral diseases and outbreaks in Central, East and West Africa by Gould *et al*. [61], Mbanzulu et al. [62], Marchi *et al*. [63], Nyaruaba et al. [64] and Agboli et al. [65]. However, comprehensive data from the Southern African Development Community (SADC), a region that consists of 16 countries in Southern Africa, are lacking [66]. This knowledge gap hinders public health preparedness and implementation of effective emergency response strategies in Southern Africa, with the potential for significant health and economic consequences [27,28]. Southern Africa has demonstrated a greater level of preparedness for zoonotic diseases compared to other sub-Saharan countries, but current emergency response strategies are still limited in scope, and current capacities for arboviral surveillance, detection, and control remain inadequate [28,67]. The SADC countries, like other sub-Saharan countries, already faces challenges from other pathogens including HIV/Acquired Immunodeficiency Syndrome (AIDS), malaria, and tuberculosis which further strain the ability to respond to potential new arbovirus outbreaks. Consequently, strategies against 'pathogen x' remain rudimentary. Expanded research on arboviruses in the

Table 1. Polyphyletic classification of arboviruses.

Nucleic acid	Family	Genus	Examples of species
Positive sense single stranded RNA	Flaviviridae (4 genera and 89 species)	Alphavirus	Chikungunya virus
			O'nyong-nyong virus
	Flaviviridae (4 genera and 89 species)	Flavivirus	Yellow fever virus
			Dengue virus
			West Nile virus
		Hepacivirus	Hepatitis C virus
			Hepatitis B virus
		Pegivirus	Pegivirus A
			Pegivirus B
		Pegivirus	Pegivirus D
			Pegivirus E
Negative sense single stranded	Bunyaviridae (5 genera and 95 species)	Othorbunyavirus	Bunyamwera virus
			Bwamba virus
		Nairovirus	Dugbe virus
			Crimean-Congo haemorrhagic fever virus
		Phlebovirus	Rift Valley Fever virus
			Sandfly fever Naples virus
		Hantavirus	Hantaan virus
			Puumala virus
		Tospovirus	Tomato spotted wilt virus
			Impatiens necrotic spot virus
	Orthomyxoviridae (7 genera and 9 speices)	Thogotovirus	Dhori thogotovirus
			Thogoto thogotovirus
		Alhpainfluenzavirus	Influenza A virus
		Betainflunezavirus	Influenza B virus
		Deltainfluenzavirus	Influenza D virus
		Gammainfluenzavirus	Influenza C virus
		Isavirus	Salmon isavirus
		Quaranjavirus	Johnston Atoll quaranjavirus
			Quaranfil quaranjavirus
	Rhabdoviridae: Subfamily Alpharhabdovirinae (26 genera and 169 species)	Almendravirus	Balsa almendravirus
			Coot Bay almendravirus
		Alphanemrhavirus	Xingshan alphanemrhavirus
			Xingzhou alphanemrhavirus
		Alphapaprhabdovirus	Hubei alphapaprhabdovirus
			Pararge alphapaprhabdovirus
		Alpharicinrhavirus	Blanchesco alpharicinrhavirus
			Bole alpharicinrhavirus
		Arurhavirus	Inhangapi arurhavirus
			Xiburema arurhavirus
		Barhavirus	Bahia barhavirus
			Muir barhavirus
		Caligrhavirus	Caligus caligrhavirus
			Lepeophtheirus caligrhavirus
		Curiovirus	Iriri curiovirus
			Iriri curiovirus
		Ephemerovirus	Ephemerovirus
			Hayes ephemerovirus
		Hapavirus	Flanders hapavirus
			Joinjakaka hapavirus
		Ledantevirus	Fikirini ledantevirus
			Fikirini ledantevirus
		Lostrhavirus	Hyalomma lostrhavirus
			Lonestar lostrhavirus
		Lyssavirus	Rabies Lyssavirus
			Taiwan bat lyssavirus
		Merhavirus	Merida merhavirus
			Tritaeniorhynchus merhavirus
		Mousrhavirus	Moussa mousrhavirus

(*Continued*)

Table 1. (Continued).

Nucleic acid	Family	Genus	Examples of species
		Ohlsrhavirus	Culex ohlsrhavirus
			Ohlsdorf ohlsrhavirus
		Perhabdovirus	Perch perhabdovirus
			Sea trout perhabdovirus
		Sawgrhavirus	Island sawgrhavirus
			Minto sawgrhavirus
		Sigmavirus	Lousefly sigmavirus
			Drosophila melanogaster sigmavirus
		Sprivivirus	Carp sprivvirus
			Pike fry sprivivirus
		Sripuvirus	Chaco sripuvirus
			Chaco sripuvirus
		Sunrhavirus	Dillard sunrhavirus
			Garba sunrhavirus
		Tibrovirus	Bas-Conga tibrovirus
			Ekpoma 1 tibrovirus
		Tupavirus	Durham tupavirus
			Klamath tupavirus
		Vesiculovirus	Jurona vesiculovirus
			Yug Bogdanovac vesiculovirus
	Rhabdoviridae: Subfamily Betarhabdovirinae (6 genera and 57 species)	Iphanucleorhabdovirus	Peach alphanucleorhabdovirus
			Eggplant mottled dwarf alphanucleorhabdovirus
		Betanucleorhabdovirus	Sowthistle yellow vein betanucleorhabdhovirus
			Trefoil betanucleorhabdovirus
		Cytorhabdovirus	Wuhan 4 insect cytorhabdovirus
			Yerba mate cytorhabdovirus
		Dichorhavirus	Orchid fleck dichorhavirus
			Coffee ringspot dichorhavirus
		Gammanucleorhabdovirus	Maize fine streak gammanucleorhabdovirus
		Varicosavirus	Alopecurus varicosavirus
			Trifolium varicosavirus
	Rhabdoviridae: Subfamily Gammarhabdovirinae (8 genera and 20 species)	Alphacrustrhavirus	Wenling alphacrurhavirus
			Zhejiang alphacrustrhavirus
		Alphadrosrhavirus	Hubei alphadrosrhavirus
			Shayang alphadrosrhavirus
		Alphahymrhavirus	Cinereus alphahymrhavirus
			Hirtum alphahymrhavirus
		Betahymrhavirus	Austriaca betahymrhavirus
			Heterodontonyx betahymrhavirus
		Betanemrhavirus	Hubei betanemrhavirus
			Shayang betanemrhavirus
		Betapaprhavirus	Frugiperda betapaprhavirus
			Sylvina betapaprhavirus
		Betaricinrhavirus	Chimay betaricinrhavirus
			Scapularis betaricinrhavirus
		Novirhabdovirus	Piscine novirhabdovirus
			Salmonid novirhabdovirus
Double stranded RNA	Reoviridae: Subfamily Sedoreovirinae (6 genera and 35 species)	Cardoreovirus	Eriorcheir sinensis reovirus
		Mimoreovirus	Microminas pusilla reovirus
		Orbivirus	Bluetongue virus
			Chenuda virus
		Phytoreovirus	Rice dwarf virus
			Wound tumor virus
		Rotavirus	Rotavirus A
			Rotavirus B
		Seadornavirus	Banna virus
			Kadipiro virus

(*Continued*)

Table 1. (Continued).

Nucleic acid	Family	Genus	Examples of species
	Reoviridae: Subfamily Spinareovirinae (9 genera and 49 species)	Aquareovirus	Aquareovirus B
			Aquareovirus G
		Coltivirus	Colorado tick fever virus
			Eyach virus
		Cypovirus	Cypovirus 1
			Cypovirus 2
		Dinovernavirus	Aedes pdeudoscutellaris reovirus
		Fijivirus	Maize rough dwarf virus
			Pangola stunt virus
		Idnoreovirus	Idnoreovirus 1
			Idnoreovirus 2
		Mycoreovirus	Mycoreovirus 1
			Mycoreovirus 2
		Orthoreovirus	Avian orthoreovirus
			Mammalian orthoreovirus
		Oryzavirus	Echinochloa ragges stunt virus
			Rice ragged stunt virus
Double stranded DNA	Asfarviridae (1 genus and 1 species)	Asfarvirus	African Swine Fever Virus

SADC, conducted with data-driven approaches within a One Health framework, is therefore critical.

This scoping review aims to provide a historical overview of reported arboviral species in the SADC region. The outcomes of such research will contribute to raising regional awareness about arboviruses, highlight opportunities for improving surveillance and intersectoral collaborations, and guide emergency management strategies. Recognition of existing research and knowledge gaps is essential to enhance our understanding of arboviruses across all sectors, as zoonotic diseases know no borders and pose a constant threat.

Methods

A literary search was conducted to identify articles reporting on arboviral diseases found in ten of the sixteen SADC countries [66]. The following 20 SADC countries were included due to their sub-tropical and temperate climates, which create relatively similar climatic conditions for arbovirus vectors: Angola, Botswana, Eswatini, Lesotho, Malawi, Mozambique, Namibia, South Africa, Zambia, and Zimbabwe. The Democratic Republic of Congo and Tanzania were excluded due to their equatorial climates. Comoros, Madagascar, Mauritius, and Seychelles were excluded because they are islands, thus are geographically isolated and more prone to extreme weather conditions, such as hurricanes, when compared to conditions of the continental mainland.

Relevant studies were searched in health-related databases (PubMed, Google Scholar, and SABINET) using the keywords: arboviruses OR arboviral disease AND Southern Africa OR both the old (pre-independence) and the current name of a Southern African country, as shown in Table 2. The search was limited to studies published in English or at least having an English abstract from the 1st of January 1900 until NaN Invalid Date . Case reports and cross-sectional, retrospective, prospective, and observational studies were included in the assessment criteria. Titles and abstracts of the selected articles were examined to identify reported arboviral disease cases. Relevant papers were thereafter manually crosschecked to identify further references. The taxonomy of arboviruses was cross-referenced with the International Committee on Taxonomy of Viruses (ICTV) for accurate and updated classifications [68].

For consistency in reporting the geographic locations of historical data, here we use the country names that were given in the original publications, which do not necessarily match the current country names.

Table 2. Southern African countries names pre- and post-independence.

Country: Old Name *Pre-Independence*	Country: New Name *Post-Independence*	Year of Independence	Country: Old Name *Pre-Independence*	Country: New Name *Post-Independence*	Year of Independence
Reino de Angola	Angola	1975	Portuguese East Africa	Mozambique	1975
Bechuanaland Protectorate*	Botswana	1966	South-West Africa	Namibia	1990
Swaziland	Eswatini**	1968	Union of South Africa	South Africa	1961
Basutoland	Lesotho	1966	Northern Rhodesia	Zambia	1964
Nyasaland	Malawi	1966	(Southern) Rhodesia	Zimbabwe	1980

*'Protectorate' is dropped to make communication easy to read in the main text.
**The name Swaziland was changed to Eswatini during its 50th post-independence celebration (2018).

Results

From the literature reviewed, we identified 266 publications that described arboviruses in the SADC region. Publications most often referenced South Africa (143, 51.1%), followed by Mozambique (34, 12.1%), multiple countries of the SADC (36, 12.9%), Angola (28, 10.0%), Zimbabwe (15, 5.4%), Zambia (11, 3.9%), Botswana (7, 2.5%), Namibia (5, 1.8%), and Malawi (1, 0.4%) (Figure 2).

A total of 31 arboviral species were described within the SADC region. Ten species have only been reported in limited fashion in Southern Africa. The majority of the identified species were last intensively studied prior to the 1990s. After an approximate 20-year gap with minimal published arbovirus work within the SADC region, research gradually resumed from 2009 onwards (Figure 3). These recent studies primarily focused on reported cases of DENV, YFV, CHIKV, ZIKV, Rift Valley fever virus (RVFV), WNV, and SINV. In all serosurveys, cross-reactivity amongst arboviral groups was a confounding factor that complicated result interpretation, and necessary precautions were implemented to prevent misinterpretation and/or misdiagnosis.

Published vector surveillance studies within the SADC identified numerous arboviral vector species, mostly mosquitoes from the *Aedes* and *Culex* genera, and have contributed greatly to the general understanding of arboviral vector competence. Importantly, recent surveillance has also revealed the presence of vectors previously thought to be absent from the SADC, namely *Ae. albopictus* [69].

Family: bunyaviridae

Bunyamwera virus

Bunyamwera virus (BUNV) was first isolated from *Aedes* mosquitoes caught in the Semliki Forest of Uganda [70]. A second strain, isolated in 1955 from *Ae. circumluteolus* captured in Tongaland, Union of South Africa, was the first report of BUNV in Southern Africa [71]. *Aedes circumluteolus* appears to serve as the primary vector species for BUNV in the region [71,72]. In humans, BUNV infection tends to cause a febrile illness with mild symptoms (headache, rash, joint pains), though symptoms may be more pronounced in children and immunocompromised hosts [73]. The first described case of human BUNV infection in the SADC was that of a 13-year-old boy who developed fever, headache, and neck stiffness after assisting with mosquito captures in coastal Natal Province, Union of South Africa, in 1957 [72]. Serosurveys subsequently identified antibodies in residents of the Okavango Basin (Bechuanaland Protectorate) and Caprivi Strip (South-West Africa), in addition to the Natal Province [72,74]. No published data exist on more recent BUNV presence in the SADC.

Shuni virus

Shuni virus (SHUV) was first isolated in Nigeria in the 1960s from cattle, *Culicoides* biting midges, and humans [75–77]. Since then, several mosquito species belonging to the genera *Mansonia, Culex, Anopheles*, and *Aedes* have been identified as potential vectors of SHUV [78,79]. Due to their wide host and vector ranges, identifying epidemiological vectors of SHUV has proven difficult. In their study based in the North-Eastern portion of South Africa, Guarido et al. [78] found that *Ma. uniformis, Ae. mcintoshi* and *Ae. subargenteus* mosquitoes were positive for SHUV and may potentially act as bridge vectors for the virus. However, SHUV was easily detected in *Culicoides* midges from Mpumalanga and Limpopo provinces with a higher infection rate

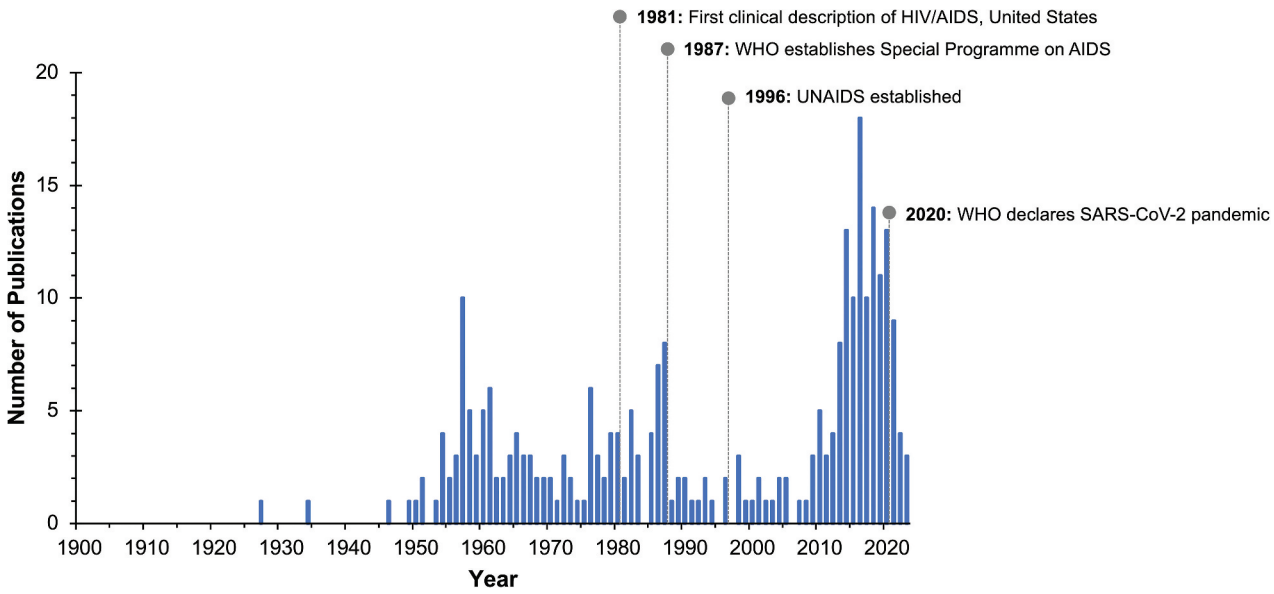

Figure 2. Publications related to arboviruses in 10 countries of the Southern African Development community (SADC), 1900–2023, and other key public health events. Abbreviations: AIDS, acquired Immunodeficiency syndrome; HIV, human Immunodeficiency virus; UNAIDS, joint united nations programme on HIV/AIDS; WHO, World Health Organisation.

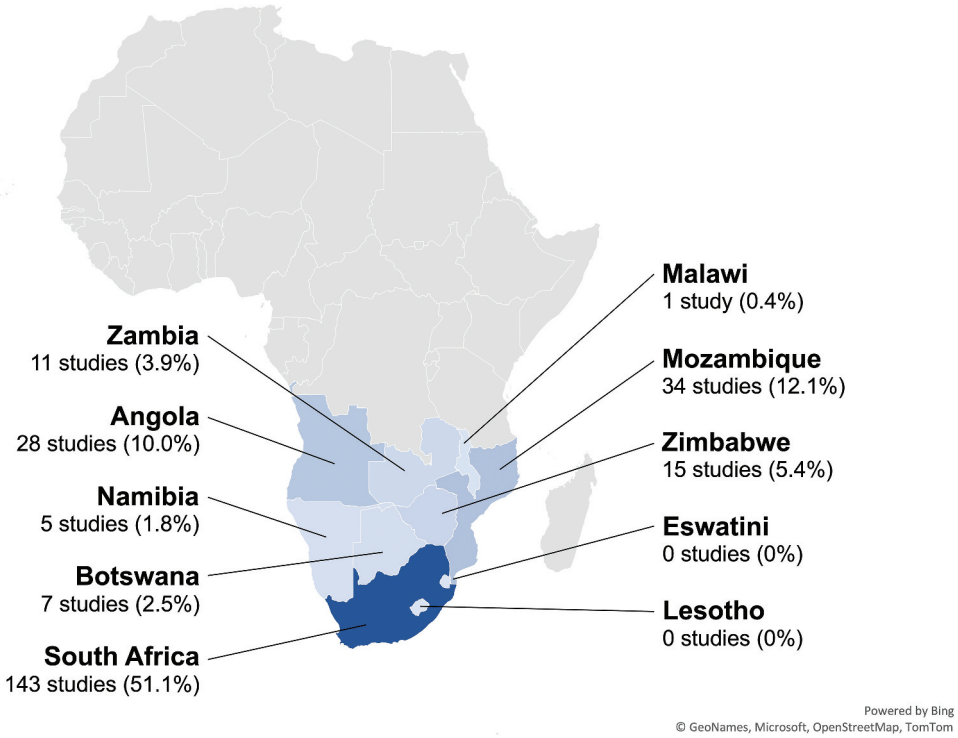

Figure 3. Publications related to arboviruses in 10 countries of the Southern African Development community (SADC), 1900–2023, by country. An additional 36 studies (12.9%) included data from multiple SADC countries.

when compared to other arboviruses [80]. This may allude to *Culicoides* midges being better SHUV transmitters than mosquitoes. In South Africa, SHUV appears to circulate annually as an equine virus which causes severe neurological disease and death in horses [79,81], and [82]. SHUV has also been isolated in South Africa from multiple species of nonequine domestic animals, birds, and wildlife, including springboks, giraffes, and crocodiles [83]. The virus's impact on humans is less well characterized. SHUV was recently detected in the cerebral spinal fluid of humans, most of them children, who were hospitalized in South Africa with neurologic symptoms [84]; however, there have been no other confirmed human cases of SHUV infection since the 1960s. Examining the potential risk to humans in contact with SHUV-infected animals, van Eeden *et al*. [82] found that 5 of 123 veterinarians had detectable SHUV antibodies [82]. No reports have been made on SHUV transmission out of this context. Overall, much work needs to be conducted to elucidate how SHUV transmission to humans occurs, together with its potential to cause clinical disease in humans.

Family: flaviviridae

Banzi virus

Banzi virus (BANV), originally named strain H336, was first isolated from the serum of a febrile child in Tongaland, Union of South Africa, in 1956 [85,86]. Working in South Africa and Rhodesia, McIntosh and colleagues isolated BANV from wild *Cx. rubinotus*, identifying that species as the primary vector [87–90]. *Culex quinquefasciatus*, by contrast, was an inefficient vector for BANV [91]. Serosurveys conducted in subjects across South Africa during the following decade identified communities with a high prevalence of BANV antibodies, interspersed with communities who had no evidence of BANV exposure [85]. Residents of Reino de Angola, the Caprivi Strip, and the Okavango Basin also had sera with protective antibodies to BANV [74,92]. BANV infection in sheep causes febrile illness and severe manifestations, including abortion and neonatal congenital abnormalities, in pregnant ewes [93]. Clinical manifestations of BANV infection in humans have not been well-described but appear to be mild and self-limiting [86,94]. Apart from recent vector surveillance that detected BANV in pools of *Cx. rubinotus* and *Cx. annulioris* in South Africa, no current data exist on the presence of BANV in Southern Africa [95].

Dengue virus

Dengue virus (DENV), which has four distinct serotypes (DENV-1, DENV-2, DENV-3 and DENV-4) and unclear geographic origins, is the leading arboviral cause of illness and death in humans worldwide [26,96]. Clinical features of dengue in humans range from asymptomatic infection to a nonspecific febrile illness that may include myalgias, vomiting, or rash, to potentially fatal shock or hemorrhage. DENV is primarily transmitted *by Ae. aegypti* mosquitoes in Africa as an urban vector. In

temperate regions, *Ae. albopictus* mosquitoes can act as a secondary vector [49].

Although dengue has been a recognized illness for centuries, its etiology and transmission remained uncertain until the virus was isolated in 1942 [97,98]. Dengue-like illness was first described on the African continent in 1779, when an outbreak affected residents of Cairo [99]. In 1926–1927, a large outbreak in Durban, Union of South Africa, marked the first documented cases of dengue in Southern Africa [100]. Serosurveys of South African residents done by Kokernot and colleagues later showed that this was a localized DENV-1 serotype outbreak [101]. Over the next 50 years, circulation of DENV in the SADC region appears to have been either unrecognized or entirely absent, with no published cases within this period. Interval serosurveys conducted among residents of Reino de Angola (1960, 1971–1972), the Okavango Basin (1959), and the Caprivi Strip (1959) demonstrated that no residents had antibodies to DENV-1 or DENV-2 [74,92,102].

In 1984, a DENV outbreak in urban northern Mozambique introduced the current era of DENV circulation in Southern Africa. This outbreak was the first described in Mozambique as well as the first documented occurrence of the DENV-3 serotype in Africa. An estimated 45% of residents were infected over four months, suggesting a highly susceptible population [103]. Low-level DENV transmission may have persisted in Mozambique after this outbreak. In 2012, a small serosurvey among residents of southern Mozambique found evidence of DENV exposure, though that region had not documented outbreaks [104]. Consecutive bouts of DENV-2 serotype outbreaks were reported in northern Mozambique in 2013 and 2014 [105,106]. Limited surveillance to monitor DENV circulation has occurred in Mozambique since late 2016 [107]).

Data on the current presence of DENV in other Southern African countries remains scant. In 2013, a DENV outbreak occurred in Angola [108]. Retrospective PCR-based serotyping identified all four DENV serotypes, suggesting multiple introduction events and emphasizing the possibility of unrecognized low-level transmission [108]. In Namibia, although no autochthonous cases of DENV have been documented, probable or confirmed dengue has been reported in returning travelers captured by global travel surveillance networks [109,110]. A serosurvey of prospective blood donors in Namibia using DENV antibodies, identified some individuals with positive results, which could also reflect cross-reactive exposure to other flaviviruses such as WNV known to circulate in the country [111].

Vector studies specific to DENV in the SADC region, while very limited in number and geographic range, indicate that regional variation in *Aedes* mosquitoes may restrict DENV transmission. The only study of vector competence for DENV in the SADC region, which was performed in eastern South Africa, identified populations of *Ae. aegypti* that were susceptible to and capable of transmitting DENV-1 and DENV-2 [112]. Moreover, the same study identified populations of *Ae. furcifer* and *Ae. streliziae* from South Africa that were susceptible to DENV-1 and 2. The transmission rates were 0 to 50% for *Ae. furcifer* and 0 to 29% for *Ae. streliziae* [112]. Vector sampling in Mozambique noted a predominance of *Ae. aegypti aegypti* in the north and the predominance of *Ae. aegypti formosus* in the south, a difference that may have contributed to the DENV outbreaks in the north [113].

Spondweni virus

Spondweni virus (SPOV) was first described in 1955 after isolation from *Ma. uniformis* mosquitoes in northern Zululand, Union of South Africa [114]. Subsequently, another survey in 1958 in the same area, identified the virus in the following mosquito species: *Ae. circumluteolus*, *Ma. africana*, *Ae. cumminsii* (subspecies *mediopunctatus*) and *Eretmapodites silvestris* [115]. Recently it has been demonstrated in laboratory-based experiments on mice that *Ae. aegypti* can efficiently transmit SPONV, whereas *Cx. quinquefasciatus* could not [116]. SPOV generally causes mild, nonspecific febrile illness in humans with symptoms similar to ZIKV, leading to potential misclassification [117]. Unlike ZIKV, the transmission cycle and geographical distribution of SPOV remains poorly understood [117]. Available data on SPOV in Southern Africa suggests that low level transmission occurs at irregular times and in variable regions. Serosurveys conducted in the mid-20th century among residents of Reino de Angola, the Okavango Basin, and the Caprivi Strip identified a low prevalence of exposure to SPOV [74,92]. Serosurveys in Natal found evidence of limited exposure among livestock and a high prevalence of exposure among some human residents, albeit variably distributed [85,118]. Mosquito surveillance conducted between 1956 and 1968 in Natal identified mosquitoes carrying SPOV [119] and found similar prevalence and distribution patterns to the results previously obtained by Smithburn *et al.* [85] and Kokernot *et al.* [118].

Usutu virus

Usutu virus (USUV) was first isolated in the Union of South Africa from a *Cx. neavei* mosquito in 1959 [120,121] and has been found to be transmitted primarily by *Culex* mosquitoes, with birds acting as amplifying hosts [122]. Phylogenetic analysis suggests that the virus likely emerged in South Africa from an ancestral strain present as early as the 16th century [123]. Cases of USUV infection causing fever, rash, and

neurologic symptoms in humans have been well described in northern sub-Saharan Africa and Europe [124]. However, in the SADC, no further data on the presence of USUV or of its vectors exist.

West Nile virus

West Nile virus (WNV) was first isolated in 1937 from a human with febrile illness in the West Nile district of Uganda [125]. The natural sylvatic hosts for WNV are birds, with over 900 species of birds identified as hosts [60,126,127]. Ornithophilic *Culex* species are the primary vector for WNV. *Culex univittatus* is the most frequent and efficient transmitter in the SADC, whilst *Cx. theileri* and *Cx. neavei* are also competent vectors [128–133]. *Aedes* species do not appear to be as capable vectors compared as *Culex* within the region [134].

Two major lineages of WNV have been described: isolates from lineage 1 generally circulate in Northern Africa, Europe, Asia, North and South America, and Australia, and isolates from lineage 2 generally circulate in Africa and Madagascar [135]. Until 2010, all sequenced WNV isolates from the SADC belonged to lineage 2, a distinction that was attributed to bird migration patterns [135–138]. However, isolates from lineage 1 have since also been identified among wildlife in South Africa [139,140].

WNV infection in humans is often asymptomatic and usually mild, with symptoms resembling a dengue-like illness that resolves after 3–6 days [141,142]. Fewer than 1% of infected humans develop severe illness with neuroinvasive disease (encephalitis, meningitis, or flaccid paralysis) [143]. In horses, neuroinvasive disease develops in as many as 20% of the cases and is often fatal [136,138,140]. Progression to severe disease in humans appears to be mediated in part by the neurovirulence of infecting WNV strains and in part by host immune factors [137,144,145]. Humans and other non-avian vertebrates are considered dead end hosts of WNV as their viremia levels are too low to support transmission back to mosquito vectors [130].

In Southern Africa, WNV was first isolated in the Tongaland in 1958; similar WNV strains were isolated from an ill human and from a captured bird that had presumed contact with the human patient [146]. Sporadic WNV infections, punctuated by epidemics that follow periods of heavy rainfall or unusually hot weather, have since been recorded throughout the region especially in South Africa [147,148]. Serosurveys conducted in the 1950s and 1960s identified WNV at low prevalence levels in indigenous residents of the Union of South Africa and the North-Western region of Reino de Angola, as well as in NHP and birds from South Africa [85,92,142]. In 1974, the largest known epidemic of human WNV infections in SADC occurred in the Cape Province, South Africa, after heavy rainfall. Though no deaths were reported 18,000 people developed acute febrile illness, and post-epidemic serosurveys identified WNV antibodies in 55% of residents [138,149]. Additional serosurveys in the 1980s identified antibodies to WNV among one-third of residents of northern South-West Africa, just to the north of the region affected by the 1974 epidemic [111,150,151]. More recent serosurveys among humans have been conducted only in Zambia. A cross-sectional study provided evidence of WNV infections in humans of all ages in the North-Western and Western provinces of Zambia, indicative of WNV prevalence and persistence within the country [152,153].

WNV circulation in mosquito vectors and avian hosts in Southern Africa tends to overlap with that of another arbovirus, SINV [89,94,131,147,154–156]. Similarly, transmission of WNV to humans has been identified during outbreaks of human SINV infection, though at lower rates than SINV, and their indistinguishable symptoms make the two viruses challenging to diagnose by symptoms alone [141,147,149,157].

In Southern Africa, documented severe WNV infections in humans are few, but infections may go unrecognized due to limited awareness and scarce diagnostics [158]. Severe and fatal WNV infections have also occurred in non-human vertebrates. A dog in Botswana developed fatal encephalitis that was initially attributed to infection with Wesselsbron virus (WESV), later confirmed to be WNV [135,159]. Later serosurveys of dogs in South Africa demonstrated that dogs, while susceptible to WNV infection, developed only low levels of viremia and thus were dead end hosts [160]. Currently, WNV is a cause for concern for horse owners in South Africa, causing annual equine outbreaks with a 34.2% fatality rate [139,161]. Laboratory transmission of WNV to humans has occurred in conjunction with equine cases in South Africa [145,162]. WNV was also recently isolated following fatal infections in non-equine wildlife in South Africa and farmed crocodiles from the Southern Province of Zambia [139,163]. Recent published entomological surveillance data for WNV in the SADC are limited to Namibia, South Africa, and Zambia, where WNV has been identified in *Culex* mosquitoes [95,164,165]. Overall, available data suggest that WNV circulates throughout the SADC, but many knowledge gaps remain.

Yellow fever virus

Yellow Fever virus (YFV) originated in Africa and spread globally through the slave trade in 1650 or even earlier [5]. Due to the extensive research done on YFV globally, the clinical symptoms of infection in humans have been well described. Most humans infected with YFV experience no or only mild symptoms, but those who develop severe disease have a mortality rate as high as

85% in some regions [166]. YFV has a 3- to 6-day incubation period followed by a brief acute febrile phase, which may be followed by a second morbid phase marked by jaundice, hematemesis, hemorrhage and renal injury [167]. The use of YFV vaccines has remarkably reduced the incidence rate in endemic areas. However, with waning immunity and barriers to widespread vaccination, communities in sub-Saharan Africa still experience sporadic YFV outbreaks [167–169].

In the SADC, much work aiming to understand the regional sylvatic transmission cycle of YFV has been conducted in the North-Western Province of South Africa and North-Western Province of Zambia [170]J. H [171; 172]. Key NHP species that have been identified in YFV sylvatic transmission and have habitat in Southern Africa are the green and vervet monkeys (*Cercopithecus* spp.), Cape baboon (*Papio ursinis*), and possibly bush babies (*Galago* spp.) [5,52,173]. *Aedes aegypti* and in some cases *Ae. africanus* are primarily responsible for YFV transmission to humans in Southern Africa [5,172,174]. However, earlier reports showed that *Ae. simpsoni* plays a secondary role in YFV transmission [175] in the SADC region. Other species with experimentally demonstrated, but not proven, roles in natural transmission are *Ae. vittatus*, *Ae. metallicus*, *Ae. taylori* [175]. Importantly, *Ae. luteocephalus*, a competent vector for YFV [176], has been found to occur in Mozambique [177], though its vectorial role was not assessed.

YFV research in the SADC began as early as the 1930s. Serosurveys conducted among residents of Angola in 1933 found antibodies at very low prevalence, leading the authors to declare the region 'practically negative' [178]. In Northern Rhodesia, following a suspected YFV case, serosurveys conducted from 1937–1943 detected YFV antibodies in residents of the Western Province [170,179]. This led to further YFV investigations in other Southern African countries. Serosurveys conducted in 1945, 1949, and 1959 determined minimal incidence rates of YFV in South-West Africa and Bechuanaland whilst the Union of South Africa experienced periodic YFV outbreaks between 1951–1953 [74;180,181]. In 1971, a YFV outbreak developed in Luanda, Angola, and was halted with a combination of mass vaccination and mosquito control [182,183].

Although Southern Africa is now generally regarded as a low-risk region for YFV (currently, only Angola is considered endemic and mandates YFV vaccination), sporadic YFV outbreaks still occur, and additional cases likely go undetected [168,184,185]. Recent serosurveys have confirmed that YFV still circulates in the North-Western and Western Provinces of Zambia [170]. Additionally, a new outbreak in Luanda in 2015–2016 developed into the largest YFV outbreak reported in the last 30 years, with approximately 4,306 suspected cases and as many as 393 deaths [167,169,186]. Travel from Angola during the outbreak led to imported YFV cases in countries outside the SADC including China, causing the first reported cases of YFV in Asia [167,187,188]. These statistics underscore the vulnerability and limited preparedness in the SADC when it comes to YFV response.

Zika virus

Zika virus (ZIKV) was first isolated in 1947 from a rhesus macaque used for YFV monitoring in Uganda [189]. For many years, ZIKV outbreaks were confined to Africa and Asia until the beginning of this century, when the virus emerged in Micronesia, French Polynesia, and then Central and South America [190–192]. Concerning ZIKV vectors, urban vector *Ae. aegypti* is associated with ZIKV transmission in Southern Africa, while sylvatic vectors such as *Ae. furcifer, Ae. luteocephalus, Ae. africanus and Ae. taylori* are vectors in most sub-Saharan African countries except the SADC region where some species are present but have not yet been implicated with ZIKV transmission [193]. While *Aedes* are the primary arthropod vectors for ZIKV, non-biological modes of ZIKV transmission among humans (e.g. sexual, perinatal, through organ transplantation) have also been described [194]. Given the numerous possible modes of ZIKV transmission, factors driving the global expansion of ZIKV are complex [194–196]. ZIKV infection in humans typically causes a mild febrile illness with nonspecific, self-limiting symptoms (rash, headache, arthralgia, myalgia) [197,198]. Severe neurologic complications also occur: a small proportion of adults experience Guillain-Barré syndrome, and maternal-fetal transmission has been associated with the development of microcephaly and other congenital abnormalities [194,197,198].

In Southern Africa, serosurveys conducted prior to 1990 identified ZIKV in the Reino de Angola, Northern Rhodesia, and Portuguese East Africa [194]. These data were used to map regions of ZIKV circulation [194,199]. However, with missing or omitted information, these maps likely understate ZIKV's presence in some regions [5,194]. For instance, Kokernot *et al*. [200] documented a 4.0% seroprevalence of ZIKV in Portuguese East Africa, contradicting historical maps which suggest that ZIKV has never circulated in Mozambique [5,199–202]. From 2009–2015, Chelene et al. [203] conducted a nationwide retrospective seroprevalence study in children and found a 4.9% ZIKV antibody positivity, suggesting recent ZIKV transmission in Mozambique.

There is limited recent data on ZIKV in sylvatic hosts in the SADC. A study by Buechler et al. [204] examined the prevalence of ZIKV in NHP in Zambia, South Africa, and elsewhere and found no evidence of active ZIKV infection or prior ZIKV exposure among NHP from the SADC region [204]. While these findings suggest no current circulation of ZIKV in the natural hosts, this does not exclude the possibility of ZIKV circulation within the SADC region.

Imported ZIKV cases also represent a threat to the SADC region. Since the 2016 ZIKV outbreak in Brazil, its spread has become a major concern particularly in Angola, where imported cases have been reported [192]. Angola was the first Southern African country to report circulation of the Asian lineage of ZIKV and ZIKV-associated microcephaly [205,206]. However, the first African country to report the circulation of the Asian lineage of ZIKV was Cabo Verde [207]. Hill *et al*. [205] conducted a phylogenetic analysis and found that ZIKV may have been circulating in Angola 28 months prior to the reported cases, suggesting that the outbreak was larger than initially reported. In response to these imported cases, scaled-up surveillance and monitoring efforts in Angola have involved tracking passengers' in-flight data to assess the likelihood of ZIKV introduction and transmission to Angola and other African countries [205,208].

Family: nairoviridae

Crimean-Congo haemorrhagic fever virus

Crimean-Congo Haemorrhagic Fever virus (CCHFV) is a severe tick-borne zoonotic disease with a high case fatality rate in humans. The first case of this hemorrhagic fever reported in Southern Africa was that of a 13-year-old boy from the Transvaal Province, South Africa, who was diagnosed in 1981 with CCHFV, and shortly thereafter died [209]. This was the first reported case of fatal human CCHFV infection in the region. Transmission occurred via a tick belonging to the *Hyalomma marginatum* species complex (of which *H. rufipes* is one member that circulates in Southern Africa), and the boy progressed rapidly from symptom onset to hemorrhagic state and death within nine days [209]. In response, a thorough ecological and serosurvey of CCHFV was conducted by Swanepoel et al. [210] in five provinces of South Africa. Their investigation isolated CCHFV from several tick species (*H. rufipes, H. truncatum* and *Rhipicephalus evertsi*) and detected CCHFV antibodies in both humans and livestock, indicating that CCHFV was prevalent in the country. The numerous veterinary cases suggest that livestock act as amplifying hosts, enabling sporadic outbreaks and direct transmission to humans [211]. In their study of infected ticks, Swanepoel et al. [210] also confirmed that *H. rufipes* was capable of transovarial CCHFV transmission.

The Crimean-Congo Haemorrhagic Fever virus generally circulates in nature in unnoticed enzootic tick – vertebrate–tick cycles, and asymptomatic CCHFV infection has been reported in numerous vertebrate species and appears to be pervasive in both wild and domestic animals [212]. Surveys conducted in South Africa showed evidence of the asymptomatic circulation of CCHFV among cattle [213,214] with a seroprevalence between 26.5 and 28%. Moreover, antibodies were found to be highly prevalent in large mammals such as giraffe, rhinoceros, eland, buffalo, kudu and zebra [215]. In small mammals, antibodies were found in the sera of hares, rodents, and wild carnivores [215]. Antibodies were also detected in domestic dogs from South Africa and Zimbabwe [215]. To conclude, some evidence has also been provided on the circulation of CCHFV among wild birds (guinea fowl) in South Africa [216].

Reports of CCHFV coinfections with other pathogens have also been documented, complicating understanding of its interactions. The first coinfection to be documented was that of CCHFV and MARV, with 486 cases reported in Rhodesia [217]. The second was that of CCHFV and malaria detected by Gear [218] in a traveler who had returned to South Africa from Rhodesia [218]. Gear [218] notes that hemorrhagic fever diagnosis becomes even more complex once the patient is coinfected with another pathogen.

Family: peribunyaviridae

Germiston virus

Germiston virus (GERV), first isolated in 1958 from a mixed pool of *Culex* mosquitoes in Germiston, Union of South Africa [219], appears to be prevalent but of unclear significance in the SADC region. Not all *Culex* species are capable of efficient GERV transmission: *Cx. rubinotus* is a competent vector for GERV, but *Cx. quinquefasciatus* transmits GERV only at a very low rate [87,90,91,220]. Wild rodents have been identified as hosts [88,94]. Serosurveys have detected antibodies to GERV in as many as one-third of residents in the Okavango Basin, three regions of Reino de Angola, and coastal Natal Province in South Africa [74,92]; B. L [221]. McIntosh [94] found that humans with GERV infection experienced brief, nonspecific symptoms of febrile illness. These data suggest that GERV infections are mild but are not enough to draw conclusive remarks about its epidemiology.

Pongola virus

Pongola virus (PONV) was first isolated from *Ae. circumluteolus* in Tongaland, Union of South Africa [222]. Studies by Jupp and McIntosh [223], also in Tongaland, further identified *Ae. circumluteolus* as a potential vector but only in an incidental capacity [223]. Serological surveys conducted in 1959 and 1960 indicated high prevalence (9–26%) of PONV infection in humans in Reino de Angola, Northern Natal, the Caprivi Strip, Bechuanaland and Mozambique, primarily in coastal and low-lying areas [74,85,92,200,224]. However, it is worth noting that PONV is highly cross-reactive with Bwamba virus (BWAV) in many serological tests [224], making it difficult to distinguish the distribution and relative

abundance of these two viruses. Scant information on PONV exists in Southern African states.

Simbu virus

Simbu virus (SIMV) was first isolated from *Ae. circumluteolus* mosquitoes captured at Lake Simbu, Union of South Africa, in 1955 [225]. SIMV has since been categorized in the Simbu serogroup as one of 25 phylogenetically related arboviruses distributed worldwide, transmitted by various mosquito species and *Culicoides* midges [226]. Whilst other viruses in the Simbu serogroup have been identified as important causes of human and veterinary infection outside Southern Africa [226], the significance of SIMV remains unclear. Laboratory infection with SIMV caused viral encephalitis in mice but no apparent signs or symptoms of illness in rabbits, guinea pigs, or vervet monkeys [225]. Clinical features of SIMV infection in humans are unknown. Early serosurveys found positive SIMV antibodies in a few humans in the Okavango Delta and the northern Natal region of South Africa [74,85]. Apart from additional isolation of SIMV from *Ae. circumluteolus* captured in the Ndumu Game Reserve, South Africa, in 1964 [119], no other reports on SIMV in SADC exist.

Witwatersrand virus

Witwatersrand virus (WITV) was isolated in 1958 from wild *Cx. rubinotus* during an outbreak of febrile illness among the residents of Germiston, Union of South Africa [227]. After initial investigations established that *Cx. rubinotus* is a competent vector and wild rodents are likely hosts. Since, WITV has not been further studied in Southern Africa [87,88,90,91,220].

Family: phenuiviridae

Rift valley fever virus

Rift Valley fever virus (RVFV) causes disease primarily in domestic ruminants, leading to febrile illness and abortion, and was first reported as the cause of sheep deaths in Kenya in 1931 [228,229]. RVFV has since been described in livestock, humans, and arthropod vectors throughout Africa and the Middle East [230]. The first report of RVFV in the SADC occurred during a livestock epidemic in 1951 in the Union of South Africa [231]. Subsequent studies done in the same country identified multiple species from the *Aedes* and *Culex* genera as potential RVFV vectors, with *Cx. theileri* and *Cx. zombaensis* likely responsible for RVFV outbreaks in the region [71,232–237]. Moreover, it has been demonstrated that *Ae. juppi* can easily be infected with RVFV but has no transmission capabilities, whilst *Ae. unidentatus, Ae. dentatus* and *Cx. poicilipes* were identified as potential epizootic vectors in South Africa [238]. More recently, Linthicum et al. [239] curated an extensive and comprehensive list of RVFV vectors found globally including those circulating in Southern Africa. This list shows *Aedes and Culex* as dominant and effective transmitters of RVFV. The recent confirmation of *Ae. durbanensis* as a vector of RVFV in the endemic region of Kwazulu-Natal, South Africa, was a result of a thorough genomic and phylogenetic analysis done by van den Bergh et al. [240]. Phylogenetic analysis also revealed that the identified virus is closely related to two isolates from the earliest outbreaks, which occurred in central South Africa more than 60 years ago, indicating long-term endemicity in the region (van de Bergh et al., 2022).

RVFV infection in livestock can be asymptomatic, but its role as a cause of abortion in pregnant livestock and fatal infection in newborn livestock gives it great economic importance [230,241–243]. Numerous RVFV outbreaks have been documented in livestock across Southern Africa, including Mozambique, Northern Rhodesia, Rhodesia, and especially South Africa (where continuous surveillance occurs) [229,230,232,237,244,245]. Serosurveys conducted in livestock in Mozambique, South Africa, and Zambia have also shown evidence of RVFV circulation in herds without signs of clinical disease [244,246,247]. For example, in Mozambique, serosurveys in cattle identified RVFV seroprevalence as high as 62.2% in a province that had no documented RVFV outbreaks in the previous four decades [246].

In humans, RVFV infection may be asymptomatic, uncomplicated (with acute influenza-like fever, headaches, and muscle pains), or complicated (with gastrointestinal symptoms, hepatic or renal failure, encephalitis, visual symptoms, and/or hemorrhagic manifestations) [248]. Human RVFV infection was first recognized in farmers and veterinarians who directly handled the tissues of infected animals [228]. The first documented human infections in Southern Africa occurred in 1951, when farmers and veterinarians in the Union of South Africa developed febrile illness after performing an autopsy on an infected bull; all recovered, except for a few individuals who had residual visual defects [232,249,250]. Similar self-limited infections occurred in Rhodesia in 1957 among farmers and a laboratory worker [251,252]. Descriptions of severe and fatal human RVFV infections, including hemorrhagic fevers, began to emerge in Southern Africa in the 1970s [253,254]. In 1975, an RVFV outbreak in South Africa led to seven human deaths from gastrointestinal hemorrhage [218]. Serosurveys among asymptomatic humans in the Caprivi Strip and Okavango Basin (1959), Angola (1971–1972), South Africa (1981–1982), northern South-West Africa (1983), and Botswana (1984–1986) also demonstrated evidence of RVFV exposure separate from documented livestock outbreaks, suggesting vector-borne transmission [74,102,150]; B. L [221; 255]. Niklasson et al. [256] examined the effects of RVFV infection in pregnant women

in Mozambique; the authors identified RVFV antibodies in 2% and found that women with evidence of past RVFV exposure experienced no significant difference in pregnancy outcomes. However, studies elsewhere in Africa have linked acute RVFV infection to an increased risk for miscarriage in pregnant women [257].

More recent studies have demonstrated ongoing low-level circulation of RVFV among humans across Southern Africa. Serosurveys of humans in northern Botswana (2013–2014) and KwaZulu-Natal, South Africa (2018–2019), found overall RVFV seroprevalences of 5% and 2.8%, respectively [258,259]. A cross-sectional study of febrile patients in Mozambique identified RVFV as the causative agent in 5% of them [260].

Overall, these studies indicate that RVFV circulates in Southern Africa in a heterogeneous fashion, characterized by low-level maintenance transmission punctuated by periodic and sporadic outbreaks. In the last decade, genome sequencing and spatiotemporal modeling techniques have begun to better describe the factors influencing RVFV transmission in the SADC, but much is still unclear. Sequencing of RVFV from infected livestock in Mozambique isolated RVFV belonging to lineage C, which is also a known cause of livestock deaths along the eastern coastline of Africa, Madagascar, and some Middle Eastern countries [245]. Recently sequenced RVFV isolates in South Africa, however, belonged to multiple other lineages, indicating high genetic diversity within a relatively small geographic area [261]. In Botswana, Pachka et al. [262] developed a mechanistic model to explore how environmental factors influence the dynamics of *Cx. pipiens* as a potential RVFV vector. In South Africa, other models have explored the impact of temperature, rainfall, soil conditions, and other local environmental characteristics on the likelihood of RVFV transmission [263–265]. Such models can aid in vector control. Vaccination provides another approach for outbreak prevention; vaccines against RVFV have been developed for livestock and humans, but problems with vaccine safety, efficacy, and delivery have hindered widespread use [236,266].

Lunyo virus

Lung virus (LUNV), an atypical strain of RVFV, was first isolated in 1955 from a pool of collected *Aedes* mosquitoes in Uganda [267]. Recent genomic and phylogenetic analysis of LUNV by Lumley et al. [268] confirm LUNV is indeed a strain of RVFV.

Family: togaviridae

Chikungunya virus

Chikungunya virus (CHIKV) was first isolated in Tanganyika (mainland part of present-day Tanzania) in 1952 [269,270]. However, evolutionary studies conducted by Weaver and Forrester [271] suggest that CHIKV may have existed for at least a century prior to its discovery. In the SADC, baboons (*Papio ursinus*) and vervet monkeys (*Cercopithecus aethiops*) have been identified as sylvatic hosts [57,272]. Sylvatic vectors are important for CHIKV transmission in Africa. In particular, *Ae. furcifer*, and possibly also *Ae. cordellieri*, have been identified in South Africa [272,273] as primary vectors of the virus.

Ae. aegypti and *Ae. albopictus* are urban vectors. Domestic animals and humans are both dead-end hosts [274]. Clinical symptoms of CHIKV in humans are often self-limiting and include fever, rash, and severe arthralgia, with symptoms typically lasting up to 7 days; however, as many as 40% of infected individuals develop chronic, recurrent arthralgia, and more severe manifestations have been reported [274,275].

Regional serosurveys in Reino de Angola, the Okavango Basin, the Caprivi Strip, and the Union of South Africa by Smithburn et al. [85], Kokernot, Casaca, et al. [92] and Kokernot, Szlamp, et al. [74] in the 1950s and 1960s found CHIKV antibodies in adults and some children, indicating CHIKV circulation within the surveyed regions. Children in coastal Natal Province, Union of South Africa, lacked protective sera, highlighting the possibility of a future CHIKV epidemic [85]. During the same period in Rhodesia, several serosurveys were conducted in response to an outbreak in the south of the country [58,276–278]. Swanepoel and Cruickshank [237] also found high CHIKV epidemiological patterns that were seasonal and specific to different areas in Rhodesia. These results suggest that CHIKV was previously an endemic disease in Rhodesia. In Luanda, Angola, in 1970–1971, CHIKV co-circulated with YFV and produced a dual arboviral outbreak [183,279]. In Malawi in the 1980s, a serosurvey conducted among children identified CHIKV antibodies in approximately half of children, suggesting active CHIKV circulation [280].

More recently, a report of a severe case of human CHIKV infection in Pemba, Mozambique, describes well the current knowledge of CHIKV in Southern Africa [281]. In their report, the authors clearly show the barriers that regional healthcare systems face in accurately diagnosing and treating patients with CHIKV and other arboviral infections. Much effort has been placed on understanding the current prevalence and spatiotemporal distribution of CHIKV in Mozambique [282–288]. In Zambia, a recent cross-sectional serosurvey also identified CHIKV antibodies in 36.9% of residents of a wetland area [289]. Generally, scant information is available on recent CHIKV circulation in the rest of the SADC. CHIKV infection has been diagnosed in travelers returning from Angola, yet no recent data from within Angola have been published [290,291]. The current range of CHIKV circulation in Southern Africa needs further investigation.

Middelburg virus

Middelburg virus (MIDV) was first isolated from *Aedes* species in the Eastern Cape Province, Union of South Africa, in 1957 [292], with a single human case later reported in a resident of Natal [247]. Vector surveillance conducted from 1960–1968 in South Africa by McIntosh [233], confirmed MIDV in *Aedes* species. More recently, *Ae. callabus and Ma. africana* have been implicated as vectors that are primarily found to be positive for MIDV [293]. Early serosurveys among humans did not identify any neutralizing antibodies to MIDV in residents of Reino de Angola, the Okavango Basin, or the Caprivi Strip [74,92]. Any link between MIDV infection and disease in either humans or other animals was initially unclear. However, isolation of MIDV from horses in South Africa and Zimbabwe has since confirmed MIDV as a cause of severe and fatal equine disease [294–296]. In addition, MIDV has been isolated in South Africa from a variety of non-equine wildlife and birds who died of otherwise-unexplained neurologic or febrile disease [297]. Emerging case reports from South Africa also suggest that MIDV infection may be the cause of neurologic manifestations in humans [298]. Despite this knowledge, the host ranges for MIDV remain unknown. Whether MIDV transmission currently occurs in other SADC nations is undetermined.

Semliki forest virus

Semliki Forest virus (SFV) was first isolated from the *Ae. abnormalis* group of mosquitoes in 1942 in Uganda [299]. It was subsequently isolated in coastal central Portuguese East Africa from *Ae. argenteopunctatus* in 1959 by McIntosh et al. [300]. In addition, in a lab model it has been shown that *Ae. aegypti* is capable of acting as an efficient vector of SFV, both via biological and mechanical transmission [301]. Serosurveys have since detected SFV throughout the SADC region. Evidence points to SFV virus being most prevalent near large bodies of surface water in temperate or tropical areas, such as in northern Reino de Angola, the Caprivi Strip, and in the provinces of Natal, Cape, and Transvaal, Union of South Africa [74,92,101]. For example, serosurveys conducted in coastal north-western Reino de Angola identified neutralizing antibodies to SFV in 33% of adults [92]. Limited information is available regarding the clinical symptoms of SFV infection in humans. The first reported human outbreak of SFV infection, which occurred in the Central African Republic in 1987, described fever, severe headache, myalgia, and arthralgia that lasted 2–4 days with a slow recovery [302]. To date, no human SFV infections have been documented in the SADC.

Sindbis virus

Sindbis virus (SINV) was first isolated in 1952 from *Culex* and *Culiseta* mosquitoes in Egypt [126]. Birds act as the natural host, and serosurveys and vector surveillance have identified a broad distribution globally [303]. In the SADC, SINV was first clinically diagnosed and isolated in South Africa in 1963 [304]. The primary vectors of SINV in Southern Africa are *Cx. univattatus* and *Cx. neavei*, and both are capable of transmitting SINV to humans [89,131,134,155,305]. In a recent survey (2014–2017) in several provinces of South Africa [306], the following mosquito species have been found infected with SINV: *Cx. univittatus*, *Cx. pipens* s.l., *Cx. zombaensis*, *Cx. theileri*, *Cx. annulioris*, *Ae. tarsalis/aerarius*, *Ae. durbanensis*, *Ae. mcintoshi*, *Ma. uniformis*. Sindbis virus was found to be detected slightly more frequently in peri-urban sites than in conservation and rural areas [306].

As previously noted, symptoms of SINV infection in humans are similar to those of WNV infection and include fever, arthralgia, rash, and malaise [94]. Many studies in the SADC have shown direct or indirect evidence of SINV infection in humans, including the Reino de Angola, the Okavango Basin, the Caprivi Strip, Rhodesia, Zambia, and particularly South Africa, where SINV outbreaks occurred in 1974 and 1984 [74,85,92,237,303,307,308]. Recent passive surveillance in South Africa has continued to identify human SINV infections, and no fatalities have been reported to date [303]. Latterly, SINV (2019–2020) cases were detected from hospitalized patient samples by PCR and serological methods in two northern provinces of South Africa [309].

Wesselsbron virus

Wesselsbron virus (WESV) was first isolated from an aborted lamb at Wesselsbron Farm in the Orange Free State Province, in the Union of South Africa [310,311]. This discovery spurred an onset of investigative studies across Southern Africa to determine the characteristics of WESV. *Aedes circumluteolus* was identified as a natural vector [312,313], while *Ae. caballus* was a competent vector under laboratory conditions [219]. In sheep and goats, WESV infection causes febrile illness and high rates of abortion in pregnant animals [314–316]. Cattle experience mild or asymptomatic WESV infection, without abortion [317]. WESV infection in humans, which can occur via vector or via direct transmission when handling tissues from infected livestock, causes a nonspecific, self-limited febrile illness [237,318]. Serosurveys conducted in the 1950s and 1960s in Bechuanaland, the Caprivi Strip, Northern Rhodesia, Nyasaland, Reino de Angola, Rhodesia, Mozambique, and the Union of South Africa showed presence of WESV antibodies in humans, cattle, and sheep, suggesting that WESV was endemic in Southern Africa at the time [74,92,218,237,316]. Currently, there is no active surveillance for WESV in the SADC, but occasional reports appear of WESV outbreaks among humans and livestock [315,319,320].

Other arbovirus species identified in Southern Africa

Vector and host surveys conducted in the SADC from the 1950s through the 1960s identified multiple other arboviruses that have not been well-studied in the region since. The current impact of these arboviruses on humans and animals in Southern Africa is thus unknown.

Kokernot and colleagues found sera positive for antibodies to BWAV in humans, donkeys, and a bird in Mozambique and the Union of South Africa [101,200,321]. In the low-lying region of Simbu Pan, Natal Province, Union of South Africa, BWAV was the predominant arbovirus identified among humans; 80% of those tested had neutralizing antibodies to BWAV [101]. Bwamba virus circulation appears to have been localized, in fact humans, NHP, domestic animals, and birds in other areas of the Union of South Africa had no or very infrequent antibody response [101]. Kokernot and colleagues also tested human and animal sera from the Union of South Africa for antibodies to Ntaya virus (NTAV) and found no positive sera [101]. In Reino de Angola, however, they identified a low prevalence of NTAV exposure in humans (corroborated by a later serosurvey conducted by Filipe and colleagues), as well as sera positive for antibodies to Lumbo (LUMV), Mayaro (MAYV), and Oriboca (ORIV) viruses [92,102]. Serosurveys in the neighboring Caprivi Strip and Okavango Basin also found a low prevalence of ORIV exposure in humans but did not evaluate for exposure to BWAV, NTAV, LUMV or MAYV [74].

In an extensive field survey in the Ndumu Game Reserve (Union of South Africa) from 1956–1968, McIntosh and colleagues conducted annual mosquito catches, isolated viruses and described their viral vectors, and discussed the epidemiological implications of their results [119,322]. Their work detected Ingwavuma (INGV), Lebombo (LEBV), Shokwe (SHOV), and Ndumu (NDUV) viruses for the first time in Southern Africa [119,322]. They also identified Mossuril virus (MOSV), which had previously been isolated once from a pool of *Cx. sitiens* in Mossuril, Portuguese East Africa, in 1959 [119,323]. Field collections showed that *Ae. circumluteolus* was the dominant species but was only an incidental vector that was unable to maintain viral survivability [119].

Discussion

This review provides evidence of the significant presence, both past and current, of arboviruses with medical and veterinary importance within the SADC. Environmental conditions in Southern Africa support the circulation of arboviruses and their vectors. Ecological and anthropological factors have increased the proximity between sylvatic reservoirs and humans, increasing the likelihood of spillover events and outbreaks in ecologically naïve and niche areas. Recent outbreaks of CHIKV, DENV, YFV, and RVFV, accompanied by multisectoral challenges in recognizing and controlling arboviral transmission, illustrate Southern Africa's current vulnerability [187,261,281].

Arboviral research flourished in Southern Africa in the 1950s, led by researchers at the Rockefeller Foundation Virus Program in Johannesburg, Union of South Africa, and continued intensively until the late 1980s [324]. However, a notable 20-year lull occurred before research began to resume around 2010 (Figure 3). A myriad of reasons likely contributed to the decline of arboviral research in the region. The emergence of the HIV/AIDS pandemic in the 1980s diverted scientific attention and resources [325]. Lack of recognition and underreporting of arbovirus infections, due in part to the self-limited and benign nature of most cases, may have added to a lack of research interest [281]. Concurrently, an intensive public health focus on malaria and its vectors, which has resulted in admirable progress toward malaria elimination, prioritized research in malaria over work in arboviruses [23,67]. The geopolitical turbulence and conflicts experienced when Southern African countries fought for independence also could have hindered research efforts, with limited resources reallocated to other critical needs. Indeed, social conflict is a long-recognized driver of infectious diseases, and this link is particularly true in Africa [326]. Finally, funding for arboviral research in the region has not recovered since the closure of the Rockefeller Foundation Virus Program [324]. A search of the National Institutes of Health (NIH) Research Portfolio Online Reporting Tools Expenditures and Results (RePORTER) datasets, for example, identifies no NIH-funded studies on arboviruses in the 10 SADC countries examined here from 1985 onward [327].

While an effective response to arboviruses should prioritize risk monitoring, the current infrastructure for arboviral surveillance in the SADC is inadequate and largely relies on passive rather than active measures [10,27,67]. Southern Africa is not unique in its struggle to develop a sustained arboviral surveillance network; in high-income countries like the United States, capacity for arboviral surveillance also fluctuates in reaction to outbreaks [328].

Finally, there is growing concern that climate change will alter the distribution and burden of vector-borne diseases, potentially reversing the gains of vector control programs (as achieved for malaria in Africa) and increasing the threat of emerging diseases. In this context, *Ae. aegypti* has shown ecophysiological plasticity in adapting to varying ecological conditions and has potential to expand the reach of arboviruses [7,329]. Prompt intersectoral action from governments and research institutions is needed to address the effects of climate change.

We strove here to compile a comprehensive assessment of arboviral research conducted in Southern Africa to date, but we may have overlooked some work. Incomplete cataloging terms, especially for older studies, make some arboviral research challenging to locate. Studies published in languages other than English were excluded. We also included only published data, not any findings that may have only been reported to local health systems or surveillance teams. This is a limitation of our assessment but also emphasizes the importance of developing intersectoral and inter-country information networks to readily share data on arboviral transmission and spread.

Future directions

As most Southern African states are low-middle income countries, determining transmission patterns and evaluating epidemiological data to conduct informative diagnostic investigations have been severely hampered by poor laboratory capacity [27,67,260]. In this context, new and improved diagnostics, including low-cost and point-of-care tests, will also be essential to allow rapid identification of arboviral infections [330]. Undoubtedly, these advances will shift the limits of arboviral surveillance and detection. However, vector control is currently the most effective preventative tool available to reduce the risks of arboviral transmission and associated pathological effects. With insecticide resistance threatening current control methods, biotechnological advances in modeling arbovirus-vector interactions will be essential to provide innovative and sustainable vector control solutions [331,332]. For example, we would like to highlight how *Wolbachia*, a bacterium, has been found to reduce transmission of several arboviruses including ZIKV, CHIKV and DENV [333,334]. However, unlike in most mosquito species, *Wolbachia* does not naturally occur in *Ae. aegypti* [335] and must be introduced introgressively into the *Ae. aegypti* population [336,337]. Promising efforts supported by the World Mosquito Programme that have been successively introduced *Wolbachia* into *Ae. aegypti* populations in 11 countries, on 3 continents (Oceania, South America and Southeastern Asia). Current efforts are looking at feasibly scaling up these approaches [337], and assessing the associated risks [336,338] prior to releasing *Wolbachia* infected *Ae. aegypti* into sub-Saharan Africa and other parts of the world [337,339].

To reduce arboviral transmission (together with other vector-borne diseases, such as malaria), and in response to the rise of insecticide resistance, the WHO and the international community have promoted the use of integrated vector management (IVM) as a progressive approach toward sustainable, cost-effective and enhanced vector control. The key elements for the successful implementation of IVM are: *i)* advocacy for community engagement and empowerment; *ii)* multisectoral collaboration and resource sharing; *iii)* integration of conventional approaches with novel modalities; *iv*) evidence-based decision making to guide implementation strategies; and, lastly, *v*) capacity building at local, national and regional levels [340]. Also, very recently, another WHO strategy was the launch of the Global Arbovirus Initiative on 31 March 2022 [10]. The Global Arbovirus Initiative is an integrated strategic plan to tackle emerging and reemerging arboviruses with epidemic and pandemic potential focusing on monitoring risk, pandemic prevention, preparedness, detection and response, and building a coalition of partners (https://www.who.int/news-room/events/detail/2022/03/31/default-calendar/global-arbovirus-initiative).

Additionally, ongoing efforts are taking place to understand the molecular mechanisms of transmission, disease pathogenesis and epidemiology of arboviruses using recent advances in biotechnology. These methods are high-throughput and make use of predictive abilities to reduce the gap between experimental knowledge and available resources. Combining predictive and experimental data is quintessential to providing innovative and sustainable solutions [339]. Predictive models, such as AlphaFold2 and machine learning, in combination with other bioinformatic tools, will aid in developing much improved omic results that will elucidate on underlying mechanisms [331,332]. CLIMADE, a global consortium based in South Africa, makes use of bioinformatic tools to enhance our global understanding of arboviral pathogenesis and epidemiology (https://climade.health/). Furthermore, with the AU-EU Innovation Agenda being established in 2023 (https://research-and-innovation.ec.europa.eu/system/files/2023–07/ec_rtd_au-eu-innovation-agenda-final-version.pdf), we anticipate that the many partnerships and collaborations formed will facilitate high-throughput analysis to be possible in circumstances where such analysis would never have been thought possible.

Another important recent initiative is the Quadripartite Secretariat for One Health. This multinational, multi-sectorial, multidisciplinary collaboration of four main agencies – FAO, UNEP, WHO, and WOAH – aims to achieve together what no one sector can achieve alone, thus providing a framework for collective and coordinated action to mainstream the One Health approach at all levels and provide policy and legislative advice and technical assistance to help set national targets and priorities (https://www.who.int/teams/one-health-initiative/quadripartite-secretariat-for-one-health).

Our approach to addressing these challenges ought to extend beyond mitigating the effects of climate change and should look at how we can achieve sustainable health (defined by [341] to better the lives of community members in the long run.

Conclusion

This review underscores the urgent need for heightened surveillance and control measures within the SADC region to effectively manage the risk posed by arboviruses. By understanding the factors that drive arboviral persistence and transmission in Southern Africa, public health officials and policymakers can develop more targeted and efficient strategies to mitigate the impact of arboviruses on both human and animal populations. Particularly, it is indicated that *i*) data-driven approaches with a One Health framework are critical; *ii*) vector control is currently the most effective preventive tool available to combat arbovirus transmission (despite the issue of insecticide resistance); and *iii*) strengthening surveillance is crucial to guide interventions and management of emergencies. In conclusion, continued research and collaboration across disciplines will be key in improving our understanding of these complex ecological interactions and enhancing preparedness against future arboviral outbreaks.

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