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Vectors and Vector-Borne Diseases: Biology, Epidemiology and Integrated Control Strategies

Roberta Rinaldi¹  | Carmen Scieuzo^{1,2}  | Federica De Stefano¹  | Luca Masucci^{3,4}  | Patrizia Falabella^{1,2} 

¹Department of Basic and Applied Sciences, University of Basilicata, Potenza, Italy | ²Spinoff XFlies s.r.l., University of Basilicata, Potenza, Italy | ³Department of Laboratory and Haematological Sciences, Fondazione Policlinico Universitario A. Gemelli Istituto di Ricovero e Cura a Carattere Scientifico (IRCCS), Rome, Italy | ⁴Department of Basic Biotechnological Sciences, Intensive and Perioperative Clinics, Università Cattolica del Sacro Cuore, Rome, Italy

Correspondence: Carmen Scieuzo (carmen.scieuzo@unibas.it) | Patrizia Falabella (patrizia.falabella@unibas.it)

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ABSTRACT

Vector-Borne Diseases (VBDs), transmitted by arthropods such as mosquitoes, ticks, fleas and sandflies, represent a significant threat to global health. These diseases can be caused by a variety of pathogens, including bacteria, viruses, protozoa, and helminths. This article explores classification and biological characteristics of vectors with a particular emphasis on their role in disease transmission. It also discusses the key genetic, environmental and socioeconomic factors that influence vector competence and vectorial capacity. The review focuses on the epidemiology, transmission mechanisms and clinical manifestations of major VBDs, including West Nile virus, Dengue, Leishmaniasis, Lyme disease and Malaria. In addition, it examines current diagnostic methodologies, vector surveillance strategies and molecular approaches for disease monitoring. Preventive and control strategies—such as integrated vector management, biological control and vaccination programs and innovative interventions—are also evaluated. A comprehensive understanding of the complex interactions among vectors, pathogens and hosts, together with advancements in detection and intervention technologies, is essential for the development of effective public health strategies.

1 | Introduction

The branch of entomology dedicated to understanding, preventing and controlling diseases is called medical entomology (Connelly 2019). Specifically, this field investigates the relationship between insects or other arthropods and human, domestic, or animal health (Laroche et al. 2017). This branch is divided into two main subfields: public health entomology, that focuses on the relationship between arthropods and human health, and veterinary entomology, that explores the relationship between arthropods and animal health (Gloria-Soria 2022).

Today, diseases transmitted by vectors are referred to as Vector-Borne Diseases (VBDs) (World Health Organization 2024a). These are infections of varied aetiology that share transmission

through arthropods acting as vectors (Baxter et al. 2017). The global impact of VBDs is profound, resulting in high levels of morbidity, mortality and economic burden (Centers for Disease Control and Prevention 2024). Many of these diseases are zoonotic, meaning they can be transmitted directly or indirectly between animals and humans. In particular, zoonotic VBDs may spread from one animal to another and from animals to humans, although human-to-human transmission is uncommon (Rahman et al. 2020). Over 150 zoonoses have been identified, and their study is a key focus in both human and veterinary medicine (Bidaisee and Macpherson 2014). Globalisation, including the international trade of animals and animal products, has increased their significance and their spread is further exacerbated by the growing presence of wild and domestic animals in urban environments (Hassell et al. 2017).

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1.1 | Vectors

According to the WHO's 2014 definition, vectors are living organisms capable of transmitting infectious diseases between humans or from animals to humans (National Academies of Sciences, Engineering and Medicine 2016). Vectors are often arthropods, such as mosquitoes, ticks, flies, fleas and lice, mostly blood-feeding insects that carry pathogens by feeding on an infected host and subsequently transmitting the pathogen to a new host (Socha et al. 2022). A species is considered a vector if: (a) its distribution coincides with that of the disease, (b) anthropophilia (or zoophilia) is demonstrated, (c) experimental evidence confirms its vectorial competence and (d) it is naturally found infected with the pathogen (Shaw and Catteruccia 2019).

Vectors are categorised based on their role in the pathogen's life cycle, making it essential to distinguish between mechanical and biological vectors (EFSA Panel on Animal Health and Welfare et al. 2017). Mechanical vectors transmit pathogens passively, by transferring microorganisms from one surface to another. For example, flies and cockroaches may contaminate food or objects by landing on them with their body parts or defecating on them. In this case, the vector does not support pathogen development or replication, transmission occurs through physical contact only. In contrast, biological vectors, such as mosquitoes and ticks, carry pathogens that undergo development and/or replication within their bodies before transmission to a new host, typically via a bite. However, the transmission of Chagas disease by triatomine bugs, where *Trypanosoma cruzi* enters via contamination of the bite site with infected feces or mucous membranes (e.g., conjunctival, nasal, or oral mucosa), is an example of how some biological vectors spread pathogens through alternative mechanisms, such as contamination with infected faeces during blood feeding (Meraj et al. 2025).

Part of the pathogen's biological cycle occurs within these arthropod vectors, where it may replicate, mature, or transform before being transmitted to a vertebrate host. This interaction is fundamental to the pathogen's survival and dissemination, significantly influencing transmission dynamics and overall disease epidemiology (McHugh 1994).

In this review, particular emphasis is placed on biological vectors, as they are responsible for the transmission of the majority of medically relevant VBDs and involve complex biological interactions with pathogens that critically influence disease epidemiology.

1.2 | Vector-Borne Disease Factors

Vector competence (predisposition to becoming infected) (Brady et al. 2016) and vectorial capacity (efficiency in transmitting pathogens) (Kramer and Ciota 2015) are determined by various factors: intrinsic factors such as genetic, cellular, physiological and behavioural traits, including adaptation of vectors to different climates, adaptation of pathogens to new vectors and/or hosts, expansion of vector/host/pathogen distribution and extrinsic factors including environmental factors (intensive agriculture, changes in land use, poor land management, urbanisation/deforestation), climatic changes (temperature,

precipitation, humidity, extreme events) and social-economic conditions (population growth, wars/conflicts, famine, inequalities, hygiene/health conditions, migration of people, availability/management of water resources, trade/transport of goods, globalisation) (Wu et al. 2025).

Vector competence refers to an arthropod's intrinsic ability to acquire, maintain and transmit a specific pathogen (Lewis et al. 2023). This capacity involves a combination of biological factors such as: the presence of suitable receptors in the vector for pathogen attachment, the ability of the pathogen to overcome the vector's immune responses and the pathogen's replication and development within the vector (de la Fuente et al. 2017) the transmission ability, defined as the percentage of infected vectors capable of infecting new hosts through blood feeding (Kramer 2016).

Vectorial capacity refers to the ability of a vector population to circulate a pathogen within a host population at a given place and time (Wu et al. 2025). The vectorial capacity quantifies the efficiency of a vector population in transmitting a pathogen over a defined period and it depends on multiple parameters, including: vector density (relative to the host population), biting rate (frequency of blood meals taken by vectors), survival rate (daily probability of vector survival) and extrinsic incubation period (time required for the pathogen to become infectious within the vector) (William et al. 2018).

2 | Parasitism

All relationships between species living in close association are symbiotic, including parasitism, where one species gains benefits at the expense of the other, to obtain survival advantages such as nutrients and shelter (Solomon et al. 2015).

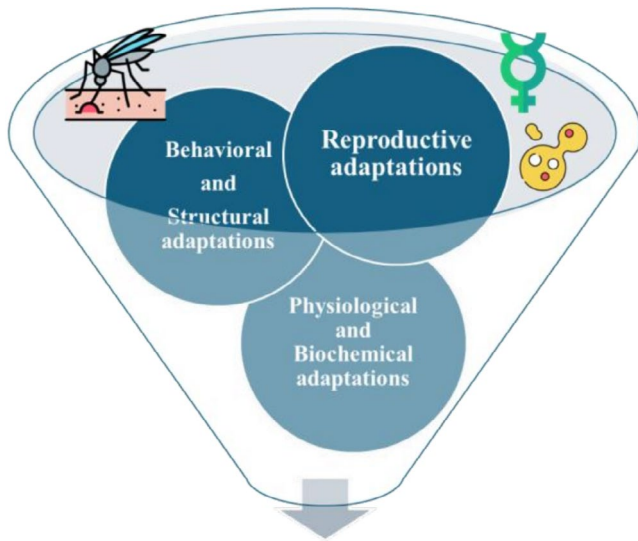
2.1 | Parasites and Their Classification

Parasites can be classified according to their dependence on the host, their location or biological characteristics and their life cycle (Olano et al. 2011). Based on host dependence, parasites are broadly classified as facultative or obligate. Facultative parasites are capable of living independently in the environment but may become parasitic in one or more hosts. Obligate parasites, in contrast, require resources from the host for at least part of their life cycle (Overstreet and Lotz 2016). Obligate parasites can be further subdivided into permanent obligate parasites, which remain permanently associated with the host (e.g., lice and scabies mites); periodic obligate parasites, which depend on the host during specific life stages and may leave it to lay eggs (e.g., fleas and ticks); and temporary obligate parasites, which interact with the host only briefly, typically for blood feeding (e.g., mosquitoes) (Mahmud et al. 2017). According to the location of the parasite they can be discerned into: ectoparasites, that live on the surface or outside the host's body (Hopla et al. 1994); endoparasites, that live inside the host and rely on at least one host genetic product, such as enzymes, signalling molecules, metabolic intermediates, or host-derived nutrients, to complete their life cycle. They can inhabit various tissues and organs, often exhibiting complex life cycles that may involve multiple hosts or developmental stages (Smyth 1994). The classification based on

life cycle is: Direct Life Cycles (Monoxenous) and Indirect Life Cycles (Heteroxenous). In the first case parasites complete their entire life cycle within a single host species (or closely related species). They move from one host to another through resistant forms such as cysts (protozoa) or eggs/larvae (metazoa). The transmission occurs directly from one infected individual to another of the same species. Heteroxenous parasites require two or more host species to complete their life cycle (Despommier and Karapellou 2012).

2.2 | Adaptations to Parasitic Life

Parasitic organisms exhibit enhanced and specialised structures and functions to maximise the efficiency of parasitism



Adaptations to parasitic life

FIGURE 1 | Key adaptations that facilitate parasitic life. These include reproductive strategies like hermaphroditism, parthenogenesis and pedogenesis, behavioural, structural, physiological and biochemical adaptations. [Colour figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com)]

(Janouškovec and Keeling 2016), including reproductive adaptations (such as hermaphroditism), parthenogenesis and pedogenesis (Thomalla and Wolfner 2023); behavioural adaptations (nocturnal feeding habits in particular for most *Anopheles* mosquito species align with human resting times, increasing transmission efficiency; or in *Aedes aegypti* preference for urban habitats enhances its proximity to human hosts (Rose et al. 2020)); structural adaptations (evolution of anchoring structures), such as piercing-sucking mouthparts capable of penetrating host skin (Bianco and Maizels 1989); physiological and biochemical adaptations/specialisations (loss of metabolic capabilities), relying instead on the host for pre-formed molecules; salivary secretions in vectors contain anticoagulants, vasodilators and immunomodulators that ensure blood flow and evade host immune responses (Šimo et al. 2017) (Figure 1). The greater the degree of adaptation to parasitism, the higher the dependence on the host, eventually leading to extreme parasitic specificity, in which the parasite can only exploit a single host species (Forbes et al. 2017; Buckingham and Ashby 2022).

2.3 | Biology and Classification of Vectors

Vectors of human diseases are organisms belonging mainly to the phylum Arthropoda (Figure 2). A large proportion of medically important insect vectors belong to the Diptera order and to the Acarina order, for arachnids (Socha et al. 2022). Of the approximately 100,000 species belonging to the Diptera order, about 10% are included in families that are of health importance because they are vectors of pathogens (mosquitoes, phlebotomes, simuliids, horseflies, tsetse flies) (Sarwar 2020). Various non-stinging flies, including the house fly, on the other hand, are mechanical vectors of pathogens because, by feeding on various substrates including faeces, they can contaminate food and drink with pathogens that are simply carried (Barin et al. 2010). Some species of non-hematophagous flies (*Cochliomyia hominivorax*, *Cordylobia anthropophaga* and in general the Sarcophagidae family) have the ability to colonise in the larval stage body cavities or tissues of vertebrates, including humans, giving rise to temporary infestations that go by the name of myiasis (Francesconi and Lupi 2012). Larvae of different dipteran vector species develop specific characteristics depending on the environments in which

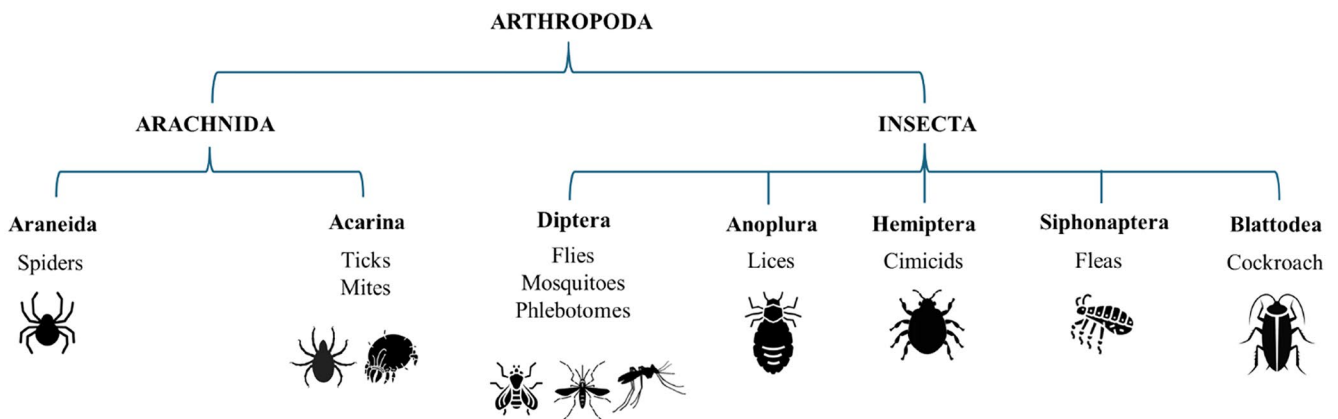


FIGURE 2 | Schematic representation of the main vectors species of parasitic diseases, involved in pathogen transmission. [Colour figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com)]

they live: for example, the larvae of the families Culicidae (standing water) and Simuliidae (running water) are aquatic, while the larvae of the families Psychodidae, Tabanidae and Glossinidae are terrestrial: in these cases, the female always chooses moist organic substrates to oviposit (Rodríguez-Martínez et al. 2023). A general characteristic peculiar to Culicidae (mosquitoes of greatest interest for VBD are those of the species: *Culex*, *Aedes*, *Anopheles*) is the capacity of their peculiar mouthparts, present exclusively in females, which enable them to which enable them to bite other animals and take their vital fluids, rich in proteins necessary for the completion of egg maturation (Ribeiro 2000). The Ixodidae, on the other hand, commonly known as 'hard ticks', are a family of arachnids characterised by the presence of a chitinous dorsal shield at all stages of development that covers the entire back in the male, while in the female it is present only anteriorly, as the posterior part consists of elastic tissue that allows the ingestion of large amounts of blood, due to their characteristic of being obligately hematophagous (Kahl 2018). Anoplura (true lice) are obligatory and permanent ectoparasites, hematophagous at all stages of development and can live exclusively on the body of the host (mammals, including humans), feeding on blood (Light et al. 2010).

3 | Major Vector-Borne Diseases and Transmission Mechanisms

Vector-borne diseases can have different aetiologies (Table 1): Protozoan Diseases (e.g., Malaria, Leishmaniasis, African Trypanosomiasis); Viral Diseases (e.g., Dengue, Chikungunya, Zika Virus Disease, Yellow Fever, West Nile Fever); Bacterial Diseases (e.g., Lyme); Rickettsial Diseases (e.g., Spotted Fever). Transfer from sources and reservoirs to the human host is ensured by a large number of vectors, each of which has its own behaviour and characteristics. Infection can be transmitted from vector to host through biting and piercing (stings) as the direct injection of pathogens during blood feeding (e.g., Malaria, Dengue) and the contamination via mechanical transfer, like passive transfer of pathogens via contaminated body parts such as deposition of vector faeces on host skin lesions or mucous membranes (e.g., Epidemic Typhus).

The VBDs discussed in the following sections were selected to provide representative case studies encompassing different classes of pathogens and vectors with distinct biological and ecological features. This approach was adopted to allow a structured and in-depth comparison of vector, pathogen and disease characteristics, rather than an exhaustive enumeration of all known vector-borne infections.

West Nile Fever (WNF): the history of the spread of WNF begins in 1937 in Uganda, in the West Nile district where the first isolation from the blood of a febrile woman occurred; subsequently, in 1957 in Israel, there were a dozen cases of encephalitis in humans, in 1960 in Egypt and France the first equine cases and in 1996 in Romania, 350 human cases with 17 deaths (Johnston and Conly 2000). In 1998, in Italy, in Padule di Fucecchio (Tuscany) there were 14 cases in horses, 6 of which were fatal and no human cases, but the presence of antibodies in people who shared the risk of mosquito bites with the horses has been detected (Lelli et al. 2012). Currently, the virus is endemic and causes sporadic

cases or epidemics in Africa, Asia, North America, Europe and Australia (Klingelhöfer et al. 2023). It is an acute disease caused by a virus from the Flaviviridae family (West Nile virus—WNV) and transmitted by the bite of infected mosquitoes (genus *Culex*). The reservoir of the disease includes certain types of birds (passerines and corvids) and mammals, among which horses and humans are incidental hosts (Campbell et al. 2002). The vector is a common mosquito widely distributed in tropical areas and temperate climate countries on all continents except Antarctica and appears in summer-autumn; it includes two main biological forms, distributed in Europe as follows: *Culex pipiens* (urban/city) and *Culex molestus* (rural areas, marshes). It usually bites birds, horses and humans and can serve as a bridge vector for the transmission of infections from birds to humans: wild birds, as reservoirs of infection, are amplifying hosts, while mammals are incidental hosts, as the low viremia is not sufficient to infect the vector (Wang et al. 2024). The virus is maintained in the environment through the continuous passage between hematophagous insects, which harbour the virus in their salivary glands and there is not a person-to-person transmission, except rare cases (e.g., transfusion, transplantation) (Colpitts et al. 2012). The symptoms of WNF in animals are: temperature 38.4°C–39.4°C, ataxia in all limbs, paresis and paralysis, hypermetria, hyperreactivity to touch and noise, acute or sub-acute spinal syndrome; while the symptoms in humans begin 3–15 days after the bite of the infected mosquito and, although often asymptomatic, can present: in 15%–20% of cases flu-like symptoms (2–5 days), fever, myalgia, headache, malaise, nausea, skin rashes, in less than 1% of cases neurological symptoms such as very high fever, meningitis, meningoencephalitis, or ataxia, weakness and rarely fulminant hepatitis, pancreatitis, myocarditis (Sejvar 2014). Certainly, the groups most at risk of contracting the virus are people over 50 years old, who are at high risk of developing severe neurological disease and need to take special precautions related to an integrated multidisciplinary system (clinicians, hygienists, veterinarians, etc.) and a conscious response plan, especially during the periods of increased mosquito activity, which vary geographically and are strongly influenced by climatic and ecological condition and mainly in high-risk areas such as irrigation areas, deltas and riverbanks, canals (Pal and Rebuma 2024).

Chikungunya: the term Chikungunya in the African language means 'that which bends' or 'contorts' and was first used during an epidemic in the 1950s in Tanzania to describe the impact of the joint symptoms caused by the disease (Galán-Huerta et al. 2015). Viruses that belong to the Togaviridae family of the *Alphavirus* genus are responsible for the disease and they are transmitted by the bite of infected mosquitoes of the *Aedes* genus (mainly *A. aegypti* and *Aedes albopictus*) (Bartholomeeusen et al. 2023). Monkeys, rodents, birds and other unidentified vertebrates are reservoirs of the viruses and, during epidemics, humans can also be carriers (Chhabra et al. 2008). The first known epidemic was described in 1952 in Tanzania, but as early as 1779 an epidemic had been described in Indonesia, possibly attributable to the same viral agent (Halstead 2015). Currently, the infection has been identified in over 60 countries in Asia, Africa, Europe and the Americas (Ali Mude et al. 2024). In Italy, local transmission outbreaks of chikungunya occurred in 2007 and 2017, supported by the *A. albopictus* mosquito (the so-called 'tiger mosquito') (Rezza 2018). The latter lays its eggs in dry

TABLE 1 | Summary table of major vector-borne diseases and their characteristics.

Disease	Etiological agent	Vector	Main symptoms	Prevention and treatment
West Nile Fever	West Nile Virus (Flaviviridae)	<i>Culex</i> spp.	Fever, myalgia, headache, ataxia, meningoencephalitis	Protection against mosquito bites, symptomatic support
Chikungunya	Chikungunya Virus (Togaviridae)	<i>Aedes</i> spp. (e.g., <i>A. aegypti</i> , <i>A. albopictus</i>)	High fever, severe arthralgia, skin rash	Repellents, hydration, anti-inflammatories
Zika Virus Disease	Zika Virus (Flaviviridae)	<i>Aedes</i> spp. (e.g., <i>Aedes aegypti</i> , <i>A. albopictus</i>)	Mild fever, rash, conjunctivitis, muscle/joint pain, possible complications like microcephaly in neonates	Mosquito repellents, avoiding mosquito bites, symptomatic treatment; no specific antiviral therapy
Yellow Fever	Yellow Fever Virus (Flaviviridae)	<i>Aedes</i> spp. (e.g., <i>Aedes aegypti</i>)	High fever, jaundice, vomiting, hemorrhagic manifestations	Vaccination, mosquito bite prevention; symptomatic treatment in severe cases
Dengue	Dengue Virus (Flaviviridae)	<i>Aedes</i> spp.	High fever, muscle pain, rash, hemorrhagic fever	Tetavalent vaccine (QDENGA), repellents, symptom management
Crimean-Congo Hemorrhagic Fever	Nairovirus (Bunyavirales)	<i>Hyalomma</i> spp. (ticks)	Fever, headache, muscle pain, bleeding	Tick protection, Ribavirin (antiviral therapy)
Lyme Disease	<i>Borrelia burgdorferi</i>	<i>Ixodes</i> spp. (e.g., <i>I. ricinus</i>)	Erythema migrans, fever, arthralgia, fatigue	Tick removal, antibiotics (e.g., doxycycline)
Leishmaniasis	<i>Leishmania infantum</i>	<i>Phlebotomus</i> spp.	Skin lesions, fever, hepatosplenomegaly (visceral form)	Repellents, specific therapy (liposomal amphotericin B)
Malaria	<i>Plasmodium</i> spp. (e.g., <i>P. falciparum</i> , <i>P. vivax</i>)	<i>Anopheles</i> spp.	Fever, chills, anaemia, neurological symptoms (cerebral)	Bed nets, chemoprophylaxis, specific therapy (e.g., artemisinin)
Sleeping Sickness	<i>Trypanosoma brucei gambiense/rhodesiense</i>	<i>Glossina</i> spp.	Fever, lymphadenopathy, neurological changes (advanced stage)	Protection from tsetse flies, pharmacological treatments (e.g., eflornithine)
Relapsing Fever	<i>Borrelia</i> spp. (e.g., <i>Borrelia recurrentis</i> , <i>Borrelia hermsii</i>)	Ticks (soft ticks) or lice (<i>Pediculus humanus corporis</i>)	Recurring fever episodes, chills, headache, myalgia, nausea	Antibiotics (e.g., doxycycline, penicillin), vector control (lice or ticks)
Tick-borne encephalitis	Tick-borne Encephalitis Virus (Flaviviridae)	<i>Ixodes</i> spp. (e.g., <i>I. ricinus</i> , <i>I. persulcatus</i>)	Fever, headache, seizures, weakness, encephalitis	Vaccination (three initial doses), symptomatic support

(Continues)

TABLE 1 | (Continued)

Disease	Etiological agent	Vector	Main symptoms	Prevention and treatment
Epidemic Typhus	<i>Rickettsia prowazekii</i>	Lice (<i>Pediculus humanus corporis</i>)	High fever, headache, rash, myalgia, severe complications like meningoenzephalitis	Antibiotics (e.g., doxycycline, chloramphenicol), hygiene to prevent lice infestation
Spotted Fever	<i>Rickettsia</i> spp. (e.g., <i>R. rickettsii</i> , <i>R. conorii</i>)	Ticks (e.g., <i>Ixodes</i> spp., <i>Dermacentor</i> spp.)	Fever, headache, rash (spotted), muscle pain, complications like organ failure (severe cases)	Antibiotics (e.g., doxycycline), tick control, supportive therapy for severe symptoms

and dimly lit environments (saucers, flower beds, ornamental basins and fountains, containers, gutters, etc.) where they can survive even harsh winters. It is a very aggressive insect, biting during the cooler hours of the day and resting at night on vegetation and it also has a typically urban distribution (Caputo et al. 2020). *A. aegypti*, on the other hand, is more efficient, bites during the day, causes epidemics, remains infected for life, is more anthropophilic, has transovarial transmission, is a poor flyer and does not survive the winter season, although the eggs can survive desiccation for a period of over a year (Li et al. 2017). Among the symptoms of the disease, it is necessary to distinguish between the first and second phases. During the first phase, lasting approximately 6–10 days, the patient exhibits very high fever (over 39°C) which usually resolves after 4 days, headache, severe fatigue, muscle pain and severe joint pain capable of limiting movement, more frequently in the peripheral joints and the patient tends to remain still to avoid pain. Meanwhile, in the second phase (lasting 2–3 days), a rash with widespread itching all over the body may appear and sometimes the fever may reoccur; less common, but possible, are the appearance of conjunctivitis, gastrointestinal symptoms, including nausea and vomiting and seizures. Healing occurs in about a week or a little more, but some patients continue to complain of joint pain for months (Cunha and Trinta 2017).

Zika Virus Disease: the Zika Virus (ZIKV), belonging to the Flaviviridae family and the Flavivirus genus, is an arbovirus mainly transmitted by the *A. aegypti* and *A. albopictus* mosquitoes (Song et al. 2017) and was identified for the first time in 1947 in Uganda; the virus remained confined to sporadic outbreaks until the explosive epidemic of 2015–2016 in Latin America, which revealed its ability to cause severe congenital anomalies, such as microcephaly and neurological complications in adults, including Guillain-Barré syndrome, a rare autoimmune disorder that attacks peripheral nerves, leading to progressive weakness and paralysis and that is often triggered by infection from *Campylobacter jejuni*, as well as viruses like influenza and SARS-CoV-2 (Metzler and Tang 2024). The 2015 epidemic also highlighted the potential of the virus to spread through sexual transmission and vertical transmission from mother to foetus, confirming its relevance within the TORCH virus family (*Toxoplasma gondii*, rubella virus, cytomegalovirus and herpes simplex) (Fazecas et al. 2024). The epidemiology of the ZIKV is influenced by human mobility and the habitat of the vectors, with epidemics recorded in Brazil, Colombia and more recently in Bangladesh, where a concurrent outbreak with dengue was reported in 2023 (Hasan et al. 2025). Infection is usually asymptomatic or mild, with symptoms such as fever, rash, conjunctivitis, arthralgia and myalgia that resolve spontaneously (Song et al. 2017). However, in infants exposed in utero, the virus can cause a variety of persistent neurodevelopmental deficits (Mulkey et al. 2024). The etiopathology of ZIKV is linked to its neurotropism, with a direct action on neuronal progenitors leading to cortical dysfunctions, inflammation and brain atrophy (Agner et al. 2025). It has also been demonstrated that a previous ZIKV infection can predispose individuals to more severe forms of dengue due to antibody-dependent enhancement (Katzelnick et al. 2020).

Yellow Fever: Yellow Fever is an acute viral disease caused by the Yellow Fever Virus (YFV), a member of the Flaviviridae

family and the *Flavivirus* genus, transmitted mainly by *A. aegypti* mosquitoes in urban environments and *Haemagogus* spp. and *Sabethes* spp. in wild transmission cycles (Monath and Vasconcelos 2015; Damasceno-Caldeira et al. 2023). Identified for the first time in the 17th century, yellow fever has caused numerous devastating epidemics in the Americas, Africa and Europe. The epidemiology of yellow fever is characterised by cyclical outbreaks in Africa and Latin America, with an increase in incidence between 2011 and 2021 (Liang and Dai 2024). Nigeria was one of the most affected countries between 2017 and 2023, with climatic and socio-demographic factors favouring viral transmission (Akar et al. 2025). Brazil has also recorded a resurgence of the infection with an increasing risk of urban transmission (Damasceno-Caldeira et al. 2023). The virus infects monocytes and dendritic cells, spreading to the liver, kidneys and other organs, causing massive hepatic necrosis and multiorgan failure in the most severe cases (van Leur et al. 2021). Prevention is based on vaccination with the 17D vaccine, which provides long-lasting immunity and is recommended for at-risk populations and travellers heading to endemic areas (Coulter and Messer 2025). However, vaccination coverage remains uneven, with concerns related to safety and immune response in different population groups (Silva-Fernandes et al. 2025). Climate change and the expansion of mosquito habitats are increasing the risk of transmission, highlighting the need for more effective control strategies (Aliaga-Samanez et al. 2024). There is no specific antiviral therapy for yellow fever; the treatment is supportive and includes the management of hemorrhagic and metabolic complications. Recent studies have explored the use of therapeutic plasma exchange to treat the more severe forms of the disease with promising results (Oh et al. 2025). Epidemiological surveillance and vaccination remain the most effective strategies to prevent future epidemics.

Dengue fever: it is a viral disease transmitted by the bite of infected mosquitoes of the *Aedes* genus, especially *A. aegypti* (the most effective vector) and *A. albopictus*, which mainly bite during dawn and dusk. The reservoir is represented by humans. In some regions, there is the possibility of a wild (enzootic) cycle involving monkeys and other primates (Weaver and Barrett 2004; Gwee et al. 2021). The Dengue virus (DENV), member of the genus *Orthoflavivirus*, belonging to the Flaviviridae family (like that of Yellow Fever), exists in four serotypes of viruses: Den-1, Den-2, Den-3 and Den-4 and it is transmitted to humans through the bites of mosquitoes that have, in turn, bitten an infected person (Murugesan and Manoharan 2020). There is therefore no direct transmission between humans, even though humans are the main host of the virus. The virus circulates in the blood of the infected person for 2–7 days, and during this period, the mosquito can pick it up and transmit it to others. Infection with one serotype generally confers lifelong immunity, but only against that serotype (Islam et al. 2021). It is endemic in more than 100 countries in the southern hemisphere, and 2.5 billion people (2/5 of the world population) are currently at risk of dengue, and the WHO estimates 50 million cases worldwide each year (World Health Organization 2023). In Europe, in recent years, the cases reported by expatriates, members of humanitarian organisations, military personnel, immigrants, but often also travellers from endemic areas, have increased (Duvignaud et al. 2024). Already in 1999, the European Network reported 1117 cases among European travellers (Gardner et al. 2012).

There are no particular at-risk groups: anyone exposed to the infected vector could get infected, and since there are 4 serotypes, it is possible to get sick with Dengue multiple times throughout life (Rodriguez-Roche and Gould 2013). Transmission of DENV occurs through mosquito bites (Yang 2017). Preventive measures include restricting blood donations in affected areas, reducing mosquito breeding sites and avoiding high-risk zones.

Crimean-Congo Hemorrhagic Fever: Crimean-Congo Hemorrhagic Fever (CCHF) is a viral hemorrhagic fever caused by a virus from the Nairovirus family of the Bunyavirales order, transmitted by ticks belonging to the *Hyalomma* genus, commonly found in livestock and wild animals (Flick and Whitehouse 2005). The virus causes severe epidemics of viral hemorrhagic fever, with a mortality rate of 10 to 40%. The disease spreads to humans through a tick bite or through contact with infected animal tissues during and immediately after slaughter. Person-to-person transmission can occur through contact with the blood, secretions, organs, or other bodily fluids of infected individuals (Karanam et al. 2025). The disease was first described in Crimea in 1944 and only in 1969 was it discovered that the pathogen causing hemorrhagic fever in Crimea was the same virus that affected a child in Congo in 1956: from the combination of these two place names, the name CCHF was created (Okesanya et al. 2024). The geographical area of the virus circulation is very extensive and includes parts of Asia, Africa, the Middle East, Eastern Europe and Southwestern Europe. The duration of the incubation period depends on the mode of infection: after infection from a tick bite, the incubation period is usually from one to 3 days, with a maximum of 9 days and the incubation period following contact with infected blood or tissues is 5 or 6 days, with a maximum of 13 days (Kaya et al. 2011). The onset is sudden, with symptoms such as fever, myalgia, dizziness, neck pain and stiffness, back pain, headache, eye burning, photophobia (sensitivity to light) and nausea, vomiting, diarrhoea, abdominal pain and sore throat may also appear in the initial phase, followed by mood swings and confusion; after 2–4 days, agitation may be replaced by drowsiness, depression and fatigue (Whitehouse 2004). Among the symptoms of advanced disease, the following can be noted: abdominal pain may localise in the upper right quadrant, with hepatomegaly, tachycardia, lymphadenopathy, petechial rash (a skin eruption caused by bleeding), hemorrhagic rash of the mucous membranes of the mouth and throat and petechiae may give rise to larger eruptions called ecchymoses and other hemorrhagic phenomena. Usually, there is evidence of hepatitis and in patients with severe forms of the disease, rapid renal failure, sudden hepatic or pulmonary failure occurs after the fifth day of illness (Eslava et al. 2024). The main approach in managing the disease is general supportive therapy and symptom treatment. The antiviral drug Ribavirin has been used to treat the infection with evident benefits, but currently, there is neither a proper therapy nor a specific vaccine (Muzammil et al. 2024).

Lyme disease: the etiological agent of the disease is the bacterium *Borrelia burgdorferi*: the bacterium *B. burgdorferi sensu stricto* is present in Europe and is the only infectious agent in North America, while *B. afzelii* and *B. garinii* are present in Europe, Asia and Africa. These bacteria are transmitted to humans through the bite of hard ticks of the genus *Ixodes*, in particular *Ixodes ricinus* and possibly also by *Amblyomma*

and *Dermacentor* (dog ticks). The main reservoirs of the infection are represented by wild animals (rodents, roe deer, deer, foxes, hares) that function as amplifiers (Radolf et al. 2021). Lyme disease is primarily transmitted by four species of *Ixodes* worldwide: *Ixodes scapularis* (northeastern and north-central United States), *Ixodes pacificus* (western United States), *I. ricinus* (Europe) and *Ixodes persulcatus* (Asia) (Stone et al. 2017). Lyme disease was identified in 1976 during an outbreak in the town of Lyme, Connecticut and it is now the most reported tick-borne disease in the United States (Steere et al. 2004). *B. burgdorferi* penetrates through the skin at the tick bite site, and after 3–32 days, the bacterium migrates locally in the skin around the puncture area, spreads via the lymphatic system and causes regional adenopathy, or disseminates hematogenously to organs or other skin sites (Hyde 2017). Initially, an inflammatory reaction (erythema migrans) occurs before the activation of a significant antibody response to the infection (serological conversion) and this is why Lyme disease is divided into three stages: early localised, early disseminated and late stage (Mahajan 2023). The early and late stages are typically separated by a symptom-free period. The symptoms of early disseminated disease begin days or weeks after the appearance of the primary lesion, when the bacterium spreads throughout the body. Immediately after the onset, about half of untreated patients develop multiple lesions; patients also develop a musculoskeletal, flu-like syndrome (malaise, fatigue, chills, fever, headache, neck stiffness, myalgia and arthralgia) that can last for weeks (Hatchette et al. 2014). The characteristic erythema migrans, the best clinical indicator of Lyme disease, is the first sign of the disease: it appears in at least 75% of patients, starting as a red macule or papule at the bite site, generally located on the proximal part of a limb or on the trunk, especially at the level of the thigh, buttock, or armpit, 3–32 days after the tick bite. In very severe cases, neurological changes may occur, in about 15% of patients, weeks or months after erythema migrans (usually before the arthritic attack), typically lasting months and generally resolving completely; late manifestations (which occur years after onset) include antibiotic-sensitive skin lesions (chronic atrophic dermatitis) and chronic alterations of the central nervous system, such as polyneuropathies or mild forms of encephalopathy with mood, memory and sleep disturbances. Some patients experience symptoms such as fatigue, headaches, joint and muscle pain and cognitive problems after effective antibiotic treatment. These symptoms are collectively known as post-treatment Lyme disease syndrome (Marques 2022). Although Lyme disease responds to antibiotics in all stages, early treatment is more effective in preventing complications and antibiotics such as doxycycline, amoxicillin, or cefuroxime taken orally for 10–14 days are effective in the early stages of the disease (Talbot et al. 2023). Antibiotics can also help alleviate many of the symptoms caused by Lyme disease such as meningitis, arthritis and a heart rhythm disturbance (arrhythmia), for which a temporary pacemaker may also be needed (Radesich et al. 2022). Subsequently, antibiotics may also be effective in advanced stages of the disease, as they eradicate the bacteria and, in most individuals, relieve the arthritis (Talbot et al. 2023). However, even after effective antibiotic treatment, other symptoms, collectively referred to as post-treatment Lyme disease syndrome, persist in some cases, such as fatigue, headaches, joint and muscle pain and mental problems; the cause of the persistence of these symptoms is unknown, but treatment with additional antibiotics is not effective (Marques 2022).

Leishmaniasis: Leishmaniasis is an infectious and contagious disease caused by the parasite *Leishmania* spp., a protozoan characterised by possessing a particular structure called kinetoplast, containing extranuclear DNA and closely associated with a basal body from which a single flagellum originates, transmitted by the bite of sandflies (hematophagous insects belonging to the genus *Phlebotomus*) (Serafim et al. 2020). It affects both domestic and wild animals (canids and rodents), in addition to humans, with the elderly, children and immunocompromised individuals being particularly exposed (Morales-Yuste et al. 2022). Human Leishmaniasis is primarily present in the tropical and subtropical regions of Africa, Central and South America, Asia and the Mediterranean region. In recent years, the number of regions becoming Leishmania-endemic has significantly increased, accompanied by a rise in the number of animal and human cases (Cosma et al. 2024). The biological cycle of *L. infantum* begins with the blood meal of the sandfly on an infected dog and the ingestion of the aflagellated form of the protozoan (amastigote); in its digestive system, the pathogen assumes the infective flagellated form (promastigote), which is reinoculated into the skin of the vertebrate host with the subsequent meal, thus restarting the cycle (Dostálová and Volf 2012). Following their release, the amastigote forms will infect other macrophages and, through the bloodstream, primarily spread to the hemolymphopoietic organs (such as the liver, spleen, lymph nodes and bone marrow) and the skin, generating a systemic infection (Martín-Martín et al. 2015). Other sandflies will ingest the amastigotes with the infected blood meal, thus starting a new cycle. The vector takes a blood meal on an infected animal and becomes infected by ingesting the amastigotes. These transform into promastigotes in the midgut of the insect and then migrate anteriorly to the pharynx where they transform into highly mobile metacyclic promastigotes, which localise in the insect's proboscis and can be transmitted to a new host through the insect's bite (Kumar et al. 2023). The duration of the cycle in the vector insect ranges from 4 to 20 days depending on the temperature; the cycle is completely inhibited below 10°C. The promastigote penetrates the blood of the vertebrate host and is phagocytosed by macrophages. In the cellular context, the transformation of the protozoan from the promastigote form to the amastigote form occurs (Díaz and Ponte-Sucre 2018). So, there is an active replication of amastigotes through binary fission, until the host cell ruptures. The free amastigotes in the blood go on to parasitize other macrophage cells, thereby invading the organism. The cycle is completed when the female sandfly introduces the amastigote into its intestine through a meal of infected blood (Qi et al. 2004). In addition to the protective mechanisms inherent to the parasite, the type of immune response developed by the host also conditions the possibility that the amastigotes can survive in the macrophage, reproduce and disseminate within the host. Indeed, if adequately stimulated by certain lymphokines, macrophages are capable of increasing their lysosomal and oxidative activity, which are toxic to the parasites (Olivier et al. 2005). Leishmaniasis manifestations in humans are divided into cutaneous, mucocutaneous and visceral, that can lead to death (Kyari 2024). Local treatment (through the use of creams or cutaneous injections) is indicated for cutaneous forms with small uncomplicated lesions (Madusanka et al. 2022). For the treatment of visceral leishmaniasis, liposomal amphotericin B (Ambisome) is indicated for intravenous infusion. Other drugs used for the treatment of leishmaniasis

include miltefosine, deoxycholate amphotericin, sodium stibogluconate, pentamidine and oral 'azoles' (ketoconazole, itraconazole and fluconazole) (Sundar and Chakravarty 2015).

Malaria: human malaria is a potentially fatal disease caused by parasitic protozoa transmitted to humans by infected female mosquitoes of the genus *Anopheles* (Arrow et al. 2004). The species of *Plasmodium* responsible for human malaria are: *Plasmodium falciparum*, the agent of the so-called malignant tertian malaria (the parasite's replication cycle in human blood, schizogonic cycle, every 48 h), endemic in sub-Saharan Africa, Asia and Latin America, Papua New Guinea and some islands in the eastern Pacific; *Plasmodium vivax*, the agent of benign tertian malaria, prevalent in temperate climate zones, as well as in East Africa, Asia and Latin America; *Plasmodium ovale*, with the two subspecies *P. ovale curtisi* and *P. ovale wallikeri*, agents of benign tertian forms, very common mainly in West Africa, rarely in the Pacific; *Plasmodium malariae*, responsible for benign quartan malaria (blood schizogonic cycle every 72 h), widespread in tropical and subtropical areas in an uneven manner and always with a rather low frequency; *Plasmodium knowlesi*, present in various areas of Southeast Asia and responsible for malaria in some forest-dwelling monkeys, shows morphological resemblances depending on the stage, with the forms in the initial blood stage similar to those of *P. falciparum* and those in the advanced stage similar to those of *P. malariae* (Antinori et al. 2012; Phillips et al. 2017; World Health Organization 2024c; World Health Organization 2024d). Due to the numerous human outbreaks initially identified in Borneo and then in many other regions of Southeast Asia, since 2008 *P. knowlesi* has been considered the fifth species of *Plasmodium* responsible for malaria in humans (Ruiz Cuenca et al. 2022). The biological cycle of *Plasmodium* takes place in two obligatory hosts, a vertebrate as the intermediate host and a female mosquito as the definitive host, also known as the vector (Venugopal et al. 2020). Malaria transmission begins when an infected female *Anopheles* mosquito injects salivary gland sporozoites into the human host during a blood meal. Following inoculation, sporozoites rapidly migrate to the liver, where they undergo the exo-erythrocytic (hepatic) stage of development. After maturation, merozoites are released into the bloodstream and invade red blood cells, initiating the erythrocytic cycle responsible for the clinical manifestations of malaria. A fraction of the blood-stage parasites differentiates into male and female gametocytes, which are the only stages infectious to mosquitoes (Bousema and Drakeley 2011). When a mosquito feeds on an infected host, it ingests these gametocytes. In the mosquito midgut, they undergo gametogenesis and fertilisation to form zygotes, which subsequently develop into motile ookinetes and then into oocysts on the gut wall. Each oocyst produces numerous sporozoites that migrate to the salivary glands, completing the sporogonic cycle and rendering the mosquito capable of transmitting the parasite to a new human host (Aly et al. 2009). Inside the human host, the parasite undergoes complex intracellular developmental changes while evading immune defenses (Chandley et al. 2023). After invading hepatocytes and multiplying through hepatic schizogony, merozoites infect red blood cells and continue asexual replication through repeated erythrocytic cycles. Depending on the *Plasmodium* species, these cycles typically occur at intervals of approximately 48 or 72 h (Goswami et al. 2022). After several replication cycles, a subset of parasites differentiates into gametocytes

that may persist in the bloodstream and enable transmission to feeding mosquitoes (Sato 2021). The symptoms, depending on the *Plasmodium* species, appear after 7, 15, or more days from the bite of the infected mosquito. They are of various natures, but usually consist of fever, often very high, headache, vomiting, diarrhoea, sweating and shaking chills, all symptoms, at least initially, common to any flu-like syndrome or other infections. The pathogenicity of the plasmodia is linked to their ability to invade and destroy red blood cells, which is followed by the main symptoms of the disease, represented by recurrent febrile episodes and anaemia (Milner 2018). *P. falciparum* represents the most severe form in terms of morbidity and mortality. It reaches very high parasitemia and, in addition to causing severe anaemia, can obstruct the capillaries of the brain (cerebral malaria) or other vital organs (kidneys, spleen, liver) (Bogovic and Strle 2015). Worldwide, there are more than 400 different species of *Anopheles* mosquitoes; about 60 are capable of transmitting malaria, but only 30 are of major importance as malaria vectors (Pimenta et al. 2015). Adult mosquitoes, being ectothermic organisms, can reproduce and survive at environmental temperatures not lower than 15°C–18°C and at a high degree of humidity, always in the presence of water bodies, essential for their developmental cycle (Caminade et al. 2019). Mosquitoes lay their eggs in water, from which larvae emerge and, after four stages, pupate and give rise to the adult. Adults live on average from a few days (males) to several weeks (females). Female mosquitoes need to take blood meals every 3–4 days for egg maturation. The biting habits of *Anopheles* are crepuscular and nocturnal, times when the risk of contracting malaria is higher in endemic areas (Singh et al. 2024). According to the 2024 World Malaria Report, in 2023, an estimated 263 million malaria cases were reported globally, with an incidence of 60.4 cases per 1000 people at risk. The WHO African Region continues to bear the heaviest burden, accounting for 94% of malaria cases worldwide in 2023 (World Health Organization 2024e). Chemoprophylaxis is particularly indicated for travellers heading to endemic areas (Tickell-Painter et al. 2017). In an endemic area, for at-risk categories such as children under 5 years of age and pregnant women, intermittent preventive treatment is recommended (Figueroa-Romero et al. 2022). Vector control represents one of the fundamental measures to prevent and reduce the transmission of malaria (Hemingway 2014). Generally, the outcome of pathogen transmission is largely influenced by the biological and immune characteristics of insect vectors (Pascale et al. 2014). However, recent challenges in malaria control also arise from pathogen-related factors, particularly the emergence and spread of artemisinin-resistant *P. falciparum* strains in Southeast Asia and, more recently, in Africa, which pose a significant threat to global malaria elimination efforts. Nonsynonymous mutations in the propeller region of the *P. falciparum* kelch 13 (PfK13) gene are the primary cause of artemisinin resistance, which is typified by delayed parasite clearance after therapy; these mutations include complicated physiological responses, including proteotoxic stress pathways, oxidative stress management and metabolic adaptations and are associated with decreased drug sensitivity during the early intraerythrocytic developmental stages (Zhu et al. 2022). The main measures implemented are focused on reducing contact between mosquitoes and humans. Personal protective measures against mosquito bites are represented by insecticide-treated bed nets. Interventions to reduce mosquito density are carried out with residual insecticide

treatments in homes, with the reduction of larval breeding sites through environmental sanitation and with the use of larvicides or natural predators of mosquito larvae (Pryce et al. 2022; Tusting et al. 2013; Unwin et al. 2023).

Sleeping sickness: the sleeping sickness, also known as 'Human African trypanosomiasis', is a parasitic disease caused by protozoa belonging to the genus *Trypanosoma*, transmitted to humans by the bite of the tse-tse fly (genus *Glossina*) infected by humans or animals carrying the parasite (Franco et al. 2014; Ponte-Sucre 2016). Tse-tse flies are found only in sub-Saharan Africa, but only some species transmit this disease. Among the 36 countries considered endemic, 7 are the most affected, with the Democratic Republic of the Congo representing 84% of the cases (Papagni et al. 2023). When an infected tse-tse fly bites a person or an animal, it injects a form of protozoa into the skin that can cause infections, called trypomastigotes. The protozoa move towards the lymphatic system and the bloodstream, and, inside the host, they change shape and move towards organs and tissues throughout the body, including lymph and spinal fluid and they also multiply in the bloodstream (Alfituri et al. 2020). A fly ingests the protozoa when it bites an infected person and the protozoa form travel to the fly's salivary glands, multiply and transform into trypomastigotes (the form that is injected when the fly bites a person) (Geiger et al. 2015). Human African trypanosomiasis can take two forms, depending on the parasite involved: *Trypanosoma brucei gambiense* and *T. brucei rhodesiense* (Pepin and Meda 2001). *T. brucei gambiense* is present in 24 countries in West and Central Africa and is the form currently responsible for over 98% of reported cases of sleeping sickness, causing a chronic infection. A person can be infected for months or even years without showing the signs or symptoms pathognomonic of the disease. When more evident symptoms appear, the patient is often in an advanced stage of the disease, which affects the central nervous system (Pepin and Mpia 2006). *T. brucei rhodesiense* is present in 13 countries in East and Southern Africa and currently accounts for less than 2% of reported cases and causes an acute infection (Pepin and Meda 2001). Animals can harbour the pathogen harmful to humans, especially *T. b. rhodesiense*, for which domestic and wild animals are an important reservoir. Animals can also be infected by *T. brucei gambiense* and act as reservoirs to a lesser extent. The symptoms vary depending on the stage of the disease: the first signs and symptoms are observed a few months or a few weeks after the infection and the disease develops rapidly and invades the central nervous system (Bouteille and Buguet 2012; Chappuis et al. 2005). Indeed, in the hemolymphatic stage, there is an initial phase in which the trypanosomes multiply in the subcutaneous tissues, blood and lymph, leading to fever attacks, headaches, swollen lymph nodes, joint pain and itching (Ponte-Sucre 2016). In the neurological or meningoencephalic stage (the second phase in which the parasites cross the blood-brain barrier to infect the central nervous system), the most evident symptoms of the disease appear: behavioural changes, confusion, sensory disturbances, seizures and poor motor coordination, sleep cycle disturbances, which give the disease its name (Kristensson et al. 2010; Idro et al. 2022; Kennedy and Rodgers 2019). The drug used for the treatment of the disease depends on the species causing the infection and the potential spread to the brain and cerebrospinal fluid; if the infection has not reached the brain and cerebrospinal fluid, Suramin (for *T.*

rhodesiense) and Pentamidine (preferable for *T. gambiense*) are used; if the infection has already spread to the brain and cerebrospinal fluid, Melarsoprol, Eflornithine (only for the *T. gambiense* species) and Nifurtimox in combination with less frequent doses of Eflornithine in areas of Africa where frequent administration of Eflornithine is logistically difficult (only for the *T. gambiense* species) are employed. Fexinidazole was introduced as the first oral treatment for human African trypanosomiasis caused by both *T. brucei gambiense* and *T. brucei rhodesiense*, representing an important advance by simplifying treatment administration and improving accessibility in endemic regions (Matovu et al. 2025).

Relapsing Fever: Relapsing Fever (RF) is an infectious disease caused by spirochetes of the genus *Borrelia*, transmitted to humans by soft ticks of the genus *Ornithodoros* (Tick-Borne Relapsing Fever, TBRF) or by the human body louse *Pediculus humanus corporis* (Louse-Borne Relapsing Fever, LBRF) (Warrell 2019). Historically, LBRF has caused devastating epidemics in conditions of war, famine and overcrowding, while TBRF persists as an endemic disease in regions of Africa, the Middle East, Central Asia and the Americas (Tang et al. 2024). The epidemiology of RF varies depending on the mode of transmission. LBRF, primarily caused by *Borrelia recurrentis*, is widespread in environments with poor hygiene and tends to manifest in epidemic outbreaks (Warrell 2019). TBRF, on the other hand, is endemic and caused by various species of *Borrelia*, including *Borrelia hermsii* in North America, *Borrelia duttonii* in East Africa and *Borrelia hispanica* in the Mediterranean (Koutantou et al. 2024). The presence of soft tick vectors, which feed quickly and often go unnoticed, makes epidemiological surveillance more complex (Jakab et al. 2022). The etiopathology of RF is characterised by the ability of *Borrelia* to evade the immune response through antigenic variations of surface proteins, resulting in recurrent febrile episodes. After an incubation period of 3–18 days, the disease manifests with high fever, headache, myalgia and splenomegaly, followed by remissions and relapses due to the re-emergence of antigenic variants. LBRF can be more severe than TBRF, with a higher risk of neurological and hepatic complications (Senbill et al. 2025). The main treatment is the administration of antibiotics, with doxycycline or tetracyclines as first-line drugs. In severe cases or in pregnant women, erythromycin is preferred. The therapy can induce a Jarisch-Herxheimer reaction, an acute inflammatory response due to the massive lysis of spirochetes, which requires symptomatic management (Bissett et al. 2018). Timely access to antibiotics and the improvement of sanitary conditions remain key strategies to reduce the morbidity and mortality associated with RF.

Tick-borne encephalitis: Tick-Borne Encephalitis (TBE) is a central nervous system infection brought on by the tick-borne encephalitis virus (TBEV), which is mostly spread to people by tick bites. The virologist Lev Aleksandrovič Zilber originally identified the neurotropic TBEV as the aetiology of TBE almost 75 years ago (Bogovic and Strle 2015). It is a spherical RNA virus with a lipid envelope that belongs to the Flaviviridae family's genus of *Flavivirus*. Three TBEV subtypes—the European, Siberian and Far-Eastern subtypes—are identified by genetic study (Mansfield et al. 2009). Small rodents are the principal TBEV reservoirs and hosts in the wild; humans are only unintentional hosts and have no part in maintaining TBEV in the

wild. *Ixodes ricinus* is the primary vector of TBEV in Europe, while *Ixodes persulcatus* is the vector in parts of Eastern Europe, Russia and far-east Asia. In Japan, however, *Ixodes ovatus* ticks have been shown to carry the Far-Eastern TBEV subtype (Hudopisk et al. 2013). From northern and eastern Asia via Russia to northern and central Europe, the virus's natural foci span a wide geographic range. Additionally, it has spread to northern Africa, Greece, Italy, Turkey and the southern Balkans (Mansbridge et al. 2022). In 27 European nations, tick-borne encephalitis has been identified; the Czech Republic, Baltic nations and Slovenia have the highest yearly incidence rates (5–18.6/100,000). Additionally, there is proof that TBE is not adequately recorded in some European nations. With an estimated 10,000–15,000 cases in the northern hemisphere of Eurasia in 2010, TBE was the most significant virus illness spread by ticks and there are significant differences in the incidence change during the past 30 years between nations (Schley et al. 2023). Local TBEV replication takes place following an infected tick bite; fever, headaches, vomiting and weakness are some of the first symptoms. Seizures, arm or leg weakness, disorientation, lack of coordination and trouble speaking are some of the serious symptoms that may appear a few days later. Sometimes the early symptoms go away entirely after a few days, but more serious symptoms start to appear around a week later (Riccardi et al. 2019). It is thought that dendritic skin cells, also known as Langerhans cells, are the first cells to replicate the virus and carry it to nearby lymph nodes. The TBEV spreads from this starting point to extraneural tissues, including the liver, spleen and bone marrow, where it multiplies further to sustain viremia for a few days (Růžek et al. 2010). The virus most likely enters the brain through four different pathways during the viremic phase, which clinically corresponds to the first phase of TBE: peripheral nerves; extremely vulnerable olfactory neurons; transcytosis through brain capillary vascular endothelial cells; and virus diffusion between capillary endothelial cells (Mandl 2005). No specific antiviral treatment is available for TBE: patients are usually hospitalised due to neurological disease severity, and treatment comprises antipyretics, analgesics, antiemetics, maintenance of water and electrolyte balance and adequate nutrition and, if necessary, administration of anticonvulsant agents to prevent neurological and systemic complications and avoid further neuronal damage; intubation and ventilatory support are provided if patients have neuromuscular paralysis leading to respiratory failure (Růžek et al. 2019).

Rickettsial Diseases (e.g., Epidemic Typhus and Spotted Fever): Rickettsial diseases are zoonotic infections caused by bacteria of the genus *Rickettsia*, transmitted by arthropod vectors. Epidemic typhus is caused by *Rickettsia prowazekii*, a pathogen that spreads through the human body louse (*Pediculus humanus corporis*), particularly under conditions of overcrowding and poor hygiene, as documented in historical epidemics during wars and famines (Paris and Dumler 2016). Spotted fevers belong to the Spotted Fever Group Rickettsioses (SFGR) and are caused by several *Rickettsia* species, including *Rickettsia rickettsii*, the aetiological agent of Rocky Mountain fever and *Rickettsia conorii*, responsible for Mediterranean button fever. These pathogens are mainly transmitted by hard ticks of the family *Ixodidae*, including *Rhipicephalus sanguineus*, the main vector for *R. conorii* in the Mediterranean basin (Hammami et al. 2024) and *Dermacentor variabilis* for *R. rickettsii* in North

America (Paris and Dumler 2016). Historically, epidemic typhus has had a devastating impact, with outbreaks documented in times of war and famine, while spotted fevers comprise a group of infections that vary in severity and geographical distribution (Paris and Dumler 2016). The epidemiology of these diseases is complex and depends on environmental and socioeconomic factors. Studies in India have shown a high seroprevalence in rural and peri-forest areas, where exposure to vectors is highest (Devamani et al. 2020). In addition, a high incidence of Mediterranean spotted fever and murine typhus with neurological manifestations has been reported in the Mediterranean basin (Hammami et al. 2024). Symptoms vary depending on the pathogen, but generally include high fever, headache, skin rash and in severe cases, neurological or vascular complications (Hammami et al. 2024). Transmission of SFGR occurs predominantly via ticks, as evidenced by studies conducted in the Wayanad district of India, where ticks have been identified as key vectors (Viswanath et al. 2025). A new outbreak of spotted fever was recently described in Odisha state, indicating the need for increased diagnostic attention for acute fevers of unknown origin (Premnath et al. 2025). In Japan, epidemiological monitoring showed a different geographical distribution for Japanese spotted fever and tick-borne typhus (scrub typhus), with seasonal and regional variations (Kinoshita et al. 2021). The main treatment remains the early administration of doxycycline, which is highly effective if started early (Gupta et al. 2024). However, late diagnosis can increase the risk of serious complications, making the development of faster and more accessible diagnostic methods crucial.

4 | Prevention and Control of Vector Borne Diseases

Resolution WHA 70.16, which was adopted by the World Health Assembly in May 2017, required the member nations to create or modify national vector control plans and strategies in order to conform to guidelines. The Global Vector Control Response 2017–2030 (GVCR), approved by the World Health Assembly in the same 2017, provides a new strategy to strengthen vector control worldwide through increased capacity, improved surveillance, better coordination and integrated action across sectors and diseases (World Health Organization, UNICEF 2017; Tourapi and Tsioutis 2022). The priority activities defined in the GVCR belong to four pillars, which are: strengthening inter- and intra-sectoral action and collaboration; engaging and mobilising communities; improving vector surveillance, monitoring and evaluation of interventions; expanding and integrating tools and approaches (Robbiati et al. 2024; World Health Organization 2020a). These pillars of action are in turn supported by two key elements: improving vector control capacity and ability and increasing basic and applied research and innovation (Devine et al. 2019; Wilson et al. 2020). Refocusing vector control programs is necessary to achieve this, and it will be aided by enhanced technical capability, better infrastructure, more robust monitoring and surveillance systems, and increased community mobilisation (World Health Organization 2020b; Eastman et al. 2024). In the end, this will facilitate the adoption of a thorough vector management strategy that will help meet national and international objectives related to diseases, as well as the Sustainable Development Goals and universal health

coverage (The Sustainable Development Goals Report 2024, United Nations; Decouttere et al. 2021). The WHO Secretariat offers nations and development partners technical, normative and strategic advice on how to improve vector control as a cornerstone strategy based on GVCR for disease prevention and outbreak response by providing evidence-based guidance for controlling vectors and protecting people against infection; providing technical support to countries so that they can effectively manage cases and outbreaks; supporting countries to improve their reporting systems and capture the true burden of the disease; providing training (capacity building) on clinical management, diagnosis and vector control; and supporting the development and evaluation of disease management technologies (World Health Organization 2020b; Stensgaard et al. 2019).

Before 2023, there was no vaccine to prevent Chikungunya virus infection, nor specific medications to shorten the course of the disease or prevent its rare complications; generally, treatment consists of managing symptoms through rest, ample hydration and the use of antipyretics and analgesics for fever and pain (anti-inflammatories are generally contraindicated due to the risk of bleeding) (Côrtes et al. 2023). In 2023, Ixchiq (VLA1553) vaccine received approval in the United States in November and in 2024 in Canada and Europe (Chen et al. 2024). It is a live attenuated vaccine that contains a weakened version of CHIKV, which can cause symptoms similar to those of a natural CHIKV infection. It was administered as a single intramuscular injection and was evaluated for safety and immunogenicity in a 3rd phase clinical trial, randomised, double-blind and placebo-controlled. The safety profile of the vaccine showed uniform tolerability across all age groups tested. The most common side effects reported to the FDA for the vaccine approval included fatigue, headache, muscle and joint pain, fever and pain at the injection site. No more than 2% of vaccinated individuals experienced severe adverse reactions similar to chikungunya that required medical intervention (Ly 2024).

The prevention of ZIKV infection is based on vector control through the use of insecticides, the removal of mosquito breeding sites and individual protection with repellents and mosquito nets (Barzon et al. 2016). Currently, there is no vaccine approved for human use, although several strategies are under development, including inactivated virus vaccines, subunit vaccines and RNA vaccines (Malik et al. 2024; Buitrago-Pabón et al. 2024). A recent approach based on the rational design of attenuated live viruses has shown promising results in preclinical studies (Roopin et al. 2025). The serological diagnosis of the ZIKV is complicated by cross-reactivity with the DENV, but new multi-epitope tests are improving diagnostic accuracy (Pereira et al. 2024). There is no specific antiviral treatment for ZIKV; the therapy is symptomatic and supportive, aimed at reducing fever and pain (Tajik et al. 2024).

For example, in terms of prophylaxis, there are now two tetravalent vaccinations against Dengue fever on the market that are made with live attenuated viruses. The first to be approved is Sanofi Pasteur's DENVAXIA (chimeric yellow fever dengue [CYD]), a tetravalent attenuated chimeric vaccine based on the YFV 17D (Thomas and Yoon 2019). It expresses the membrane precursor protein (prM) and the surface protein (envelope) of the four serotypes of DENV, which can prevent the disease caused

by all four serotypes (Thomas and Yoon 2019). However, it is only approved for people who have previously contracted DENV and are between the ages of 6 and 45 who live in areas where the virus is endemic (Gubler and Halstead 2019). TAKEDA GMBH's QDENG A vaccine is the second to receive approval. It is an entirely DENV-based tetravalent attenuated vaccine that is approved for use beginning at age 4 regardless of prior DENV infection and is recommended for preventing the illness caused by all four serotypes (Angelin et al. 2023). Recent WHO recommendations emphasise that dengue vaccination strategies should be tailored to local epidemiological contexts and integrated with vector control and surveillance programs, as vaccine effectiveness varies depending on prior exposure and transmission intensity.

For some VBDs, such as CCHF, preventive measures still just include: control of animals and ticks, reduction of the risk of transmission from ticks to humans through protective clothing, approved chemical repellents on exposed skin and clothing, elimination or control of tick infestations in animals or in stables and barns, avoiding areas where ticks are abundant and the seasons when they are most active, as well as avoiding physical contact with infected individuals and controlling the infection in healthcare facilities (de la Fuente et al. 2024; World Health Organization 2024e). Also, for the majority of rickettsial diseases, prevention is based on vector control and individual protection, including repellents and personal hygiene measures; currently, there is no widely available vaccine for most rickettsiosis, although vaccine candidates for epidemic typhoid have been explored (Paris and Dumler 2016).

The same is for Leishmaniasis: to date, there is no vaccine against leishmaniasis and ongoing strategies include vaccines based on killed or attenuated parasites, recombinant proteins and DNA, aiming to generate a robust IL-12-driven Th1 immune response (Ayala et al. 2024). So, despite many progresses, the development of effective vaccines is hindered by the antigenic diversity of the parasite and the complexity of the host immune response (de Carvalho Clímaco et al. 2023), and prevention relies on avoiding sandfly bites, limiting outdoor activities at night, wearing long clothing and applying repellents to exposed skin (González et al. 2015).

After more than three decades of research, the first malaria vaccine, RTS, S/AS01, demonstrated partial protection against *P. falciparum* malaria in children. Following pilot implementation programs initiated in 2019 in Malawi, Ghana and Kenya, the World Health Organization (WHO) recommended its wider deployment in 2021 as part of integrated malaria control strategies. In 2023, WHO also recommended a second vaccine, R21/Matrix-M, marking a significant milestone in malaria prevention. Although these vaccines represent major advances in public health, their protective efficacy remains partial and they are designed to complement established interventions such as vector control, chemoprevention and surveillance (Laurens 2020; Dicko et al. 2024).

Vaccination represents the most effective preventive measure against TBE, although personal protective measures and vector avoidance remain important complementary strategies. Several TBE vaccines are currently licensed in Europe and Asia and

standard immunisation schedules include a primary series followed by periodic booster doses to maintain long-term protection. The vaccination for TBE consists of three doses of the initial immunisation in the first year, with booster shots every 3–5 years to provide adequate protection (Chrdle et al. 2016). Based on empirical data, extending the booster interval to 10 years following the completion of the original immunisation regimen may also provide enough protection (Schmidt et al. 2022).

In general, it is useful to emphasise that prevention always involves personal measures (use of repellents, insecticides, vaccines, etc.), community measures (reduction of larval habitats, environmental management, vaccination programmes, awareness and health education programmes, also having access to clean water and sanitary facilities, etc.) and farm and herd control (vaccination and monitoring; elimination or treatment of infected animals). Specifically, the main preventive measures for anthroponoses include close monitoring of the health status of animal populations to be subjected, if necessary, to vaccination; remediation interventions in the environment; stray control, isolation, chemoprophylaxis and, if necessary, culling of infected animals; interventions on the population (health education for at-risk groups); immunisation (vaccination, also of those exposed to the infection); reporting to the Health Authority and disinfestation, if necessary (Chan et al. 2011; Carpenter et al. 2022; Al-Osaimi et al. 2024). For instance, environmental management aimed at reducing breeding sites can be combined with biological control strategies to achieve long-term vector suppression, while chemical control may be selectively employed to rapidly reduce vector populations during outbreaks or periods of increased transmission risk. The integration of these approaches allows for greater flexibility, sustainability and adaptation to local ecological and epidemiological conditions, ultimately facilitating the control and potential elimination of VBDs. Changes in behaviour are essential to lowering the burden of vector-borne illnesses. To help people learn how to defend themselves and their communities against mosquitoes, ticks, bugs, flies and other vectors, WHO collaborates with partners to raise public awareness and educate the public (World Health Organization 2024b).

5 | Biological and Biological-Based Strategies for Control of Vectors

Biological control represents an environmentally sound and efficient approach to suppressing pests and reducing their impact by employing natural enemies. It can also be economically advantageous and is often considered one of the most sustainable components within integrated strategies for limiting malaria transmission. Among its key benefits are its eco-friendly nature, as it does not introduce chemical pollutants into the environment and its high degree of selectivity, which minimises risks to non-target organisms. Another important strength is the self-perpetuating capacity of biological control agents, which can reproduce and disperse naturally, thereby maintaining long-term effects without continuous reapplication. In addition, pests generally develop resistance to biological control agents far more slowly, if at all, compared to chemical control strategies. However, biological control also has some limitations. The success of biological control agents is strongly influenced

by environmental conditions, which can lead to variability in outcomes. Their action is relatively slow compared to chemical insecticides, often requiring several days to weeks before noticeable reductions in mosquito populations occur. Moreover, the effectiveness of biological control is sometimes unpredictable, as it depends on complex ecological interactions. Different biological control strategies are currently employed, including direct killing of vectors, altering vector behaviour in ways that increase mortality, and generating populations of mosquitoes that are sterile or incapable of transmitting pathogens (Okumu and Moore 2011; Wilson et al. 2020). Biocontrol aims to reduce the density of vectors, their longevity and their competence in transmitting pathogens, using natural organisms or symbionts, and is therefore now considered a cornerstone of long-term strategies for preventing insect-borne diseases and is the base for the Integrated Vector Management (IVM), a comprehensive approach that relies on the coordinated use of multiple complementary strategies, including biological, environmental and chemical interventions, tailored to local ecological and epidemiological conditions (Campbell-Lendrum et al. 2005; Venegas et al. 2024; Wyckhuys et al. 2025). Although biological and biological-based interventions constitute a major component of modern vector control, they represent only one element of the broader IVM framework. Effective and sustainable vector suppression requires the coordinated integration of biological strategies with environmental management and carefully targeted chemical interventions. Considering these complementary components is essential for understanding the full operational scope and potential of IVM. Even though IVM techniques have been shown to be beneficial, there are a number of difficulties in their development and execution. These include the necessity of ongoing entomological and epidemiological surveillance, the need for intersectoral collaboration among public health, environmental and governmental entities and logistical and financial limitations. Furthermore, the effectiveness of IVM initiatives depends on community acceptance and engagement, especially for treatments that need repeated control measures or environmental alterations. The creation of successful IVM methods is further complicated by ecological variability, pesticide resistance and socioeconomic issues, underscoring the significance of long-term commitment and context-specific planning.

While biological strategies represent a central pillar of IVM, effective vector control also relies heavily on environmental management and judicious chemical interventions. Environmental approaches focus on reducing or eliminating vector breeding habitats through source reduction, improved water management, waste disposal and urban planning (World Health Organization 2012a; van den Berg et al. 2012). Permanent environmental modifications, such as drainage and infrastructure improvements, can sustainably suppress vector populations, whereas temporary environmental manipulations, including seasonal water management and habitat disruption, provide flexible responses to fluctuating transmission risks. These strategies not only decrease reliance on insecticides but also generate broader public health and ecological benefits (Beier et al. 2008; World Health Organization 2017).

Chemical control remains an essential component of IVM when applied in a targeted and evidence-based manner. Interventions such as indoor residual spraying (IRS), insecticide-treated nets

(ITNs and LLINs) and larvicidal applications are particularly valuable during outbreaks or in high-transmission settings (Wilson et al. 2020). Within an IVM framework, chemical tools are guided by entomological surveillance and resistance monitoring to minimize environmental impact and delay the evolution of insecticide resistance (Hemingway et al. 2016). The coordinated use of biological, environmental and chemical approaches allows for complementary action across spatial and temporal scales (Beier et al. 2008; van den Berg et al. 2012; Wilson et al. 2020). For example, environmental management can reduce breeding sites, biological agents can maintain long-term suppression of larval populations and selective chemical interventions can rapidly decrease adult vector density during peak transmission periods. The strength of IVM lies in this adaptive integration of multiple tools, tailored to local ecological, epidemiological and socio-economic conditions.

For example, the use of aquatic predators is one of the oldest and most proven forms of biocontrol: larvivorous fish such as *Gambusia affinis*, *Esomus danricus*, *Rasbora daniconius*, *Trichogaster fasciata*, *Trichogaster lalia*, *Poecilia reticulata*, *Cyprinus carpio*, *Ctenopharyngodon idella*, *Clarias fuscus* and many others have been used in several countries for the control of *Culex* and *Aedes* larvae, with significant results in stagnant and peri-urban environments (Rawani 2023; Kendie 2020). Simultaneously, cyclopoid copepods such as *Mesocyclops* spp. have shown effectiveness in reducing *A. aegypti* populations, particularly in large-scale community projects in Asia and Latin America (Thomas 2018; Roudine et al. 2023). Predatory insects, including Odonata, Notonectidae and Dytiscidae, also play a significant ecological role in natural and agricultural systems by substantially limiting larval proliferation (Huang et al. 2017; Machtinger and Geden 2018; Niang et al. 2018). However, these approaches require careful assessments of the impacts on other aquatic species and integration with environmental measures to ensure effectiveness and safety (Bueno-Marí et al. 2015). Additionally, among microbial agents, *Bacillus thuringiensis israelensis* (Bti), *Bacillus sphaericus* (Bs) and *Lysinibacillus sphaericus* are the most widely used biolarvicides, characterised by high specificity towards culicids and a reduced impact on non-target organisms (Rawani 2023; Kendie 2020): commercial formulations have demonstrated effectiveness in both urban and rural settings and are an integral part of the WHO guidelines for larval control without risks for humans and other non-target organisms (World Health Organization 2005). Once ingested by larvae, Bti releases crystal proteins that act as stomach toxins, causing mortality. Bti is often applied as a water-dispersible powder in mosquito breeding sites. Bs functions like Bti but shows enhanced effectiveness in organically polluted water, particularly against *Culex* species. Symbiont-based strategies have had a transformative impact. The identification of various symbiotic bacteria residing in the digestive tract and reproductive organs of arthropods has opened new opportunities for developing innovative strategies to combat major VBDs. In mosquitoes, such microbial partners can negatively affect host survival, disrupt reproductive processes and diminish their capacity to transmit pathogens. *Wolbachia* is a maternally inherited bacterium capable of colonising both the reproductive tissues, allowing it to persist across generations and somatic tissues, where it can interfere with pathogen development. Transmission typically occurs vertically through the host's eggs

and the bacterium is known to alter host biology in multiple ways. The main outcome affects reproductive apparatus, including: feminization of genetic males, induction of parthenogenesis (although rare), male-killing and cytoplasmic incompatibility (the most widespread phenotype), where infected male sperm are modified in a way that causes embryonic failure when mated. Because infected males cannot transmit *Wolbachia* to their progeny, converting males into females or inducing parthenogenesis increases the likelihood of maternal transmission (Niang et al. 2018). Beyond reproductive alterations, *Wolbachia* can also modulate host immunity, physiology, behaviour and ecology, making it a particularly promising tool for reducing the transmission of medically and veterinary relevant pathogens. Transinfection approaches have successfully established *Wolbachia* in *A. aegypti*, where different strains have been shown to shorten adult lifespan, lower susceptibility to arbovirus infection and induce cytoplasmic incompatibility to suppress mosquito populations (Bian et al. 2013). In malaria vectors such as *Anopheles gambiae* and *Anopheles stephensi*, experimental or transinfected *Wolbachia* strains have been associated to impaired *Plasmodium* development and reduced fecundity (Gomes et al. 2017). *Wolbachia*-mediated antiviral effects can also have an impact on host genetics. For instance, studies in *Culex quinquefasciatus* and *A. aegypti* suggest that *Wolbachia* can enhance expression of the host Vago protein, thereby activating innate immune pathways that restrict virus infection such as WNV and DENV (Asad et al. 2018). Strains such as wAnga (*A. gambiae* *Wolbachia*), which stably infect the ovaries and also colonise somatic tissues where *Plasmodium* develops, may suppress malaria transmission by competing for host resources or enhancing immune responses that kill parasites (Shaw et al. 2016). The introduction of *Wolbachia pipiensis* into *A. aegypti* populations has been shown to reduce longevity and competence for arboviruses such as Dengue, Zika and Chikungunya (Iturbe-Ormaetxe et al. 2011; Niang et al. 2018) and specifically, pilot programs in Australia, Vietnam and Brazil have demonstrated substantial reductions in Dengue incidence (Huang et al. 2017; Thomas 2018). Other agents for biological control are fungi that do not need to be ingested to establish infection; indeed, they invade their hosts directly through the cuticle. This unique infection route makes them effective against a broad spectrum of insects, including those with piercing-sucking mouthparts (Olagunju 2024). While most entomopathogens rely on oral uptake and subsequent colonisation of the gut, entomopathogenic fungi typically breach the host cuticle. Species such as *Beauveria bassiana* and *Metarhizium anisopliae* have been demonstrated to effectively reduce mosquito vector populations, highlighting their potential role in IVM programs, reducing survival, blood-feeding activity and fertility, and have the additional advantage of being effective even against insecticide-resistant populations (Thomas 2018; Wyckhuys et al. 2025; Kudesia et al. 2025; Rani et al. 2023; Doloi 2021). Renuka et al. (2023) reported that entomopathogenic fungi significantly decreased the survival of adult *A. stephensi*. Darbro et al. (2011) reported 93 isolates of entomopathogenic fungi from six species (*B. bassiana*, *M. anisopliae*, *Isaria fumosorosea*, *Isaria farinosa*, *Isaria flavovirescens* and *Lecanicillium* spp.) with potential activity against *A. aegypti*. These findings highlight the diverse arsenal of biological agents available for mosquito control and the potential of integrating them into broader vector management programs. In addition to fungi,

entomopathogenic nematodes (EPNs) (mermithids) and their symbiotic bacteria represent another promising class of biocontrol agents, which can develop inside mosquito larvae and drastically reduce their survival and microsporidia, which are capable of interfering with development and reproduction (Okamoto and Amarasekare 2012; Kendie 2020). El-Sadawy et al. (2020) showed that EPNs could efficiently target of *Phlebotomus papatasi* larvae, while Cardoso et al. (2015) observed high mortality in third- and fourth-instar *A. aegypti* larvae following exposure to infective juveniles of *Heterorhabditis indica*. Importantly, EPNs and their bacterial partners are generally considered safe for warm-blooded vertebrates, including humans, further supporting their suitability for use in vector control. Simultaneously, paratransgenesis is also being explored to manipulate commensal or symbiotic gut microbes of vectors to produce antiparasitic or antiviral molecules, reducing pathogen transmission (Morchón et al. 2023; Jeyakodi et al. 2024; Olagunju 2024; Venegas et al. 2024). While this approach is promising, it requires thorough evaluations of genetic stability and ecological effects. Mosquito transgenesis has emerged as a tool based on the introduction of novel genetic elements into their genomes. Two main approaches are commonly used: reducing or eliminating vector competence, and decreasing vector populations by shortening their lifespan. Both strategies limit the spread of pathogens affecting humans and animals. For example, *A. gambiae* and *A. stephensi* have been engineered to interfere with parasite development, thereby reducing or preventing disease transmission. Despite this potential, genetic modification often leads to a reduction in mosquito fitness, which may hinder the stable dissemination of desired traits into wild populations. Among the most innovative strategies are genetic techniques that involve releasing incapable of producing fertile offspring males: (1) the Sterile Insect Technique (SIT) involves raising males in a laboratory and sterilising them through irradiation or genetic modification. Once released into the wild, these males mate normally, but the females lay non-viable eggs, resulting in a gradual population decline (Wang et al. 2023). In this approach, irradiated sterile males carrying damaged sperm are released to mate with wild females, resulting in no viable offspring and, if applied at sufficient scale, potential population elimination; adaptations of SIT have been applied to *A. aegypti* to reduce transmission of arboviruses such as dengue, chikungunya and Zika (Phuc et al. 2007) and it has also been successfully applied to control populations of tsetse flies (*Glossina* spp.), vectors of human African trypanosomiasis: large-scale SIT programs, including the eradication campaign on Zanzibar Island, demonstrated the effectiveness of releasing sterile males to suppress and locally eliminate tsetse populations, significantly reducing disease transmission risk (Abd-Alla et al. 2013); (2) the Incompatible Insect Technique (IIT), on the other hand, exploits the principle that infected males, when mating with uninfected females, produce eggs that do not develop. This mechanism, known as 'cytoplasmic incompatibility', results in a progressive reduction in populations (Niang et al. 2018; Baton et al. 2021). In recent years, it has been shown that combining SIT and IIT makes the strategy safer and more effective. These techniques have already shown encouraging results in pilot programs on *A. aegypti* and *A. albopictus* and are now also being studied for *Anopheles* species, the vectors of malaria (Wyckhuys et al. 2025). Unlike the classical approach, which induces sterility early, 'release of insects with dominant lethality' (RIDL) depends on

late-acting lethal gene to suppress populations. The RIDL system itself was originally developed as an autocidal method for the Mediterranean fruit fly, *Ceratitis capitata* and is based on tetracycline-regulated transcriptional control. In this system, a tetracycline-controlled transactivator (tTA) is expressed under a positive feedback loop. In the laboratory, tetracycline supplied in the diet suppresses tTA overexpression, preventing lethality and enabling colony maintenance (Gong et al. 2005). In field populations, however, the absence of tetracycline causes unchecked tTA expression, which drives lethal effects, particularly in female offspring when combined with female-specific promoters. Despite the progress, some critical issues remain, including: the heterogeneity of field results, with differences linked to local ecology, climate and social acceptability (Niang et al. 2018; Roudine et al. 2023); technical limitations such as environmental persistence (fungi) and large-scale production (nematodes, microsporidia), formulation costs (Bti, *B. bassiana*); regulatory and socio-economic aspects, which influence widespread adoption (Bueno-Marí et al. 2015; Venegas et al. 2024); and finally, ecological effects with the risk of impact on non-target organisms, which can be mitigated through selectivity and careful preliminary studies (Halbert 2008; Thomas 2018). In the years ahead, the One Health approach and the development of contextualised IVM toolboxes emerge as priorities for consolidating the role of biocontrol, highlighting the need for standardised metrics, economic evaluations and co-design with local communities (Venegas et al. 2024; Wilson et al. 2020; Wyckhuys et al. 2025). Designing and implementing effective IVM programs requires robust planning frameworks and long-term institutional commitment. Successful strategies depend on continuous entomological and epidemiological surveillance to guide evidence-based decision-making and to adapt interventions to changing transmission dynamics. Intersectoral collaboration among public health authorities, environmental agencies, urban planners and community stakeholders is essential but often difficult to achieve in practice (van den Berg et al. 2012; Wilson et al. 2020). Financial constraints, limited technical capacity and the need for sustained community engagement can further hinder implementation, particularly in resource-limited settings.

Ecological heterogeneity, rapid urbanisation, climate variability and the spread of insecticide resistance add further complexity to IVM planning. As a result, scalable programs must incorporate adaptive management principles, regular monitoring and evaluation and locally tailored intervention packages. Strengthening governance structures, investing in capacity building and promoting community participation are critical for ensuring the long-term sustainability and effectiveness of integrated vector control strategies (Venegas et al. 2024).

6 | Diagnostic Methodologies for VBDs

Research in biomedical sciences is rapidly advancing, and therapies of the future are increasingly personalised, based on the patient's genomic profile and the molecular characterisation of their disease (Chala and Hamde 2021). This progress has led to a growing interest in omics approaches based on the principle that complex systems can be better understood when analysed as a whole (Figure 3). They require vast datasets, often obtained

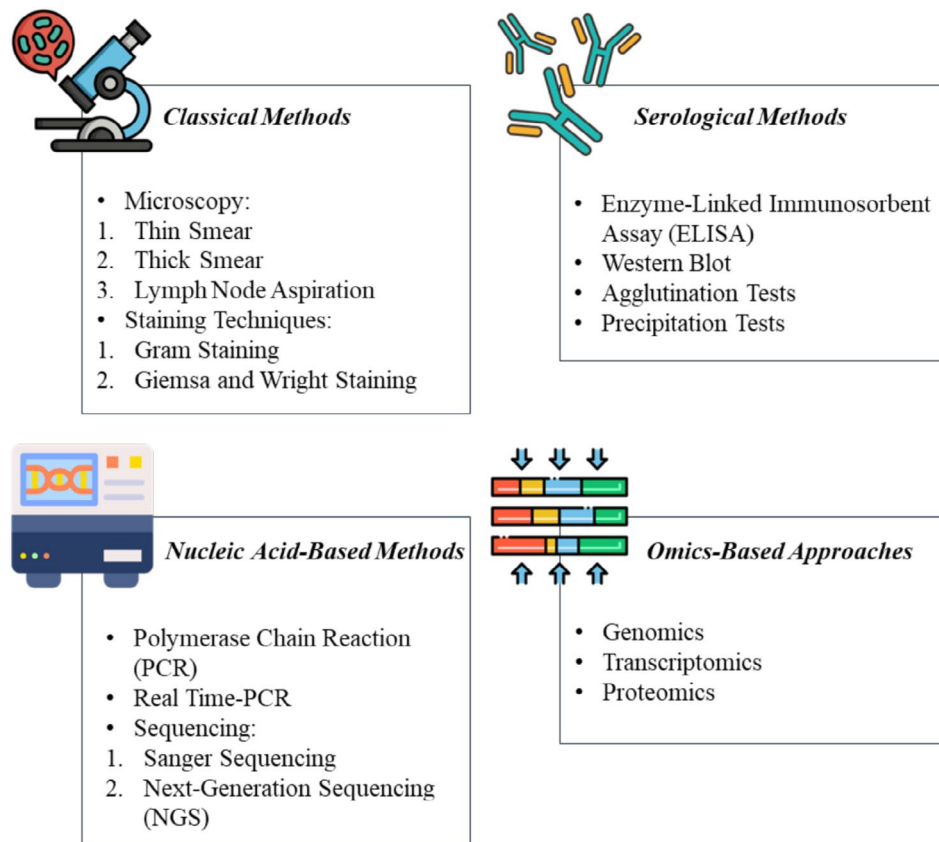


FIGURE 3 | Diagnostic methodologies for vector-borne diseases. Overview of the main techniques used for the detection and identification of pathogens, including molecular methods, serological tests, microscopy and advanced diagnostic approaches for effective disease monitoring and control. [Colour figure can be viewed at [wileyonlinelibrary.com](https://onlinelibrary.wiley.com)]

through genome sequencing (Vashisht et al. 2023). Classical methods, also known as direct methods, primarily include microscopic examination and staining techniques, which are fundamental tools for diagnosing various parasitic infections. Manual microscopic examination of blood slides, considered the reference standard for malaria detection, is widely used in most endemic zones due to its cost-effectiveness and simplicity, as it does not require a sophisticated infrastructure. There are two types of blood smear: thin smear and thick smear. (a) The thin smear provides high specificity but lower sensitivity; it is useful for identifying parasite species and stages based on morphological features and is optimal for determining parasitemia at higher levels. (b) The thick smear offers 10–15 times greater sensitivity due to the larger blood volume observed; however, it has reduced specificity as the lysed red blood cells obscure morphological details. The disadvantages of these tests are that they are time-consuming and labor-intensive and require experienced and qualified microscopists to ensure accurate diagnoses (Hernández and Ramírez 2013). Microscopic analysis remains the primary method for diagnosing *P. falciparum* infections, particularly in resource-limited regions. In a study conducted in sub-Saharan Africa, the thick smear method demonstrated a sensitivity of over 90% in detecting *P. falciparum* in symptomatic individuals (Maltha et al. 2013). Another direct method is lymph node aspiration: during early infections, such as *T. brucei gambiense*, trypanosomes can be observed in lymph node fluid using low magnification (10×–40×). Sensitivity ranges from 40% to 80%, being higher during the early stage (World Health Organization 2012b). The importance of lymph node testing in

the diagnosis of VBDs lies in the fact that, in the case of *T. brucei gambiense*, it can colonise the lymphatic system before being detectable in blood, making lymph node analysis a potentially more sensitive diagnostic tool. For this reason, a field study in the Democratic Republic of Congo demonstrated that lymph node aspiration significantly improved early-stage *T. brucei gambiense* detection compared to blood smear analysis, especially in patients with mild symptoms (Jamonneau et al. 2010). Furthermore, blocking the LYVE-1 receptor, which is essential for lymphatic colonisation, has been shown to impair the survival of the parasite, suggesting that targeted interventions in the lymphatic system could offer new therapeutic strategies against infection (Machado et al. 2023). Staining techniques also play a crucial role in microscopic diagnosis. Among them, for example, Gram staining is widely used for bacterial identification, while Giemsa and Wright stains are essential for detecting *P. falciparum* in malaria and other blood parasites. These staining methods enhance the visibility of parasites and cellular structures, allowing for more accurate identification and classification of infections (Kumar et al. 2025). A comparative study in India found that Giemsa staining had greater diagnostic accuracy than rapid diagnostic tests for malaria, particularly in cases of low parasitemia (Das et al. 2021). Serological methods, also referred to as indirect methods, are based on the detection of specific antigens or antibodies using, for example: Enzyme-Linked Immunosorbent Assay (ELISA), which enables researchers to quantify antigens or antibodies with high sensitivity and precision. Variants include direct, indirect and competitive ELISA (Hayrapetyan et al. 2023); Western Blot assay, often used as a

confirmatory test in various diagnostic applications, detecting individual proteins within a complex mixture through antigen–antibody interactions after protein separation by gel electrophoresis (Mishra et al. 2017); agglutination tests to measure antigen–antibody interactions, with results assessed through visible cross-linking in diluted solutions; and precipitation tests (Alcon-Chino and De-Simone 2022). For example, ELISA-based detection of *L. infantum* has been instrumental in diagnosing visceral leishmaniasis in endemic areas, with a sensitivity exceeding 95% in human and canine infections (Maia et al. 2007). Nucleic acid-based methods also play a crucial role in pathogen detection and genomic analysis. One of the most widely used techniques is the Polymerase Chain Reaction (PCR), which amplifies specific DNA regions to facilitate pathogen identification (Kent 2009). A more advanced version of this method is Real-Time PCR (qPCR), which amplifies and simultaneously quantifies DNA through fluorescent signal detection, providing both sensitivity and specificity in molecular diagnostics (Hernández and Ramírez 2013). qPCR has been widely employed for detecting DENV in mosquitoes, significantly reducing the time required for outbreak surveillance in tropical regions (Uppoor et al. 2024).

In addition to PCR-based techniques, sequencing methods have revolutionised genetic analysis: Sanger sequencing, a traditional approach, is particularly suitable for targeted DNA studies where specific genetic regions need to be analysed with high accuracy. In contrast, Next-Generation Sequencing (NGS) offers a more comprehensive approach by enabling simultaneous genomic characterisation, detection of chromosomal rearrangements and identification of genetic variations. This high-throughput technology has significantly improved our understanding of pathogen biology, providing deeper insights into their evolution, drug resistance and transmission patterns (Nafea et al. 2024). For example, whole-genome sequencing has allowed the differentiation of ZIKV strains and their geographic origins, supporting epidemiological tracking during outbreaks in the Americas (Faria et al. 2017). Finally, omics-based approaches are also an evolving field in pathogen identification and functional analysis, integrating data from genomics, transcriptomics and proteomics. These approaches provide a comprehensive understanding of pathogen biology, host–pathogen interactions and the molecular mechanisms underlying infections. For example, transcriptomic analysis of *A. gambiae*, the primary malaria vector, has identified key genetic markers associated with insecticide resistance, aiding vector control strategies (Neafsey et al. 2015). In general, genomics enables the identification of genetic variations and evolutionary traits, while transcriptomics reveals gene expression patterns under different conditions, and proteomics characterises protein profiles, offering crucial insights into pathogen virulence factors and host immune responses. Additionally, metagenomic sequencing, for example, has been successfully applied to *Xenopsylla cheopis*, the flea vector of *Yersinia pestis*, revealing the bacterial load and potential co-infections that influence plague transmission. Ongoing advancements in high-throughput sequencing technologies and bioinformatics tools are driving the growth of omics-based methodologies, improving diagnostic accuracy, disease surveillance, outbreak prediction and personalised medicine in the management of infectious diseases and paving the way for new therapeutic and epidemiological strategies (Jean Beltran et al. 2017).

7 | Knowledge Gaps and Future Perspectives

Building on the current knowledge summarised in this review, it is important to highlight several critical research gaps that must be addressed to advance the understanding and control of VBDs. Despite substantial progress in understanding VBDs, major gaps remain in predicting how environmental change will reshape vector ecology and transmission dynamics. Current models often fail to capture the complex interactions among climate change, urbanisation, land-use modification and host–vector behaviour. Long-term, interdisciplinary field studies integrating entomology, ecology, climatology and epidemiology are needed to improve predictive frameworks. In particular, future research should prioritise high-resolution surveillance systems and standardised ecological indicators capable of linking environmental variability with changes in vector competence and disease risk. These environmental uncertainties are closely linked to how vector control strategies are designed and implemented in real-world settings. Another important gap concerns the operational integration of IVM strategies. Although IVM is widely promoted as a comprehensive framework, empirical evidence comparing the long-term effectiveness of different combinations of biological, environmental and chemical interventions remains limited. Future studies should focus on controlled, large-scale implementation trials and cost-effectiveness analyses to identify optimal intervention packages for diverse ecological and socio-economic settings. Standardised evaluation metrics are also needed to facilitate comparisons across regions and programs. In parallel with improvements in conventional control strategies, rapid advances in biotechnology are opening new frontiers in vector management. Emerging biological technologies, including symbiont-based strategies and genetic modification of vectors, present promising opportunities but also raise unresolved questions regarding ecological safety, evolutionary stability and long-term effectiveness. There is a need for longitudinal studies assessing the persistence of introduced traits in natural populations and their potential unintended ecological consequences. Research should also address ethical, regulatory and social acceptance dimensions, which are critical for the successful deployment of novel vector control technologies. However, the effectiveness of both traditional and emerging interventions ultimately depends on robust surveillance systems capable of detecting and responding to changing transmission patterns. Significant gaps persist in the integration of human, animal and environmental health surveillance within a One Health framework. Fragmented monitoring systems often limit the early detection of emerging vector-borne threats. Future research should explore interoperable surveillance platforms, real-time data sharing and the incorporation of genomic and digital epidemiology tools to strengthen early warning systems and coordinated responses. Beyond technical and surveillance challenges, human behaviour and social dynamics play a decisive role in determining the success of vector control efforts. Finally, the social and behavioural determinants of vector control effectiveness remain underexplored. Community engagement, risk perception and behavioural adaptation strongly influence the success of intervention programs, yet these factors are rarely integrated into entomological research. Interdisciplinary studies combining social sciences with vector biology are essential to design culturally appropriate and sustainable control strategies. Addressing these interconnected research gaps will require

sustained interdisciplinary collaboration and will be essential for developing more predictive, adaptive and sustainable strategies to control VBDs in a rapidly changing world.

8 | Conclusions

Vector-borne diseases continue to pose a major global health challenge due to their broad geographical distribution, complex transmission dynamics and the diversity of pathogens involved. These diseases have emerged in previously non-endemic regions as a consequence of increased vector activity driven by climate change, urbanisation, deforestation and greater global mobility. Advancements in molecular biology, genomics and bioinformatics have revolutionised the study of vector competence and vector capacity, providing novel insights into pathogen transmission mechanisms and offering innovative tools for surveillance and early diagnosis. Understanding the multifaceted interactions between vectors, pathogens, hosts and environmental factors is crucial for the development of effective control strategies. IVM—which combines chemical, biological and environmental approaches to reduce vector populations while minimising ecological impact—should be prioritised in preventive interventions. To enhance effectiveness, especially in high-risk areas, these strategies must be reinforced by public health policies, behavioural change initiatives and community education programs. Moreover, the implementation of targeted vaccination campaigns and the development of novel vaccines, where feasible, represent critical components in reducing the burden of VBDs. The advancement of adaptive, evidence-based solutions will rely on technological innovation, strengthened surveillance systems and the promotion of global data sharing. Ultimately, comprehensive strategies that integrate biological, technological and community-based interventions are essential to mitigate the impact of VBDs and improve global health outcomes. Continued research and interdisciplinary collaboration will be the key to advancing our understanding and control of these diseases.

Author Contributions

Patrizia Falabella: conceptualisation and supervision. **Patrizia Falabella, Carmen Scieuzo** and **Roberta Rinaldi:** writing – original draft. **Patrizia Falabella, Carmen Scieuzo, Roberta Rinaldi, Federica De Stefano** and **Luca Masucci:** writing – review and editing. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The authors have nothing to report.

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