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Vector biology and insect-borne disease dynamics: A comprehensive review

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Abstract

Vector-borne diseases are caused by pathogens (parasites, viruses or bacteria) that are transmitted between hosts by insect vectors. These insects (most prominently mosquitoes, sandflies, tsetse flies, triatomine bugs, fleas and lice) ingest the pathogen in a blood meal from an infected host and later transmit it when they bite a new host. This review provides a detailed synthesis of insect vector biology and disease dynamics. It covers the life cycles and ecology of major vectors (for example, *Anopheles* mosquitoes transmit malaria, *Aedes* mosquitoes transmit dengue and Zika, triatomine bugs transmit Chagas disease, etc.), the historical discoveries that revealed their roles (with pioneers such as Ross, Manson and Finlay), and the biological and environmental factors that influence their capacity to spread disease. Vector competence an intrinsic measure of a vector's ability to acquire, support and transmit a pathogen depends on genetic traits (species or strain differences, immune genes and receptor proteins), physiological traits (age, nutrition, microbiome, temperature sensitivity) and behavioural traits (host preference, biting frequency, activity times). Together these determine the basic reproduction number (R_0) of vector-borne diseases. For example, certain *Aedes* populations may be genetically more susceptible to dengue virus, or a warmer environment may shorten the incubation period in mosquitoes, increasing transmission. Preventing and controlling vector-borne diseases requires an array of surveillance and intervention tools. Traditional control measures include insecticide-treated bed nets, indoor spraying, larval habitat management and chemical larvicides. Newer strategies involve biological controls (e.g. releasing *Wolbachia*-infected or sterile insects) and environmental management. Modelling approaches are critical for understanding transmission and planning control. Classic compartmental models (RossMacdonald, SIR/SEIR) yield analytical measures like R_0 , while stochastic, agent-based and network models capture heterogeneity and randomness. Climate-driven and time-series models forecast seasonal risk using weather or historical data. This review adopts a global perspective (with no one-country focus) and assumes broadly that insect vector principles apply worldwide. We have concentrated on insects of medical importance and have not covered tick-borne or plant diseases. We also note that this is a narrative synthesis (with four summary tables) written in an academic style without in-text citations. Assumptions and data gaps are noted in context. The aim is to give a comprehensive, cohesive overview of vector biology and disease dynamics.

Keywords: Pathogen, host, insect vectors, immune gene, biological control, disease

Introduction

Vector-borne diseases arise when pathogens are transmitted between hosts by blood-feeding insects (vectors). In a typical biological transmission cycle, an infected human (or animal) carries a pathogen in the blood that is picked up by a feeding insect [47, 7]. The pathogen then must survive and often replicate within the vector's body (over an "extrinsic incubation period") before the vector can transmit it to another host during a subsequent bite. This interaction involves three components: the pathogen, the vector (insect) and the vertebrate host, with environmental and social factors influencing each [65, 48]. These diseases cause a large global burden of illness and death. For example, malaria alone causes hundreds of millions of cases annually, and dengue virus infects tens of millions [15]. Other important examples include Chagas disease, leishmaniasis, yellow fever and lymphatic filariasis. The distribution of these diseases is determined by the ecology of the vectors (which often thrive in tropical and

subtropical climates) as well as by human factors such as urbanisation and travel [25, 40]. Climate variables (temperature, rainfall) influence vector breeding and survival, which can expand or contract transmission seasons. Key epidemiological metrics in vector-borne disease include vector competence (the intrinsic ability of a given vector population to become infected by and transmit a pathogen) and vectorial capacity (a broader quantity that includes competence, vector density, biting rate and longevity). Together, these determine the basic reproduction number R_0 (the expected number of secondary cases from one primary case) [72, 41]. For instance, the classic RossMacdonald model relates R_0 for malaria to parameters such as the mosquito biting rate, the probability of parasite transmission per bite, the mosquito mortality rate, and the duration of the mosquito's infectious period. In general, higher vector abundance, biting frequency, or survival increases R_0 , as does shorter extrinsic incubation (allowing vectors to become infectious sooner) [102, 39, 16].

This review covers multiple aspects of insect-vector biology and disease dynamics. We begin with a historical overview of major discoveries and scientists in the field. We then survey major insect vectors and associated diseases (see Table 1) and discuss their lifecycles and control challenges. Next, we examine determinants of vector competence genetic, physiological and behavioural factors and how they are measured in the lab and field (Table 2). Following that, we compare surveillance and control tools/technologies ranging from traps and pesticides to genetic strategies (Table 3). We also discuss modelling approaches for disease transmission (compartmental, agent-based, statistical, etc.) and summarize their data needs, assumptions and typical outputs (Table 4). Throughout, we emphasize an analytical perspective, integrating foundational concepts with current advances.

History and Pioneers of Vector Biology

- The field of vector biology has a long and rich history. In 1877 Sir Patrick Manson demonstrated that filarial worms (*Wuchereria* spp.) could be transmitted by mosquitoes, establishing the concept of insects as carriers of human parasites [8, 100]. In the 1880s and 1890s, Alphonse Laveran identified the malaria parasite in human blood, and researchers including Manson and Giovanni Grassi suspected that *Anopheles* mosquitoes played a role [49]. The definitive proof came in 1897 when Sir Ronald Ross dissected a mosquito (*Anopheles*) that had fed on a malaria patient and found the parasites in its gut. For this discovery of malaria transmission, Ross was later awarded the Nobel Prize [64, 1].
- Similarly, in the 1880s Cuban physician Carlos J. Finlay proposed that *Aedes aegypti* mosquitoes transmitted yellow fever virus [27]. Although his work was not initially accepted, it was validated in 1900 by the US Yellow Fever Commission led by Walter Reed. Reed and his team confirmed that *Aedes* mosquitoes could carry and transmit yellow fever, cementing Finlay's hypothesis. These breakthroughs established the model of insect vector-borne cycles for human pathogens [37, 99].
- In the first half of the 20th century, many other vectors were

identified. Italian and British scientists showed that *Triatoma* bugs transmit *Trypanosoma cruzi* (Chagas disease) in the Americas, and that blackflies (*Simulium*) transmit *Onchocerca volvulus* (river blindness) in Africa. The discovery of DDT's insecticidal properties in 1939 (by Paul Müller) revolutionized control: by the 1950s WHO was launching a Global Malaria Eradication Program based on DDT spraying and bed nets. Although that campaign achieved local successes (in Europe, North America, and parts of Asia), it ultimately failed to eliminate malaria worldwide due to logistical, ecological and resistance issues [63, 50].

- Insecticide resistance became a major challenge by the 1970s. In response, new strategies emerged: biological larvicides (such as *Bacillus thuringiensis israelensis* in the 1980s) and integrated vector management approaches. From the late 20th century onward, genetic and microbial control gained interest [82]. The Sterile Insect Technique (SIT) was trialed for tsetse flies and mosquitoes. In the 21st century, releases of genetically engineered mosquitoes (e.g. carrying lethal genes or gene drives) and *Wolbachia*-infected mosquitoes have shown promise in reducing disease transmission. These modern efforts build on the century of discoveries that laid the foundations of vector biology [81, 98].

Major Insect Vectors and Diseases

1. A variety of insect taxa serve as vectors for human disease. Table 1 compares the major vector groups and the pathogens they transmit, along with their global distribution, salient life-cycle features, and control challenges. Notably, mosquitoes of several genera (*Anopheles*, *Aedes*, *Culex*, *Mansonia*) are responsible for most human parasitic and viral diseases (malaria, dengue, Zika, filariasis, etc.) [38]. Other insect vectors include tsetse flies (*Glossina* spp.) for African trypanosomiasis, sandflies (*Phlebotomus* and *Lutzomyia*) for leishmaniasis, triatomine bugs (*Triatoma* spp.) for Chagas disease, blackflies (*Simulium*) for onchocerciasis, rat fleas (*Xenopsylla*) for plague and typhus, human fleas (*Pulex*) for typhus, and body lice (*Pediculus humanus*) for epidemic typhus and relapsing fever. Biting midges (*Culicoides*) are vectors of animal viruses like bluetongue (and occasionally Oropouche virus in humans), while horse-flies and deer-flies (Tabanidae) mechanically transmit parasitic worms like *Loa loa* [73, 18].
2. These vectors occupy diverse habitats. Mosquitoes typically have aquatic larvae (fresh or polluted water), sandflies breed in soil, and tsetse flies produce larvae that pupate on the ground. Many vector species have close associations with human habitats (e.g. *Aedes aegypti* in urban containers) or with animal hosts (e.g. *Culex* birds-mammal cycle for West Nile virus) [51, 88]. Control is correspondingly diverse: for example, removing breeding sites and bed net use can greatly reduce malaria, but *Aedes* control often requires eliminating small water containers and indoor spraying. Insecticide resistance, adaptive behaviours (like biting times and places), and animal reservoir hosts often complicate control efforts [101, 26].

Table 1: The table highlights common features across vectors. For example, mosquito vectors all breed in standing water (with some species tolerating pollution or attaching to plants), and adult female mosquitoes transmit disease [89, 74, 28, 9, 17, 52, 66, 83].

Vector	Pathogen(s) / Disease(s)	Distribution
<i>Anopheles</i> spp. (mosquito)	<i>Plasmodium</i> spp. (malaria), <i>Wuchereria bancrofti</i> (filaria)	Tropical/subtropical worldwide (Africa, Asia, Americas)
<i>Aedes aegypti</i> (mosquito)	Dengue, Zika, chikungunya, yellow fever viruses, <i>Wuchereria</i> (regionally)	Urban tropics/subtropics globally
<i>Aedes albopictus</i> (mosquito)	Dengue, Zika, chikungunya, West Nile (and others)	Originally SE Asia, now widespread in tropics and temperate zones worldwide
<i>Culex pipiens/quinqüefasciatus</i> (mosquito)	West Nile virus, St. Louis encephalitis, Japanese encephalitis, lymphatic filariasis	Global (temperate and tropical regions)
<i>Mansonia</i> spp. (mosquito)	Lymphatic filariasis (<i>W. bancrofti</i> , <i>B. malayi</i>)	Asia, Africa, Pacific islands
<i>Glossina</i> spp. (tsetse fly)	<i>Trypanosoma brucei</i> (African sleeping sickness)	Sub-Saharan Africa (riverine, savannah)
<i>Phlebotomus</i> spp. (Old World sandflies)	<i>Leishmania</i> spp. (cutaneous/visceral leishmaniasis), Phlebovirus (sandfly fever)	Mediterranean, Middle East, Asia, Africa
<i>Lutzomyia</i> spp. (New World sandflies)	<i>Leishmania</i> spp. (cutaneous, mucocutaneous)	Central/South America (neotropical regions)
<i>Triatoma</i> spp. (kissing bugs)	<i>Trypanosoma cruzi</i> (Chagas disease)	Latin America (southern USA to Argentina)
<i>Simulium</i> spp. (blackfly)	<i>Onchocerca volvulus</i> (river blindness)	Africa, Yemen, parts of Latin America
<i>Xenopsylla cheopis</i> (rat flea)	<i>Yersinia pestis</i> (plague), <i>Rickettsia typhi</i> (murine typhus)	Worldwide (historic plague foci, now Central Asia/Africa)

Determinants of Vector Competence

Vector competence is the intrinsic ability of an insect population to acquire, support development of, and transmit a pathogen [35]. After taking an infectious blood meal, a pathogen must infect the midgut of the insect, escape into the body cavity (hemocoel), and finally invade the salivary glands to be transmitted to a new host [62, 2]. Each of these steps can be hindered by vector barriers (e.g. midgut infection barrier, salivary gland barrier). The overall competence is quantified by lab metrics such as the infection rate (fraction of exposed vectors that become infected), dissemination rate (fraction whose infection spreads beyond the gut) and transmission rate (fraction with virus in saliva) [54]. It is also influenced by the extrinsic incubation period (EIP): vectors must survive long enough for the pathogen to incubate [42, 10]. Many intrinsic and extrinsic factors influence competence.

Genetic factors include the vector species or strain and specific genes that affect susceptibility (e.g. receptors or immune pathways). For instance, different *Aedes aegypti* strains can vary markedly in their ability to transmit dengue virus. Mutations or variations in innate immunity genes (RNA interference, Toll, JAK-STAT pathways) also change viral susceptibility [3, 67].

Physiological factors include the insect's age (older mosquitoes have had more time to complete EIP), nutritional status, presence of endosymbionts, and microbiome composition. For example, mosquitoes infected with *Wolbachia* bacteria often show reduced dengue virus levels, drastically lowering transmission. Similarly, gut bacteria may inhibit or enhance a virus [20, 76].

Environmental factors like temperature are especially important: higher ambient temperatures usually speed up viral replication, shortening the EIP and raising transmission potential [42, 96].

Behavioural factors such as host preference (feeding on humans vs. animals), biting frequency (gonotrophic cycle length) and timing of activity affect how often vectors encounter susceptible hosts. For example, highly anthropophilic vectors like *Aedes aegypti* tend to sustain human epidemics, while zoophilic species do not [55, 30, 75].

The following table (Table 2) lists representative determinants of competence, categorised by type, with examples and methods used to assess them. For each determinant, we note its impact on transmission potential [90, 19, 29, 36, 56, 68, 84, 97, 103].

Determinant (example)	Type	Measurement / Assay	Implication for transmission
Vector species or strain (genetic background)	Genetic	Laboratory infection experiments by species/strain, genomic sequencing	Some species or genetic strains are inherently more susceptible, so their presence can increase disease risk
Innate immune genes (e.g. RNAi or Toll pathway)	Genetic	Gene knockdown/knockout or expression analysis, assay of antiviral activity	Strong antiviral responses reduce competence, mutation or suppression of immunity allows higher pathogen replication
Midgut/salivary gland barrier proteins	Genetic	Dissection and PCR/imaging for virus in tissues	If the virus cannot infect the gut or glands, transmission fails, genetic changes in these tissues can block or permit transmission
<i>Wolbachia</i> or other symbiont infection	Physiological	PCR screening for <i>Wolbachia</i> , compare virus levels in infected vs. uninfected insects	<i>Wolbachia</i> often inhibits virus replication (e.g. dengue in <i>Aedes</i>), sharply lowering transmission by that mosquito population
Gut microbiome composition	Physiological	16S rRNA sequencing, antibiotic treatment of vectors, gnotobiotic studies	Certain gut bacteria (or lack thereof) can impede or enhance pathogens, changes in microbiota can raise or lower vectorial capacity
Insecticide exposure/resistance status	Physiological	Expose mosquitoes to sublethal doses and then infect, test resistant vs susceptible strains	Insecticide resistance can incur fitness costs or affect immunity, surviving mosquitoes may be longer-lived carriers, or conversely more vulnerable
Host feeding preference (anthropophily vs zoophily)	Behavioural	Bloodmeal analysis (host DNA), host choice assays	Vectors that prefer humans sustain human-to-human transmission (raising R_0), while zoophilic vectors may mostly bite animals, diluting human transmission
Biting frequency / gonotrophic cycle	Behavioural	Lab or field measurement of time between blood meals, mark-recapture studies	More frequent feeding increases number of contacts per mosquito, leading to more transmission events (higher effective R_0)
Genetically modified traits (engineered)	Genetic	Release-recapture or cage trials of modified mosquitoes, ecological fitness assays	Engineered resistance genes or lethality can greatly reduce vector competence or population in the wild, if traits spread as intended

Surveillance and Control Tools

Effective management of vector-borne diseases depends on both surveillance of vectors/pathogens and control of vector populations. Table 3 compares key tools and technologies. Classical chemical methods include insecticide-treated nets (ITNs/LLINs) and indoor residual spraying (IRS), which are highly effective against indoor-feeding mosquitoes and have dramatically reduced malaria in many regions [44]. Larval control (by source reduction, chemical larvicides or biological agents like *Bacillus thuringiensis israelensis* (Bti)) targets mosquitoes at the immature stage. Personal protection (repellents, protective clothing) protects individuals. Vaccination (where available, e.g. yellow fever) prevents disease independent of the vector [69, 4]. More recent innovations include biocontrol approaches: for example, releasing sterile male mosquitoes (SIT), *Wolbachia*-infected mosquitoes, or genetically modified mosquitoes (Oxitec's *Aedes*) to suppress or modify wild populations. These methods can be species-specific and sustainable but face cost,

regulatory and public acceptance hurdles [56]. Surveillance tools range from mosquito traps (light traps, BG-sentinel, gravid traps) to ovitraps for container-breeders, which help estimate vector abundance or detect presence of pathogens [21, 31]. Geographic Information Systems (GIS) and remote sensing (satellite data) are used to predict habitat suitability and risk areas [43, 77, 91]. Molecular diagnostics (PCR, ELISA on collected insects) allow early detection of pathogen circulation in vectors. Each tool has trade-offs. Table 3 summarises their scale (local/community vs regional/global), strengths, limitations and cost/feasibility [57]. For example, ITNs are low-cost and community-wide but only protect against night-biting vectors. Larvicides and environmental management are effective but labor-intensive. Genetic strategies promise large impacts but currently have very high research costs and strict regulatory controls. Community engagement and education, while hard to quantify, are critical enablers of all other interventions [11, 95].

Table 3: summarises their scale (local/community vs regional/global), strengths, limitations and cost/feasibility [12, 22, 34, 33, 58, 60, 78, 85, 92, 104].

Tool / Technology	Scale	Strengths	Limitations
Insecticide-treated nets (ITNs/LLINs)	Local / Regional (household)	Proven to reduce malaria transmission, passive protection during sleep	Only effective for night-biting species, less impact on day-biting vectors (e.g. <i>Aedes</i>), insecticide resistance can reduce efficacy
Indoor residual spraying (IRS)	Local / Regional (houses, villages)	Kills or repels indoor-resting mosquitoes, highly effective for malaria vectors	Only works on indoor-resting/biting vectors, labor-intensive (repeating sprays), community acceptance needed, resistance
Larval source management (habitat removal)	Local (community level)	Eliminates breeding sites, effective against container-breeders and ricefield mosquitoes	Extremely labor-intensive, requires mapping and sustained community effort, limited in broad or inaccessible areas
Sterile Insect Technique (SIT)	Area-wide (isolated populations)	Species-specific (only target pest), environmentally friendly (no new insecticide)	High production costs (mass rearing and sterilizing), less effective if wild population is high, requires isolated or controlled area
<i>Wolbachia</i> biocontrol (pathogen interference)	Local / Regional	Self-dispersing symbiont that reduces virus replication, demonstrated reduction of dengue incidence	Effect depends on strain, requires high initial release, long-term ecological effects unclear, not population-suppressing
Mosquito traps (light, BG-Sentinel, gravid traps)	Local / Regional (surveillance)	Monitor vector abundance and species, some (e.g. BG) also reduce <i>Aedes</i> numbers	Limited catch radius, require power or CO ₂ , catch data may not represent all species
Ovitraps (egg traps for container-breeders)	Local (community surveillance)	Inexpensive, effective at detecting <i>Aedes</i> presence, community-friendly	Only indicate egg-laying activity, not adult count, require many traps for large areas
GIS / Remote sensing	Regional / Global	Identifies potential breeding habitats and risk zones, integrates large-scale data	Requires technical expertise, depends on data quality, indicates risk indirectly

Modelling Disease Transmission Dynamics

Mathematical and computational models are essential for understanding and predicting vector-borne disease spread and control. Classic deterministic compartmental models (such as RossMacdonald or SEIR/SEI) divide the host and vector populations into compartments (Susceptible, Exposed, Infectious, etc.) and use differential equations to simulate transmission over time [79]. These models yield quantities like the basic reproduction number R_0 , threshold criteria for control, and projected epidemic curves [5, 93]. More complex approaches include stochastic simulations, which capture random events and chance fade-out, and agent-based models where individual humans and vectors interact in space and time. Network models focus on specific contact patterns (for example, travel or social networks) [13, 70]. Climate-driven models incorporate weather inputs to forecast seasonal risk. Statistical time-series models (ARIMA, machine learning) use past incidence data (with or

without climate covariates) to predict short-term trends. Each modelling approach has specific data requirements, core assumptions and typical outputs. In contrast, an agent-based model requires detailed demographic and movement data, assumes explicit individual behaviour, and outputs spatial case maps [23]. Bayesian and data-assimilation models combine multiple data sources and update forecasts with real-time data. Phylogenetic (phylodynamic) models use pathogen genomic data and assume a molecular clock to reconstruct transmission history (outputting estimates of R or origin of an outbreak) [86, 45]. Ecological niche models use occurrence data and environmental layers to predict where vectors might exist, assuming species-environment equilibrium. In practice, models are chosen based on the questions and data available (e.g. predicting next month's dengue cases in a city vs. evaluating long-term elimination strategies for malaria) [32, 59].

Table 4: provides an overview. For example, a simple RossMacdonald model requires parameters like mosquito biting rate, human infectious period and vector lifespan, it assumes homogeneous mixing and constant parameters, and outputs an R_0 estimate [24, 6, 14, 46, 71, 80, 87, 94, 105]

Modelling Approach	Data Requirements	Assumptions	Typical Outputs	Example Applications
RossMacdonald compartmental model (deterministic)	Vector density, human population, biting rate, infection probability, EIP, lifespan	Homogeneous mixing, constant parameters, no spatial or age structure	Basic reproduction number (R_0), transmission threshold, equilibrium prevalence levels	Estimating malaria R_0 and required coverage of bed nets or spraying to interrupt transmission
SEIR/SEI compartmental models	Case incidence, exposure/incubation periods, recovery/death rates	Mass-action mixing, fixed parameter values over time	Epidemic curves (incidence vs time), peak timing and magnitude, effect of interventions	Modeling dengue outbreak dynamics and evaluating impact of reducing vector lifespan
Climate-driven models	Weather/climate data (temperature, rainfall, humidity), vector and parasite biology parameters	Vector life-cycle parameters (survival, EIP) depend on climate, stationarity of relationships	Seasonal risk forecasts, maps of outbreak risk under different climate scenarios	Forecasting seasonal peaks of malaria or dengue in response to El Niño events
Time-series forecasting (ARIMA, machine learning)	Historical disease incidence (possibly plus climate or environmental indices)	Future incidence statistically depends on past patterns (stationarity or learned features)	Short-term case forecasts (next weeks or months), confidence intervals of predictions	Predicting the upcoming dengue season cases in a city for early warning of public health response
Phylogenetic / phylodynamic models	Pathogen genomic sequences with sampling dates and locations	Molecular clock evolution, transmission events coalescence model	Reconstruction of transmission tree, estimates of transmission rate (R_t or R_0), origin of outbreaks	Tracing the introduction and spread of Zika virus in the Americas using viral genome sequences

Conclusion

Insect vectors underpin many of the most significant infectious diseases globally. Understanding vector biology and disease dynamics is essential for control. Advances in genomics, microbiology, ecology and data science have greatly improved our knowledge of what makes some vectors efficient transmitters. Integrated control strategies combining old tools (nets, insecticides, source reduction) with new biotechnologies (genetic modification, symbionts) and enhanced surveillance are being implemented and iterated in many settings. Mathematical models continue to play a vital role by predicting transmission under different scenarios (including climate change and urbanization) and evaluating interventions before large-scale rollout. This review has taken a broad, global perspective (not focusing on a single country) and has centered on insect vectors of human diseases. Accordingly, we have implicitly assumed that key principles apply across regions (for example, the ecology of *Aedes aegypti* in Asia is analogous to that in the Americas). We have not covered non-insect vectors (ticks, mites, etc.) or plant/animal diseases, which are important fields in their own right. Our synthesis is based on the current scientific literature and fundamental concepts rather than new empirical data. Future challenges include managing insecticide resistance, adapting to changing vector ranges under climate warming, and understanding emerging vectors (such as *Aedes albopictus* establishing in temperate zones). Important research frontiers are developing reliable gene-drive systems for vector control, harnessing symbionts or microbiomes, and integrating human behavioral data (e.g. travel patterns) into transmission models. A One-Health approach recognizing the links between human health, animal reservoirs and the environment will be essential for controlling diseases like zoonotic flaviviruses or filarial worms. Finally, sustained community engagement and investment in surveillance infrastructure are critical, since the most effective control relies on local participation and real-time data. In sum, vector biology remains a dynamic field requiring continuous innovation to keep pace with evolving public health threats.

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