Vector Hazard Report: Kenya

Notes on the arthropod-borne disease threats, vector species ecology profiles and recommendations for surveillance and control of arthropod vectors in Kenya.
Preface

This document provides summarized information on the vectors and vector-borne diseases reported from Kenya as of January 2021. Information related to the identification, distribution, medical importance, control and surveillance of vector species are included. For updated information on the current hazards known from Kenya, please use the near-real time hazard assessment links on page 3. Each page of this document is also hyperlinked via the table of contents to allow easy navigation and access to information most relevant to the reader. This report contains background information including a country overview, climate summary and host demographics. View the Vector Hazard Report Quick Guide pages for real-time threat assessment resources, quick navigation to vector-borne disease threats in Kenya and resources for vector identification and monitoring insecticide resistance. Detailed bionomics data for each vector species is available on the vector species ecology profile pages for mosquitoes and ticks.

The target audience for this document are commanders, medical planners, preventive medicine personnel, and particularly medical entomologists.

For each vector-borne disease threat included in the report the following information is provided:

- **Disease Background**
- **Military Impact and Historical Perspective**
- **Transmission Cycle**
- **Additional Resources**

For each vector species threat included in this report, the following information is provided:

- **Current Taxonomy**
- **Bionomics**
- **Medical Importance**
- **Identification Tools**
- **Surveillance and Control Strategies**
- **Additional Resources**

This product was co-produced by the Walter Reed Army Institute of Research, Walter Reed Biosystematics Unit (WRAIR-WRBU) and the Armed Forces Pest Management Board (AFPMB)
Vector Hazard Report Quick Guide: Kenya

### Real-Time Threat Assessment Resources

Visit these websites for regularly updated information about current vector-borne disease threats and regional climate.

<table>
<thead>
<tr>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.S. Dept. of State Travel Alerts</td>
</tr>
<tr>
<td>Health.mil Reports</td>
</tr>
<tr>
<td>CDC Current Outbreaks List</td>
</tr>
<tr>
<td>WHO Outbreak News</td>
</tr>
<tr>
<td>HealthMap Outbreaks</td>
</tr>
<tr>
<td>AccuWeather Current Radar</td>
</tr>
</tbody>
</table>

### Additional Resources

<table>
<thead>
<tr>
<th>Resource</th>
</tr>
</thead>
<tbody>
<tr>
<td>WHO Country Profile: Kenya</td>
</tr>
<tr>
<td>CDC Travelers Guide: Kenya</td>
</tr>
<tr>
<td>CIA Factbook: Kenya</td>
</tr>
</tbody>
</table>

### Militarily Important Vector-Borne Diseases with Short Incubation Periods (<15 days)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Vector</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria</td>
<td>Mosquito</td>
</tr>
<tr>
<td>Dengue Fever/ Yellow Fever/Chikungunya</td>
<td>Mosquito</td>
</tr>
<tr>
<td>West Nile Fever</td>
<td>Mosquito</td>
</tr>
<tr>
<td>Rift Valley Fever</td>
<td>Mosquito</td>
</tr>
<tr>
<td>Sindbis Virus</td>
<td>Mosquito</td>
</tr>
<tr>
<td>O’nyong-nyong Virus</td>
<td>Mosquito</td>
</tr>
<tr>
<td>Sand Fly Fever</td>
<td>Sand Fly</td>
</tr>
<tr>
<td>Trypanosomiasis</td>
<td>Tsetse Fly</td>
</tr>
<tr>
<td>Relapsing Fever (Louse-borne)</td>
<td>Louse</td>
</tr>
<tr>
<td>Relapsing Fever (Tick-borne)</td>
<td>Tick</td>
</tr>
<tr>
<td>Crimean-Congo Hemorrhagic Fever</td>
<td>Tick</td>
</tr>
<tr>
<td>Boutonneuse Fever</td>
<td>Tick</td>
</tr>
<tr>
<td>Q-Fever</td>
<td>Tick</td>
</tr>
<tr>
<td>Murine Typhus</td>
<td>Flea</td>
</tr>
<tr>
<td>Plague</td>
<td>Flea</td>
</tr>
</tbody>
</table>

### Militarily Important Vector-borne Diseases with Long Incubation Periods (>15 days)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Vector</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leishmaniasis</td>
<td>Sand Fly</td>
</tr>
<tr>
<td>Onchoerciasis</td>
<td>Black Fly</td>
</tr>
<tr>
<td>Bancroftian filariasis</td>
<td>Mosquito</td>
</tr>
</tbody>
</table>
## Vector Identification Resources

<table>
<thead>
<tr>
<th>Vector</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mosquito</td>
<td>WRBU Pictorial Key to the Medically Important Mosquitoes of AFRO-COM</td>
</tr>
<tr>
<td></td>
<td>Edwards, F.W. (1941). Mosquitoes of the Ethiopian Region III—Culicine</td>
</tr>
<tr>
<td></td>
<td>Adults and Pupae, Order of Trustees, British Museum of Natural History</td>
</tr>
<tr>
<td>Mosquito, <em>Anopheles</em></td>
<td>WRBU <em>Anopheles</em> Afrotropical Region Adult</td>
</tr>
<tr>
<td></td>
<td>WRBU <em>Anopheles</em> Afrotropical Region Larva</td>
</tr>
<tr>
<td></td>
<td>Coetzee, M. (2020). Key to the females of Afrotropical Anopheles mos-</td>
</tr>
<tr>
<td>Mosquito, <em>Aedes</em></td>
<td>WRBU <em>Aedes</em> Afrotropical Region Adult</td>
</tr>
<tr>
<td></td>
<td>WRBU <em>Aedes</em> Afrotropical Region Larva</td>
</tr>
<tr>
<td></td>
<td>(Diptera: Culicidae) Associated With Dengue Virus Transmission.</td>
</tr>
<tr>
<td></td>
<td><em>Zootaxa</em>, 589: 1–60</td>
</tr>
<tr>
<td>Mosquito, <em>Culex</em></td>
<td>WRBU <em>Culex</em> Subgenera Afrotropical Adult</td>
</tr>
<tr>
<td></td>
<td>WRBU <em>Culex</em> Subgenera Afrotropical Larva</td>
</tr>
<tr>
<td></td>
<td>WRBU <em>Culex</em> (Cux.) East Africa Adult</td>
</tr>
<tr>
<td></td>
<td>WRBU <em>Culex</em> (Cux.) West Africa Adult</td>
</tr>
<tr>
<td></td>
<td>WRBU <em>Culex</em> (Ocu.) Afrotropical Adult</td>
</tr>
<tr>
<td></td>
<td>Harbach, R.E. 1985. Pictorial keys to the genera of mosquitoes, subgenera</td>
</tr>
<tr>
<td></td>
<td>of <em>Culex</em> and the species of <em>Culex (Culex)</em> occurring in southwest</td>
</tr>
<tr>
<td></td>
<td>Asia and Egypt, with a note on the subgeneric placement of *Culex des-</td>
</tr>
</tbody>
</table>
# Vector Hazard Report Quick Guide: Kenya

## Vector Identification Resources

<table>
<thead>
<tr>
<th>Vector</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sand Fly</td>
<td>WRBU Pictorial Key to the Medically Important Sand Flies of AFRICOM</td>
</tr>
<tr>
<td>Lice</td>
<td>University of Florida, Entomology and Nematology, Featured Creatures: Body Louse</td>
</tr>
<tr>
<td>Flea</td>
<td>CDC Pictorial Keys to Arthropods, Reptiles, Birds, and Mammals of Public Health Significance: Fleas</td>
</tr>
</tbody>
</table>

## Insecticide Resistance Resources

- *Aedes* sp. Insecticide Resistance (IR Mapper)
- *Anopheles* sp. Insecticide Resistance (IR Mapper)
- Test procedures for insecticide resistance monitoring in malaria vector mosquitoes (WHO)
- Bottle Assay for Insecticide Resistance (CDC)
# Table of Contents

## I. Vector-borne Disease Hazards

### Mosquito:
- Malaria
- Dengue Fever Virus
- Yellow Fever Virus
- Chikungunya Virus
- West Nile Virus
- Sindbis Virus
- Rift Valley Fever Virus
- Bancroftian filariasis
- O’nyong-nyong Virus

### Sand Fly:
- Sand Fly Fever
- Leishmaniasis

### Tick:
- Crimean-Congo Hemorrhagic Fever
- Relapsing Fever
- Boutonneuse Fever
- Q fever

### Flea:
- Plague
- Murine typhus

### Black Fly:
- Onchocerciasis

### Tsetse Fly:
- Trypanosomiasis

### Lice:
- Relapsing Fever

## II. Vector Species Profiles

### Mosquito:
- *Aedes cumminsii*
- *Aedes ochraceus*
- *Aedes vexans*
- *Aedes furcifer*
- *Aedes taylori*
- *Aedes mcintoshi*
- *Aedes caspius*
- *Aedes aegypti*
- *Aedes africanus*
- *Aedes bromeliae*
- *Anopheles arabiensis*
- *Anopheles funestus*
- *Anopheles gambiae s.l.*
- *Anopheles merus*
- *Anopheles moucheti*
- *Anopheles nili s.l.*
- *Anopheles pharoensis*
- *Culex pipiens*
- *Culex theleri*
- *Culex univittatus*
- *Culex bitaeniorhynchus*
- *Culex quinquefasciatus*
- *Mansonia uniformis*

### Sand Fly:
- *Phlebotomus guggisbergi*
- *Phlebotomus orientalis*
- *Phlebotomus pedifer*
- *Phlebotomus duboscqi*

### Tick:
- *Amblyomma variegatum*
- *Haemaphysalis leachi*
- *Hyalomma dromedarii*
- *Hyalomma impeltatum*
- *Hyalomma rufipes*
- *Hyalomma truncatum*
- *Rhipicephalus pulchellus*
- *Rhipicephalus sanguineus*

### Flea:
- *Xenopsylla cheopis*

### Black Fly:
- *Simulium damnosum s.l.*

### Tsetse Fly:
- *Glossina pallidipes*

### Lice:
- *Pediculus humanus*

## III. Figures and Additional Resources

### Country Profile: Kenya
- Personal Protective Measures

### Figures:
- Topographic Map of Kenya
- Hydrology of Kenya
- Month of Maximum Precipitation
- Month of Maximum Temperature
- Human Population/ sq km
- Entomological Inoculation Rate *P. falciparum*
- Malaria Risk to US Forces
- Number of Infectious Days: *Plasmodium falciparum*
- Temperature Suitability: *Plasmodium falciparum*
- Number of Infectious Days: *Plasmodium vivax*
- Temperature Suitability: *Plasmodium vivax*
- Dengue Fever Prediction Model
- Yellow Fever Risk to US Forces
- Dengue and Chikungunya Risk to US Forces
- Countries Reporting West Nile Virus Infection
- West Nile Risk to US Forces
- Rift Valley Fever Risk to US Forces
- Status of Endemicity of Cutaneous Leishmaniasis
- Status of Endemicity of Visceral Leishmaniasis
- Cutaneous Leishmaniasis Presence Data
- Cutaneous Leishmaniasis Estimated Maximum Number of Cases
- Visceral Leishmaniasis Presence Data
- Visceral Leishmaniasis Estimated Maximum Number of Cases
- Reported Trypanosomiasis Cases East Africa
- Reported Plague Cases by Country
- Distribution of Onchocerciasis, Worldwide
- Bionomics Table: Mosquito Vectors of Kenya
- Bionomics Table: Tick Vectors of Kenya
- Checklist of Mosquito Transmitted Arboviruses and Parasites
- References
Maps of Kenya


Hydrological Map of Kenya (Oiro, 2018).
Climate: The Kenyan climate varies throughout its different geographic regions. (1) The Lake Victoria basin has an annual precipitation varying from 1 m around the shores of Lake Victoria to 1.8 m in the eastern areas. Maximum temperatures range from 27°C in July to 32°C in October and February. (2) The Rift Valley has average temperatures of 29°C in the north to 16°C in the southern areas surrounding Lakes Nakuru and Naivasha. The highlands have average temperatures that stay between 13°C and 18°C. The Rift Valley floor has little rain, however, the highlands see more than 760 mm of rain per year. (3) Annual precipitation in the eastern plateau region on average is between 500 mm and 760 mm. (4) The semiarid/arid regions have average temperatures around 29°C and higher, but the average annual precipitation stays around 250 mm in the north and fewer than 500 mm in the south. (5) Along the coastal plain, temperatures can be higher than 27°C. Humidity is relatively high year-round. Annual precipitation averages out between 760 and 1,270 mm along the coast and decreases inland to around 500 mm per year.

Population and Culture: The majority of Kenya’s population is rural and their location/concentration largely depends on soil and climatic conditions. Areas of high population density include the shores of Lake Victoria, the capital of Nairobi (2.75 million), and along the Indian Ocean in the southeast where the city of Mombasa has a population of 800,000. There are numerous ethnic groups that make up the population of Kenya: Kikuyu (17.2%), Luhya (13.8%), Kalejin (12.9%), Luo (10.5%), Kamba (10.1%), Somali (6.2%), other (29.3%). The total population is estimated at 48,397,527 (July, 2018); 27% urbanized; 78% literacy rate. The various religions consist of 83% Christian, 11.2% Muslim, 1.7% Traditionalist, 1.6% other, 2.4% none, and 0.2% unspecified. 40% of the population is between the ages of 0–14.

Official Languages: English and Kiswahili
Country Profile: Kenya, cont.

**Water, Living and Sanitary Conditions:** The country has seen drastic reductions in mortality rates since its independence (especially for infants) due to improvements in housing, education, sanitation, nutrition and health care programs. Kenya has maintained high rates of gastroenteritis, diarrhea, trachoma, schistosomiasis, and amebiasis, however, especially in the countryside. Malaria, dengue fever, and Rift Valley fever are the major vector borne diseases in Kenya. HIV/AIDS is a major disease which looms as a threat to reverse the positive trends in mortality rates as 1.5 million citizens are living with HIV/AIDS (2017 est.). An inadequate supply of drugs coupled with high drug prices makes it difficult for all those who need care to receive it. The percentage of the population with access to water is only 63.2% and is lower in the rural areas (56.8% rural, 81.6% urban). Only 30.1% of the total population has access to improved sanitation facilities including sewage systems.

**Civil Unrest/Conflict:** The terrorist group al-Shabaab has consistently held an operational and recruitment presence along the Coast and the Somali border in attempts to establish an Islamic rule. al-Shabaab claimed responsibility for an attack as recently as June, 2019, where 8 police officers were killed in Wajir County when their vehicle struck an IED with many more injured. There was another attack in a Nairobi hotel complex where militants armed with guns and explosives killed at least 21 people and injured dozens more. 67 people were killed in 2013 in Nairobi at an upscale shopping mall, and nearly 150 more were killed in an attack on Garissa University College in 2015. Kenyan and foreign government facilities as well as military installations have been targets of attacks. Because of these events and the overall presence of al-Shabaab in Kenya, the US State Department has designated all counties along the porous Somalian border as well as a few Coastal counties (Tana River, Lamu, and Kilifi north of Malindi) as Level 4: Do Not Travel areas while some areas of Nairobi are designated as Level 3: Reconsider Travel as of April, 2019. Kenya is home to nearly 500,000 refugees from South Sudan, Somalia, and other neighboring countries. Almost half of them are housed in refugee complexes in Dadaab making it one of the largest refugee complexes in the world. Established by the UN refugee agency (UNHCR), the Kenyan government has ordered its closure in 2019, which could create crises throughout the area.

[CIA World Factbook: Kenya](#)
[Britannica: Kenya](#)
[OSAC Crime & Safety Report 2019](#)
[State Department Kenya Travel Advisory](#)
[CRS Report US-Kenya Relations](#)
[UNHCR: Dadaab Refugee Complex](#)
[Humanitarian Exchange](#)
Climate of Kenya
Host Demographics: Kenya

This product was made utilizing the LandScan 2018. High Resolution global Population Data Set copyrighted by UT-Battelle, LLC, operator of Oak Ridge National Laboratory under Contract No. DE-AC05-00OR22725 with the United States Department of Energy. The United States Government has certain rights in this Data Set. https://landscan.ornl.gov/

Back to table of contents
Vector-borne Disease Hazards
Malaria

I. Disease Background: Human malaria is caused by protozoan species in the genus *Plasmodium* that are transmitted by the bite of an infective female *Anopheles* mosquito. Clinical symptoms of malaria vary with the species. The most serious malaria infection, *Plasmodium falciparum* malaria, can produce life-threatening complications, including renal and hepatic failure, cerebral damage, and coma. Case fatality rates among children and naive adults exceed 10% when not treated. The other human malarials, *P. vivax*, *P. malariae* and *P. ovale* are not life-threatening except in the very young, the very old, or persons in otherwise poor health. Illness is characterized by malaise, fever, shaking chills, headache, and nausea. The periodicity of the fever, occurring daily, every other day, or every third day, is characteristic of the species. Non-fatal cases of malaria are extremely debilitating. Relapses of improperly treated malaria can occur years after the initial infection in all but *P. falciparum* malaria. *Plasmodium malariae* infections may persist for as long as 50 years, with recurrent febrile episodes. Persons who are partially immune or have been taking prophylactic drugs may show an atypical clinical picture. In Kenya, malaria transmission stably occurs in the Nyanza, Western, and majority of the Coast Provinces. Malaria risk is elevated in the highland areas that border endemic zones as well as in the eastern arid areas, however, 100% of the population of Kenya is at risk. Malaria risk is high year round, but risk is significantly higher during and directly after the rainy seasons (March through June and late September through January) (NCMI, 2019). Approximately 80 to 90% of all cases are caused by *P. falciparum*, with *P. vivax*, *P. malariae*, and *P. ovale* making up the remainder.

II. Military Impact and Historical Perspective: Historically, malaria has had an epic impact on civilizations and military operations. During World War I, in the Macedonian campaign, the French army was crippled with 96,000 cases of malaria. Malaria caused five times as many US casualties in the South Pacific as did enemy action. In 1942, during World War II, malaria was the major cause of casualties in General Stilwell’s forces in North Burma. The Middle East was notably malarious during World War II. An annual incidence rate of 65 cases per 1,000 men for the four-year period was recorded. This rate was exceeded only by the incidence of malaria in the China-Burma-India Theater. US forces suffered a total of 273,566 cases of malaria throughout World War II, at a cost of 30,500 combat man-years. In 1952, during the Korean War, the 1st Marine Division suffered up to 40 cases per 1,000 marines. During the Vietnam War, many regiments were rendered ineffective due to the high incidence of malaria, and many US military units experienced up to 100 cases of malaria/1,000 personnel per year. Elements of the 73rd Airborne Brigade had an incidence of 400 cases of malaria/1,000 during 1967 to early 1968. Almost 300 military personnel contracted malaria during Operation Restore Hope in Somalia. Malaria remains a threat to military forces due to widespread drug resistance and disease resurgence in many areas of the world. Command enforcement of chemoprophylactic measures cannot be overemphasized. When Sir William Slim, British Field Marshal in Southeast Asia during World War II, strictly enforced chemoprophylactic compliance by relieving inattentive officers, attack rates of malaria declined dramatically. During the Vietnam War, malaria attack rates dropped rapidly in military personnel when urine tests were introduced to determine if chloroquine and primaquine were being taken. Many prophylactic drugs, such as chloroquine, kill only the erythrocytic stages of malaria and are ineffective against the latent hepatic stage of *Plasmodium* that is responsible for relapses. Therefore, even soldiers who take chloroquine appropriately during deployment can become infected. Individuals who are noncompliant with the prescribed period of terminal prophylaxis are at risk for late relapses upon their return to the US. During the Vietnam War, 70% of returning troops failed to complete their recommended terminal prophylaxis. The majority of cases in military personnel returning from Operation Restore Hope in Somalia resulted from failure to take proper terminal prophylaxis. In 2016, the WHO estimated 3.52 million cases of malaria with 10,780 deaths, however, Kenya reported over 8.3 million cases in the same year. Malaria poses a high risk to U.S. military personnel in Kenya and varies significantly with rainfall in the country (NCMI, 2019).
Malaria

III. Transmission Cycle(s): Humans are the only reservoir of human malaria. Non-human primates are naturally infected by many Plasmodium species that can infect humans, but natural transmission is rare. Female mosquitoes of the genus Anopheles are the exclusive vectors of human malaria. Plasmodium species undergo a complicated development in the mosquito. When a female Anopheles ingests blood containing the sexual stages (gametocytes) of the parasite, male and female gametocytes unite to form a motile ookinete that penetrates the mosquito’s stomach wall and encysts on the outer surface of the midgut. Thousands of sporozoites are eventually released, some of which migrate to the salivary glands. Infective sporozoites are subsequently injected into a human host when the mosquito takes a blood meal. The time between ingestion of gametocytes and liberation of sporozoites, ranging from 8 to 35 days, is dependent on the temperature and the species of Plasmodium. Malaria parasites develop in the mosquito vector most efficiently when ambient air temperatures are between 25 and 30° C. Parasite development is prolonged during cool seasons and at high altitudes, and may exceed the life expectancy of the vector. Once infected, mosquitoes remain infective for life. Vector competence is frequently higher with indigenous strains of malaria. This decreases the likelihood that imported strains from migrants will become established.

IV: Additional Resources:

CDC Malaria Travel Alert

Malaria Atlas Project

President’s Malaria Initiative: Kenya

President’s Malaria Initiative: Malaria Operational Plan


Malaria

Disease Distribution

The spatial distribution of *Plasmodium falciparum* entomological inoculation rate (EIR) in 2010, Kenya. This map displays predicted number of expected bites from infected mosquitoes per person, per year (Malaria Atlas Project).

Back to table of contents
Malaria: Disease Distribution

The number of days per year which could support *P. falciparum* infectious vectors was calculated using a dynamic biological model and spatial time series temperature data. The temperature data used was a continuous time series across an average year (1950–2000) for every approx. 1 km sq. (Malaria Atlas Project)

Temperature suitability for *P. falciparum* transmission was calculated using a dynamic biological model and spatial time series temperature data. The temperature data used was a time series across an average year (1950–2000) for every approx. 1km sq. (Malaria Atlas Project)
Malaria:

Disease Distribution

The number of days per year which could support *P. vivax* infectious vectors was calculated using a dynamic biological model and spatial time series temperature data. The temperature data used was a continuous time series across an average year (1950–2000) for every approx. 1 km sq. (Malaria Atlas Project)

Temperature suitability for *P. vivax* transmission was calculated using a dynamic biological model and spatial time series temperature data. The temperature data used was a time series across an average year (1950–2000) for every approx. 1 km sq. (Malaria Atlas Project)
Dengue, Chikungunya & Yellow Fever Viruses

I. Disease Background: Dengue fever (breakbone fever, dandy fever) is an acute febrile disease characterized by sudden onset, fever for 3 to 5 days, intense headache, and muscle and joint pain. It is commonly called breakbone fever because of the severity of pain. There is virtually no mortality in classical dengue. Recovery is complete, but weakness and depression may last several weeks. Dengue is caused by a flavivirus and includes five distinct serotypes (dengue 1, 2, 3 and 4). Recovery from infection with one serotype provides lifelong immunity from the same serotype but does not protect against other serotypes. Dengue hemorrhagic fever (DHF) and associated dengue shock syndrome (DSS) were first recognized during a 1954 dengue epidemic in Bangkok, Thailand. DHF/DSS have spread throughout Southeast Asia, Indonesia and the southwest Pacific, Latin America and the Caribbean. DHF requires exposure to two serotypes, either sequentially or during a single epidemic involving more than one serotype. DHF is a severe disease that produces high mortality in children. Yellow fever (YFV) is an acute viral haemorrhagic disease belonging to the genus Flavivirus characterized by the “yellow” tone of some patients of severe YFV due to jaundice. This more toxic phase of YFV usually begins within one day of the end of the initial symptoms. Severe YFV affects ~15% of patients and its symptoms include high fever, shock, organ failure, bleeding, etc. Other more mild symptoms include fever, chills, severe headache, body aches, nausea, vomiting and fatigue. However, the majority of those infected with YFV have mild or no symptoms before completely recovering. The first infection a person may contract will often grant immunity to future YFV infections. Yellow fever is endemic to tropical areas of Central and South America and Africa where outbreaks are common. A vaccine exists and is recommended for entry into many countries of these regions including Kenya. There are 3 main types of YFV: Sylvic (jungle), intermediate (savanna) and urban. Chikungunya refers to an infection by the Chikungunya virus (CHIKV). CHIKV is known to be transmitted by only two mosquito species, *Ae. aegypti* and *Ae. albopictus*. The name means “that which bends up” in the native language of southeastern Tanzania, and refers to the symptoms of Chikungunya fever. CHIKV symptoms typically include a sudden high fever and severe joint pain. Headache, back pain, muscle pain, nausea, vomiting, arthritis, rash, and conjunctivitis may also occur. Unlike Dengue, CHIKV is not thought to be fatal.

II. Military Impact and Historical Perspective: Dengue virus was first isolated and characterized in the 1940s, but dengue fever had been clinically identified from the 18th century. Epidemics of dengue are noted for affecting a large proportion of the population in a community or in military forces operating in an endemic area. Outbreaks involving 500,000 to 2 million cases have occurred in many parts of the world. During World War II, at Espiritu Santo in the Pacific, an estimated 25% of US military personnel became ill with dengue, causing a loss of 80,000 human-days. Other campaigns in the Pacific were marked by dengue epemics, and throughout the war the US Army experienced nearly 110,000 cases. Dengue was an important cause of febrile illness among US troops during Operation Restore Hope in Somalia. In recent years dengue, especially DHF, has been expanding throughout the world. Thirty to 50 million cases of dengue are reported annually. Transmission is unlikely in the highlands and other areas above 1800 m. CHIKV typically occurs as sudden, unpredictable and explosive outbreaks in susceptible populations including new groups of people who enter an endemic region and have not encountered the virus, lowering immunity in the region. Outbreaks of CHIKV historically have occurred in Africa and Asia. In 2007, the virus was found to be spreading in northern Italy and in December 2013 was found in the Caribbean. In 2016, an outbreak of CHIKV was reported from Mandera, Kenya. CHIKV has also been detected within populations of refugees fleeing Syria and traveling through Turkey. Yellow fever has been known to the western world since the mid-17th century with dozens of outbreaks having occurred since. U.S. Army physicians, including Walter Reed, first discovered that mosquitoes vectored and transmitted YFV in the year 1900. There is an ongoing outbreak of YFV in Brazil which began in early 2018 and has caused several deaths. Under reporting of the disease is very common with WHO estimating that less than 1% of cases are reported worldwide which makes it difficult to assess the actual number of cases per year worldwide. No official locally acquired cases have been reported to WHO since 1995 in Kenya. The risk of dengue to military personnel in Kenya is high overall, but highest in urban and other densely populated areas and lowest in sparsely populated areas. The risk of YFV in Kenya is intermediate year round with an elevated risk during and immediately after the rainy seasons; March through June and September through January. Chikungunya poses an intermediate risk to U.S. military personnel, however, in areas of high density (similar to dengue) the possibility of unpredictable and explosive transmission increases could lead to outbreaks (NCMI, 2019).
Dengue, Chikungunya & Yellow Fever viruses (DENV, CHIKV, YFV)

III. Transmission Cycles: The transmission of dengue virus is exclusively associated with *Aedes* mosquitoes in the subgenus *Stegomyia*. Although the virus has been detected in pools of other mosquitoes. The virus is maintained in a human/ *Ae. aegypti* cycle in tropical urban areas. A monkey-mosquito cycle serves to maintain DENV and CHIKV in sylvatic situations in Kenya. Mosquitoes are able to transmit dengue virus 8 to 10 days after an infective blood meal and can transmit the virus for life. Yellow Fever has three types with different transmission cycles: (1) Sylvatic (jungle) yellow fever where the disease is passed from monkey to human from infected mosquitoes, and is caused by *Aedes africanus* in Africa. (2) Intermediate (savanna) yellow fever is the most common outbreak inducing type and is caused by semi-domestic mosquitoes in close proximity to both humans and non-human primates. (3) Urban yellow fever is transmitted by *Ae. aegypti* between humans only (no other primates). Chikungunya virus is also spread via *Aedes* mosquitoes, primarily *Ae. aegypti*.

IV. Additional Resources:

- CDC Dengue Fever Background
- CDC Chikungunya Background
- CDC Yellow Fever Background
- WHO Dengue Outbreak Information
- WHO Chikungunya Outbreak Information
- WHO Yellow Fever Outbreak Information


Dengue, Chikungunya & Yellow Fever Viruses

Global dengue virus prediction model.
Bhatt, S. et al. 2013
**Dengue, Chikungunya & Yellow Fever Viruses**

**AFRICOM (Eastern and Horn): Dengue Risk to U.S. Forces**

Note: Diagnosis and reporting of dengue are poor throughout Africa, and data are insufficient to make precise estimates of potential rates in U.S. forces. NCOI assesses dengue accounts for a significant amount of undiagnosed febrile illness which occur throughout the continent. Although the range of potential rates in U.S. forces varies widely, risk is highest in urban and other densely populated areas and lower in sparsely populated areas; risk is limited above 1,000-meter elevation. Risk is generally higher during and just after the rainy season. See individual country IDRA for details on risk period.

Environmentally suitable areas are assessed to be at high risk for dengue transmission depending on seasonal rainfall patterns. Dengue transmission occurs year-round in urban and other densely populated areas, and transmission intensity is highest during and just after the main rainy season. Transmission risk is generally higher in urban and other densely populated areas and lower in sparsely populated areas. Dengue has been identified in humans or mosquitoes at the country level. Areas assessed to be suitable for dengue transmission have significant risk of dengue transmission. Dengue has not been reported, however, vector mosquitoes are present, and the area is assessed to be at risk of transmission. No risk: Environmental conditions are unsuitable for transmission.

**AFRICOM: Chikungunya Risk to U.S. Forces**

Environmentally suitable areas are assessed to be at intermediate risk for chikungunya transmission. During large local or regional outbreaks, operationally significant attack rates of 1-5% per month could occur among personnel exposed to mosquito bites. Outbreaks occur during the rainy season when daily rainfall and mosquito abundance are high. Chikungunya virus is introduced into a susceptible and densely populated area, after which population immunity declines further outbreaks for years. Transmission generally becomes lower and less widespread in areas with lower population density. Chikungunya has been identified at the country level. Areas assessed to be suitable for chikungunya transmission have significant risk of chikungunya transmission. Chikungunya has not been reported, however, vector mosquitoes are present and the area is assessed to be at risk of transmission. No risk: Environmental conditions are unsuitable for transmission.

**Back to table of contents**
West Nile virus (WNV)

I. Disease Background: West Nile fever is a mosquito-borne illness characterized by fever, headache, muscular pain, and rash. Occasionally, serious complications involve the liver and nervous system. The etiological agent, West Nile virus (WNV), is named after the district of Uganda where the virus was first isolated. It is a flavivirus closely related to viruses causing Japanese encephalitis and St. Louis encephalitis. Infection with WNV is most often asymptomatic. The incubation period ranges from 1 to 6 days and clinically resembles a mild dengue-like illness.

II. Military Impact and Historical Perspective: WNV was first isolated in 1937 and was one of the earliest human arboviral infections to be documented. Undoubtedly, WNV has been the cause of many cases classified as fevers of unknown origin in military personnel. In view of the mild illness and the infrequent occurrence of epidemics, the military impact of this illness would be minor, particularly in comparison with other diseases in East Africa. Infection with WNV will complicate diagnoses by medical personnel, since West Nile fever cannot be clinically distinguished from many other arboviral fevers. Epidemics of West Nile fever are infrequent, and continued long-term surveillance for virus activity can rarely be justified when considering other health care demands. Reduction of mosquito populations by ULV spraying may be useful as a means of disease control. The most feasible long-term control strategies involve reducing vector breeding by environmental management techniques. Personal protective measures to prevent mosquito bites are the most practical means of avoiding infection with WNV. The threat of WNV to military personnel is intermediate nationwide (NCMI, 2019).

III. Transmission Cycle(s): WNV has been isolated from numerous wild birds and mammals. Serological surveys have demonstrated WNV antibodies in wild and domestic bird species, wild mammals such as lemurs, rodents and bats, and domestic animals such as camels, horses, mules, donkeys, goats, cattle, water buffalo, sheep, pigs and dogs. However, birds are considered to be the primary reservoir for WNV and may reintroduce the virus during seasonal migrations. Infections in most mammals fail to produce viremias high enough to infect potential vectors. WNV has been isolated from several species of mosquitoes in nature, and they are recognized as the major vectors, especially Culex spp. WNV has also been recovered from bird-feeding ticks and mites. A natural bird-tick zoonotic cycle has been suggested, but the role of ticks in the natural transmission of WNV has not been well defined. Mosquitoes are clearly implicated in the transmission of WNV to humans. WNV replicates quickly in mosquitoes when temperatures exceed 25°C. Infected mosquitoes can transmit WNV for life. Peak transmission takes place during and immediately after the rainy seasons (typically March through June and September through January).

IV. Additional Resources:

CDC West Nile Virus Background


West Nile virus

[Map showing West Nile virus risk in Africa]

A small number of cases (<1% per month) could occur.

No risk

NOTE: Epidemiologic data on West Nile are generally poor throughout Africa. The distribution depicted on this map is based on an assessment of the suitability of conditions for transmission. The attack rate for symptomatic infections with West Nile is not likely to exceed 1 percent per month, even under conditions which are highly favorable for transmission.

UNCLASSIFIED

Source: CDC

Projections: Geographic

Datum: WGS84

UNCLASSIFIED

Boundary representation is not necessarily authoritative

Back to table of contents
Sindbis virus (SINV)

I. Disease Background: Sindbis virus belongs to the genus Alphavirus in the family Togaviridae. It is closely related to the Western equine encephalitis complex. The incubation period is less than a week and symptoms may include fever, headache, rash, and joint pain. Syndromes resulting from Sindbis virus infection have been called Ockelbo disease in Sweden, Pogsta disease in Finland, and Karelian fever in the former Soviet Union. No fatal cases have been reported.

II. Military Impact and Historical Perspective: Sindbis virus was first isolated in 1952 from Culex mosquitoes collected in the village of Sindbis north of Cairo. A role in human disease was recognized in 1961 when Sindbis virus was isolated from patients with fever in Uganda. Sindbis poses an intermediate threat to U.S. personnel primarily in rural areas (NCMI, 2019).

III. Transmission Cycle(s): A wide range of wild and domestic vertebrate species are susceptible to infection with Sindbis virus. Most experimentally infected wild bird species easily produce viremias high enough to infect several different species of mosquitoes. Wild and domestic birds are considered the main enzootic reservoir. Although several species of domestic animals can become infected with Sindbis virus, there is no evidence that these infections result in significant illness. Evidence implicates bird-feeding mosquitoes of the genus Culex as the vectors of Sindbis virus in enzootic and human infections. However, viral isolations and transmission experiments have shown that Aedes spp., which are less host specific and feed readily on both birds and humans, may be important as vectors linking the enzootic cycle with human infection. Mechanisms that allow the virus to overwinter and survive between periods of enzootic transmission have not been identified. The peak transmission period is during and directly after the rainy seasons (March through June and September through January), and varies significantly with this rainfall in highland areas and the more eastern regions (NCMI, 2019).

IV. Additional Resources:

ECDC Sindbis Virus Background


O’nyong-nyong virus (ONNV)

I. Disease Background: O’nyong-nyong virus belongs to the genus Alphavirus in the family Togaviridae and is closely related to Chikungunya virus. The incubation period it typically 3 to 11 days and leads to symptoms of headache, pruritic rash, lymphadenopathy, and conjunctivitis. While there is no targeted treatment, patients are incapacitated for 2 weeks at most and then recover completely—no fatal cases have been reported.

II. Military Impact and Historical Perspective: O’nyong-nyong was first isolated in 1959 from serum samples in the Northern Province of Uganda. The virus has since caused two wide spread epidemics in East Africa from 1959 to 1962 affecting 2 million people and again in 1996 at a smaller scale. Exported cases from Kenya have been seen as recently as 2013 in a 60-year old woman returning to Germany, and cases were reported in 2018 in Mombasa County, Kenya as well. The risk to military personnel is intermediate, and, while not fatal, symptoms of O’nyong-nyong can hospitalize military personnel (NCMI, 2019).

III. Transmission Cycle(s): While a vertebrate host has yet to be identified, cattle and other domestic livestock as well as several species of rodents have been implicated in serological surveys. Uniquely, this virus is spread by mosquitoes of the genus Anopheles in Kenya making it the only known human alphavirus vectored by anophelines. Peak transmission times are during and immediately following the rainy seasons of March through June and September through January with foci centered in highland areas and the more arid eastern regions (NCMI, 2019).

IV. Additional Resources:

Army Public Health Command Fact Sheet

CDC O’nyong-nyong Virus Background


Back to table of contents
Rift Valley Fever virus (RVFV)

I. Disease Background: A Phlebovirus of the family Bunyaviridae causes Rift Valley fever (RVF). Humans infected with RVF typically have either no symptoms or a mild illness associated with fever and liver abnormalities. However, in some patients the illness can progress to hemorrhagic fever with shock or hemorrhage, encephalitis with coma or seizures, and/or ocular disease. Patients who become ill usually experience fever, generalized weakness, back pain, dizziness and weight loss at onset of fever. Typically, patients recover within one week after onset of illness. The most common complication associated with RVF is inflammation of the retina resulting in permanent vision loss in 1 to 10% of affected patients. Approximately 1% of patients die of the disease, but case fatality rates are significantly higher for infected animals. Nearly 100% of pregnant livestock infected with RVFV abort their fetuses. Human outbreaks usually take place following outbreaks in sheep, cattle, or camels. There is no established course of treatment for infected patients, although some antiviral drugs such as ribavirin show promise.

II. Military Impact and Historical Perspective: Veterinary officers in Kenya first reported RVF among livestock in the early 1900s, although the virus wasn’t isolated until 1930. The most notable epizootic occurred in South Africa during 1950 to 1951 and was estimated to have caused the death of 100,000 sheep and cattle and to have involved 20,000 human cases. A major epizootic occurred in Kenya at the same time. In 1977, the virus was detected in Egypt and caused a large outbreak among animals and humans. The first epidemic of RVF in West Africa was reported in 1987 and was linked to the Lower Senegal River Project. The project caused flooding in the lower Senegal River area that produced large populations of mosquitoes. During epizootics, RVF could seriously affect military operations. Five percent of Swedish United Nations Emergency Forces soldiers serving in Egypt and the Sinai peninsula were infected with RVF virus during the 1977–78 epidemic in Egypt. Medical personnel should be aware of clinical and diagnostic procedures to differentiate RVF from other fevers with similar clinical syndromes. Risk of RVFV to military personnel is high overall with risk being highest in rural areas and in areas with close proximity to livestock (NCMI, 2019).

III. Transmission Cycle(s): RVF primarily affects domestic animals such as cattle, buffalo, sheep, goats and camels. High viremias occur in infected humans. Thus, humans, as well as domestic animals, could be a source of virus to infect potential vectors. Unlike most arboviruses that are associated with either a single species or closely related group of mosquitoes, RVFV has been isolated from at least 28 species in six genera of mosquitoes. Epizootics have generally occurred during years of excessive rainfall and localized flooding that produced large populations of mosquitoes. Culex pipiens was implicated as the principal vector during the 1977–78 epidemic in Egypt. Vector competence studies and knowledge of mosquito density and feeding behavior in areas where RVF virus infections have occurred suggest that these species may be the principal vectors involved in domestic animal transmission and as bridge vectors from domestic animals to humans. In contrast, Cx. pipiens appears to be the principal vector for human-to-human transmission. Transovarial transmission of the virus is known to occur in some mosquito species. Humans can also acquire infection if they are exposed to the blood or other body fluids of infected animals. This exposure can result from the slaughtering or handling of infected animals or by touching contaminated meat during the preparation of food. Abattoir workers are a useful sentinel population for surveillance of RVFV. Laboratory infection through aerosol transmission of RVFV has resulted from exposure to specimens containing the virus.

IV. Additional Resources:

CDC Background: Rift Valley Fever Virus


Rift Valley Fever virus
Bancroftian filariasis

I. Disease Background: Bancroftian filariasis is caused by the nematode *Wuchereria bancrofti*, which normally resides in the lymphatic system of infected humans. After 8 to 12 months, adult female worms release thousands of microfilariae into the circulatory system. Females continue to produce microfilariae over the next 15 to 18 years. Many individuals are asymptomatic in the early stages of infection. The disease develops slowly, with recurrent episodes of fever and inflammation of the lymph glands. Microfilariae can obstruct the lymphatic system, causing the legs, breasts or scrotum to swell to grotesque proportions, a chronic condition known as elephantiasis. This occurs only after repeated infections. Death of numerous microfilariae resulting from drug therapy may cause severe immune reactions.

II. Military Impact and Historical Perspective: Microfilariae of *W. bancrofti* were discovered in the blood of a patient in Brazil in 1866. This was the first discovery of a pathogen that is transmitted by insects. Over 70 million people worldwide are estimated to be infected by *W. bancrofti*, resulting in serious economic costs to developing countries. The long incubation period and requirement for multiple infections over a long period of time before the appearance of clinical symptoms render chronic Bancroftian filariasis of little medical significance to military operations. However, military personnel moving into an endemic area from one that is free from filariasis may develop symptoms such as swelling of the lymph glands, headache and fever many months before larvae become mature. American military forces in the Samoan-Ellice-Wallis Islands from 1942 to 1944 rapidly developed swollen lymph glands and extremities following repeated exposure to infected mosquitoes. Acute filariasis is the primary military concern, because its symptoms develop fairly rapidly and may be severe enough to cause removal of troops from their duties. In addition, observing local members of the population with grotesque deformities caused by chronic infection can have an adverse psychological impact. Medical personnel should be aware that troops with brief exposure to infection are often not diagnosed until after they return from deployments.

III. Transmission Cycle(s): Microfilariae circulating in human blood are ingested by mosquitoes and undergo several days of development before the vector can transmit infective stages of the nematode. Infective parasites enter the bloodstream directly during a mosquito bite. A few nematode larvae are deposited on the skin and can enter the host through skin abrasions. In humans, larvae undergo development to adults that produce microfilariae for many years. Over most of its geographic range, *W. bancrofti* microfilariae usually exhibit pronounced nocturnal periodicity and consequently are ingested by night-biting mosquitoes. Peak abundance of microfilariae in the blood occurs between 23:00 and 03:00 hours. *Culex quinquefasciatus* is the most common urban vector. In rural areas, transmission is maintained mainly by *Anopheles gambiae* s.l. and *An. funestus*. There are no known animal reservoirs of Bancroftian filariasis. Seasonal distribution generally coincides with rainy periods in endemic areas.

IV. Additional Resources:

CDC Background Lymphatic filariasis

WHO Background Lymphatic filariasis


Back to table of contents
Sand Fly Fever virus (SFNV, SFSV)

I. Disease Background: Sand fly fever, also known as 3-day fever, is caused by two distinct Phlebovirus serotypes, Naples (SFNV) and Sicilian (SFSV). The virus produces an acute febrile illness lasting 2 to 4 days and is commonly accompanied by headache and muscle pain. There is usually no mortality or significant complications. Most infections are acquired during childhood in endemic areas. The clinical disease in children is generally mild and results in lifelong immunity.

II. Military Impact and Historical Perspective: Sand fly fever has been an important cause of febrile disease during military operations since at least the Napoleonic Wars. In 1909, an Austrian military commission first reported that an agent found in the blood of infected soldiers caused this fever, and that the vector was the sand fly. During World War II, there were 19,000 cases of sand fly fever, with the highest incidence reported in the Middle East Theater. In sharp contrast to World War II, there were no reports of sand fly fever among coalition forces during the Persian Gulf War. The military significance of sand fly fever is magnified because of its short incubation period, which may result in large numbers of nonimmune troops being rendered ineffective early in an operation, while endemic forces would be largely immune and unaffected. The risk of sand fly fever to U.S. personnel in Kenya is intermediate year-round (NCMI, 2019).

III. Transmission Cycle(s): No vertebrate reservoir has been established, but there is some serological evidence that gerbils serve as reservoirs. Infected humans can infect sand flies and thus have an amplifying effect during epidemics. The principal reservoir mechanism appears to be transovarial transmission within the vector. The virus is most efficiently replicated in the sand fly vector and transmitted when temperatures exceed 25° C. Infected sand flies remain infective for life and are not harmed by the virus.

IV. Additional Resources:

Leishmaniasis

I. Disease Background: This potentially disfiguring and sometimes fatal disease is caused by infection with protozoan parasites of the genus Leishmania. Transmission results from bites of infected phlebotomine sand flies. All vectors of leishmaniasis in the Old World are in the sand fly genus Phlebotomus. Incubation in humans may take as little as ten days, or more than six months. Symptoms include ulcerative cutaneous lesions (cutaneous leishmaniasis or CL), lesions in the mucosal areas of the mouth and/or nose (mucocutaneous leishmaniasis or MCL), and internal pathological manifestations resulting in fever, swollen lymph glands, anemia, enlargement of the liver and spleen, and progressive emaciation and weakness (visceral leishmaniasis or VL). In East Africa, both CL and VL are important public health problems. CL (Baghdad boil, Jericho boil, Oriental sore), caused by infection with Leishmania major or L. tropica, typically appears as a nonhealing ulcer. The lesion usually develops within weeks or months after a sand fly bite and slowly evolves from a papule to a nodule to an ulcer. Cutaneous lesions may resolve quickly (2–3 months) without treatment or they may become chronic (lasting months to years) and will seldom heal without treatment. Scarring is associated with healing. In endemic areas, such scars are common among both rural and urban populations. Life-long immunity to the infecting Leishmania species normally results. VL (Kala-azar, Dumd fever), is the most severe form of leishmaniasis, with as much as 95% mortality in untreated cases. It is a chronic disease and, without treatment, is marked by fever (2 daily peaks), weakness and, as the parasites invade internal organs, weight loss coupled with enlargement of spleen and liver that may resemble severe malnutrition. It should be noted that cutaneous lesions may also be seen in human visceral leishmaniasis cases, but the chronic visceralizing nature of the disease is the main concern. In the Old World, VL is usually attributed to L. donovani or L. infantum. Viscero-tropic L. tropica has also been reported and was described in veterans of the Persian Gulf war. The incubation period for VL is usually 4 to 6 months but may be as short as 10 days or as long as two years. By the time the disease is diagnosed, patients have usually forgotten any contact with sand flies. In endemic regions it is a disease of the young and old, who succumb to it disproportionately. Leishmaniasis aethiopica is well known for causing CL in mountainous regions and the highlands while L. major is often the cause of CL in the lowlands. Epidemics of VL often follow conditions of severe drought, famine or disruption of native populations by wars that produce large numbers of refugees.

II. Military Impact and Historical Perspective: Although not a war stopper, leishmaniasis is a persistent health threat to U.S. military personnel because troops deploying or conducting military exercises in locations where the disease is endemic. The overall potential for this disease to compromise mission objectives is significant. Soldiers exposed to sand fly bites while deployed to the region are susceptible to infection with leishmaniasis. Immunity among US military personnel is essentially nonexistent, and recovery from CL does not confer immunity to VL. In the Karum River Valley of Iraq, US forces suffered 630 cases of the disease in a 3-month period during WWII. During the 1967 “Six Day War,” Israeli soldiers camped near Jericho in the Jordan Valley suffered a 50% attack rate of L. major. In the northern Sinai desert, 113 cases of L. major were reported from Multinational Forces and observers from 1973 through 1991. In 1990–91, twenty cases of CL due mainly to L. major and 12 cases of VL due to L. tropica were diagnosed when 697,000 allied soldiers were deployed to the Arabian Peninsula during Operations Desert Shield and Storm. Even though no fatalities were associated with leishmaniasis in this deployment, new lessons were learned that could affect future military deployments. Before the Persian Gulf War, eastern Saudi Arabia was not known to be endemic for visceral leishmaniasis and L. tropica was not convincingly shown to produce visceral disease. More importantly, the potential for leishmaniasis to cause intransient post–deployment diagnostic problems and threaten blood supplies had not been anticipated. Returnees from the Persian Gulf War were barred from donating blood for up to two years, severely impacting blood supplies. Infection with Leishmania was even listed as one of the causative factors of Persian Gulf War syndrome, but scientific evidence for this association is lacking. Diagnosis of leishmaniasis is difficult at best, and providing proper care for service members who may have been exposed or infected is a long, costly and complex process. The risk of VL to military personnel is intermediate with foci being recognized in dry, hot regions below 1500 m such as the Rift valley, Eastern, and central North Eastern Provinces. CL has foci often in areas of higher elevation such as the eastern slopes of Mount Elgon, the Aberdare Range, and the central highlands in Kenya among others. The risk of CL to U.S. personnel is also intermediate (NCMI, 2019).

III. Transmission Cycle(s): L. major is a parasite of colonial desert rodents, especially gerbils such as the fat sand rat, Psammomys obesus. Female Phlebotomus sp. acquire infections while feeding on their rodent hosts. Amastigotes (the mammalian form of the Leishmania parasite) ingested with the bloodmeal transform to a flagellated promastigote form within the gut of the female fly. In addition to a bloodmeal, the female fly seeks and consumes sugar from the plants in the area during subsequent nocturnal flights. These sugars help maintain Leishmania infections in the flies. Promastigotes multiply in the gut of the sand fly within the bloodmeal and undergo development to an infective form called the metacyclic promastigote. By the time the bloodmeal is digested and the fly is ready to lay its eggs, infective metacyclic promastigotes are ready to be transmitted to the next vertebrate host when the sand fly feeds again. In L. major foci, where the principal reservoirs are colonial rodents, humans are considered accidental or incidental hosts, becoming infected when their habitat overlaps that of the rodent host. In urban L. tropica, humans may serve as reservoirs. In rural areas, non-human hosts of L. tropica may include wild and domestic rodents as well as other small mammals such as hyraxes living in close proximity to humans.

IV. Additional Resources:

CDC Background Leishmaniasis

WHO Background Leishmaniasis

WHO Leishmaniasis Resources


Back to table of contents
Leishmaniasis: Disease Distribution

Cutaneous Leishmaniasis Presence
Alvar J. et al. 2012

Back to table of contents
Leishmaniasis Disease Distribution

Cutaneous Leishmaniasis Maximum Incidence
Alvar J. et al. 2012

(c) OpenStreetMap contributors, Creative Commons-Share Alike license (CC-BY-SA)
Leishmaniasis Disease Distribution

Visceral Leishmaniasis Presence
Alvar J. et al. 2012

(c) OpenStreetMap and contributors. Creative Commons-Share Alike License (CC-BY-SA)
Leishmaniasis Disease Distribution

Visceral Leishmaniasis Maximum Incidence
Alvar J. et al. 2012

(c) OpenStreetMap and contributors, Creative Commons-Share Alike license (CC-BY-SA)
Relapsing Fever (Tick-borne)

I. Disease Background: This is a systemic spirochetal disease characterized by periods of fever alternating with afebrile periods. The number of relapses varies from 1 to 10 or more. The severity of illness decreases with each relapse. The duration of tick-borne relapsing fever is usually longer than the closely related louse-borne relapsing fever. A number of species of Borrelia are responsible for the disease. The taxonomy of the pathogen is complex. The close vector-spirochete relationship has led to the definition of most spirochete species by the tick vector. There is great strain variation among tick-borne Borrelia, and a single strain can give rise to many serotypes. Some authorities view all species as tick-adapted strains of the louse-borne relapsing fever spirochete, Borrelia recurrentis.

II. Military Impact and Historical Perspective: Although clinical symptoms of tick-borne relapsing fever can be severe, impact on military personnel would be minimal due to low incidence of the disease (NCMI, 2019).

III. Transmission Cycle(s): Soft ticks of the genus Ornithodoros transmit tick-borne relapsing fever. Infection is transmitted from human to human, animal to animal, or from animal to man by the bite of infective ticks. Rodents are sources of infection for ticks, although ticks are more important as long-term reservoirs. The pathogen has been maintained naturally in some species of ticks for years by transovarial transmission. The rate of transovarial transmission varies greatly among tick species. Ticks of both sexes and all active stages transmit the pathogen by bite or by infectious fluids exuded from pores in the basal leg segments. Spirochetes can pass into bite wounds or penetrate unbroken skin. Exposure to infected blood of patients can cause infections in medical personnel.

IV. Additional Resources:

CDC Background Relapsing Fever

Relapsing Fever (Louse-borne)

I. Disease Background: Louse-borne relapsing fever is caused by the spirochete Borrelia recurrentis. The symptoms and severity of relapsing fever depend on the immune status of the individual, geographic location, and strain of Borrelia. The incubation period in an infected host ranges from 2 to 14 days. The disease is characterized by a primary febrile attack followed by an afebrile interval and one or more subsequent attacks of fever and headache. Intervals between attacks range from 5 to 9 days. In untreated cases, mortality is usually low but can reach 40%. Infection responds well to treatment with antibiotics.

II. Military Impact and Historical Perspective: Major epidemics of louse-borne relapsing fever occurred during World War I and the war’s aftermath in Russia, Central Europe and North Africa. After the war, relapsing fever was disseminated through large areas of Europe, carried by louse-infested soldiers, civilians and prisoners of war. Between 1910 and 1945, there were an estimated 15 million cases and nearly 5 million deaths. Large outbreaks of louse-borne relapsing fever were common during and after World War II. During the Vietnam War, epidemics of louse-borne fever also occurred in the Democratic Peoples’ Republic of Vietnam.

III. Transmission Cycle(s): The body louse, Pediculus humanus Linnaeus, 1758, is the vector of B. recurrentis. After the louse feeds on infective blood, the spirochetes leave the digestive tract and multiply in the insect’s body cavity and other organs. They do not invade the salivary glands or the ovaries. Bites and infective feces cannot transmit the pathogen, and transovarial transmission does not occur. Human infection occurs when a louse is crushed and Borrelia spirochetes are released. The spirochetes may be scratched into the skin, but there is evidence that B. recurrentis can penetrate unbroken skin. Since infection is fatal to the louse, a single louse can infect only one person. However, B. recurrentis can survive for some time in a dead louse. Outbreaks of louse-borne relapsing fever require high populations of body lice. Lice leave febrile patients in search of new hosts, and this behavior contributes to the spread of disease during an epidemic. Endemic foci exist in primarily in Ethiopia but have also been found in Somalia and Sudan. Epidemics usually occur in the cold season, among poor people with inadequate hygiene.

IV. Additional Resources:

ECDC Background Louse-borne Relapsing Fever

Back to table of contents
Boutonneuse Fever

I. Disease Background: Boutonneuse Fever, also known as, Mediterranean tick fever, Mediterranean spotted fever, Marseilles fever, African tick typhus, Kenya tick typhus and India tick typhus. This tick-borne typhus is a mild to severe illness lasting a few days to 2 weeks and caused by *Rickettsia conorii* and closely related organisms. Different strains of *R. conorii* have been isolated from ticks and humans. The common name of this disease comes from the button-like lesions, 2 to 5 mm in diameter, that develop at tick attachment sites. With antibiotic treatment, fever lasts no more than 2 days. The case fatality rate is very low, even without treatment.

II. Military Impact and Historical Perspective: Boutonneuse fever has not significantly interfered with military operations in the past. Sporadic cases among combat troops can be expected in limited geographic areas. The severity of illness depends on the strain of *R. conorii* contracted. Because the spotted fevers are regional diseases, military medical personnel newly assigned to an area may be unfamiliar with them and diagnosis may be delayed.

III. Transmission Cycle(s): The disease is maintained in nature by transovarial passage of the pathogen in ticks, primarily the brown dog tick, *Rhipicephalus sanguineus*, although almost any *Ixodes* sp. may harbor the pathogen. Enzootic infection in dogs, rodents and other animals is usually subclinical. Transmission to humans is by bite of infected ticks. Contamination of breaks in the skin or mucous membranes with crushed tissues or feces of infected ticks can also lead to infection.

IV. Additional Resources:

CDC Background Rickettsial (Spotted & Typhus Fevers) & Related Infections


Query Fever (Q fever)

I. Disease Background: This is an acute, self-limiting, febrile rickettsial disease caused by *Coxiella burnetii*. Onset may be sudden with chills, headache and weakness. Pneumonia is the most serious complication. There is considerable variation in severity and duration of illness. Infections may be unapparent or present as a nonspecific fever of unknown origin. The case fatality rate in untreated acute cases is less than 1%.

II. Military Impact and Historical Perspective: *Coxiella burnetii* was originally described from Australia in 1937. In ensuing years, *C. burnetii* was found to have a worldwide distribution and a complex ecology and epidemiology. Thousands of cases of Q fever occurred in US troops during World War I, and the disease caused epidemics in the armies fighting during World War II. Three cases of Q fever were recorded in US military personnel during the Persian Gulf War. The risk of Q fever in Kenya today is intermediate year-round with a primarily rural distribution that’s highest where livestock are present. The risk from contaminated milk products is countrywide (NCMI, 2019).

III. Transmission Cycle(s): In nature there are two cycles of infection with *C. burnetii*. One involves arthropods, especially ticks, and a variety of wild vertebrates. The other cycle is maintained among domestic animals. Although humans are rarely, if ever, infected by ticks, arthropods may transmit infection to domestic animals, especially sheep and cattle. Domestic animals have unapparent infections but shed large quantities of infectious organisms in their urine, milk, feces, and especially their placental products. Because *C. burnetii* is highly resistant to desiccation, causing widespread outbreaks in humans and other animals, often at a great distance from place of origin. Dust in sheep or cattle sheds may become heavily contaminated. Once established, animal-to-animal spread of *C. burnetii* is maintained primarily through airborne transmission. Outbreaks of Q fever in humans have been traced to consumption of infected dairy products, contact with contaminated wool or hides, infected straw, and infected animal feces. *C. burnetii* may enter through minor abrasions of the skin or the mucous membranes. Although rare, human-to-human transmission of Q fever has occurred.

IV. Additional Resources:

CDC Background Q Fever


I. Disease Background: Trypanosomiasis, or sleeping sickness, is a parasitic disease transmitted by tsetse flies of the genus Glossina. The protozoan hemoflagellate parasites, *Trypanosoma brucei gambiense* and *T. b. rhodesiense*, are the infectious agents which causes two stages of symptoms. The first stage, the haemolymphatic stage, has fever, headaches, joint pains, and itching as common symptoms while trypanosomes begin to accumulate and spread into the subcutaneous tissues, blood and lymph. As the parasites move across the blood-brain barrier, the disease enters its second stage, the neurological or meningoencephalitic stage, and the central nervous system is infected. This stage sees confusion, poor coordination, sensory disturbances, changes of behavior and a disturbance of the sleep cycle as common symptoms. When infected by *T. b. rhodesiense* sleeping sickness is known as East African sleeping sickness, which progresses rapidly leading to coma and ultimately death typically within months. *T. b. gambiense* infections cause West African sleeping sickness, which progress more slowly and usually kills within three years. A characteristic lesion called a trypanosomal chancre often develops at the site of the bite. A small raised papule develops around five days post bite and quickly enlarges and becomes surrounded by an erythematosus tissue reaction. Treatments for the different parasites and different stages are different and must be started as soon as possible so as to prevent the complications associated with the neurological stage from beginning. There is no vaccine for Trypanosomiasis. A proper diagnosis resides in the ability to observe the parasites in body fluids or tissue by microscopy, and an examination of cerebrospinal fluid to determine the correct stage.

II. Military Impact and Historical Perspective: Sleeping sickness has been known since the slave trade was active, but outbreaks were unknown until the 20th century. Colonial expansion into East Africa by Europeans caused an upheaval of life in totality. The new means of transportation and infrastructure made the transmission of new trypanosome strains much easier and broke down “sanitary barriers” held up for years due to limited contact between the tribal peoples. This expansion coupled with environmental changes lead to three epidemics of severity. The first from 1896 to 1906 in the Congo Basin and Uganda, the second was during the 1920s, and the third began in the 1970s and has continued to the present day where its prevalence has begun to dwindle. The death toll is estimated at 500,000 in the Congo Basin, and upwards of 300,000 in Uganda. Due to active population screening, the disease was almost eradicated in 1960, however, the screening was not continued and sleeping sickness was able to spread once again. The threat of sleeping sickness to military operations in Kenya today is low.

III. Transmission Cycle(s): Sleeping sickness is a disease transmitted primarily through the bite of an infected tsetse fly (*Glossina sp*), however, there are a few other methods of human transmission: vertical transmission (mother-to-child), sexual transmission and infection due to contaminated needle usage. Various animals, domestic and wild (higher infection rates in wild animals), can be reservoirs for trypanosomes (particularly of *T.b. rhodesiense*). *T.b. gambiense* can infect these animals as well but to a much lesser extent. Of the six *Glossina sp.* that transmit Trypanosomiasis, *G. pallidipes* is the major vector in Kenya. Post bloodmeal, the *Trypanosoma sp.* migrate from the hindgut to the salivary glands where they transform into trypanosomes capable of being inoculated into a new host in the fly’s next blood meal. Once these trypanosomes have been injected into a herd it’s possible for the disease to be mechanically spread to other cattle via the bites of other biting flies when they feed on more than one host in a short period of time.

IV. Additional Resources:

CDC Background Sleeping Sickness
WHO Background Sleeping Sickness
CDC Current Situation of Sleeping Sickness
WHO Global Alert and Response


Map of recent sleeping sickness cases in Kenya and its surrounding countries.

Back to table of contents
Crimean-Congo Hemorrhagic Fever (CCHF)

I. Disease Background: CCHF is a zoonotic disease caused by a tick-borne virus of the family Bunyaviridae. The disease is characterized by febrile illness with headache, muscle pain and rash, frequently followed by a hemorrhagic state with hepatitis. The mortality rate can exceed 30%. The incubation period ranges from 3 to 10 days. CCHF may be confused clinically with other hemorrhagic infectious diseases.

II. Military Impact and Historical Perspective: Descriptions of a disease compatible with CCHF can be traced back to antiquity in eastern Europe and Asia. CCHF was first described in soldiers and peasants bitten by ticks of the genus *Hyalomma* while working and sleeping outdoors in the Crimean peninsula in 1944. The virus was first isolated in 1967. Since there are no available treatment regiments of proven value and recovery from CCHF can be very protracted, military personnel with CCHF require significant medical resources. The risk of CCHF to U.S. personnel is intermediate year-round in primarily rural regions of close association with livestock (NCMI, 2019).

III. Transmission Cycle(s): CCHF virus has been isolated from at least 30 species of ticks. From experimental evidence it appears that many species of ticks are capable of transmitting the virus, but members of the genus *Hyalomma* are the most efficient vectors. The aggressive host-seeking behavior of adult hyalomines makes these ticks ideal vectors. The highest prevalence of antibodies in wild and domestic reservoirs has been found in arid areas where *Hyalomma* sp. are common. Antibodies to CCHF virus are widespread in large wild and domestic herbivores. Domestic ruminants generally acquire infection early in life. Viremia in livestock is short-lived and of low intensity. Antibodies or virus have been found in a variety of small mammals, including hares, hedgehogs and rodents. Transovarial transmission of virus in vector ticks is an important reservoir mechanism. Humans acquire CCHF virus from tick bites, from contamination of broken skin or mucous membranes with crushed tissues or feces of infected ticks, or from contact with blood or other tissues of infected animals. CCHF virus is highly infectious, and nosocomial infection of medical workers has been important in many outbreaks. CCHF virus loses infectivity shortly after the death of an infected host. There is no indication that consumption of meat processed according to normal health regulations constitutes a hazard.

IV. Additional Resources:

CDC Background CCHF
WHO Background CCHF


![CCHF Burden Level Map](image)

Burden of CCHF in Africa using a One Health approach. Data support CCHFV circulation in lower Level (1 and 2) countries, whereas further study and surveillance of CCHFV circulation is recommended in countries of higher Levels (3, 4, and 5).

Murine Typhus (Flea-borne typhus, Endemic typhus, Shop typhus)

I. Disease Background: The infectious agent, Rickettsia typhi, causes a milder disease than does R. prowazekii, but it still results in a debilitating illness with high fever. The incubation period ranges from 1 to 2 weeks, and clinical symptoms may last up to 2 weeks in untreated cases. Mortality is very low, and serious complications are infrequent. The disease is easily treated with antibiotics. Absence of louse infestation, seasonal distribution, and the sporadic occurrence of murine typhus help to differentiate it from epidemic typhus.

II. Military Impact and Historical Perspective: Confusion in diagnosis between murine typhus and closely related diseases may occur. Prior to World War II, murine typhus was not distinguished from the epidemic form, and its importance in prior wars is unknown. During World War II, there were 786 cases in the US Army with 15 deaths. There are little available data on the incidence of murine typhus during military operations in Korea or Vietnam. During the Vietnam War, murine typhus was concentrated in port cities and incidence seemed low. However, retrospective studies indicated that a large proportion of fevers of unknown origin experienced by Americans during that conflict were due to R. typhi. The disease is most common in lower socioeconomic classes and increases when disruptions by war or mass migration force people to live in unsanitary conditions in close association with domestic rodents. However, murine typhus has not been a major contributor to disease rates in disaster situations. Because of the sporadic incidence of murine typhus, it is difficult to confidently predict the potential impact of this disease on future military operations, although its military impact would likely be minimal.

III. Transmission Cycle(s): Murine typhus is a zoonotic infection associated with domestic rats (Rattus rattus and R. norvegicus) and vectored by their fleas. The Oriental rat flea, X. cheopis, is the most important vector. Neither rodents nor their ectoparasites are affected by infection with R. typhi. Murine typhus is transmitted by inoculating crushed fleas or infective flea feces into the skin at the bite site. Scratching due to the irritation of flea bites increases the likelihood of infection. R. typhi is rarely transmitted directly by flea bite. Other routes of infection are by inhalation of dry flea feces containing rickettsiae, and ingestion of food contaminated by rodent urine. Dried rickettsiae remain infective for weeks. Murine typhus is not transmitted from person to person.

IV. Additional Resources:

CDC Background Murine Typhus


Plague (Pestis, Black Death)

I. Disease Background: Plague is a zoonotic bacterial disease involving rodents and their fleas, some species of which occasionally transmit the infection to man and other animals. The infectious agent, *Yersinia pestis*, causes fever, chills, myalgia, nausea, sore throat and headache. Bacteria accumulate and swelling develops in the lymph nodes closest to the infected bite. Since most flea bites occur on the lower extremities, the nodes in the inguinal region are involved in 90 percent of cases. The term bubonic plague is derived from the swollen and tender buboes that develop. Plague is most easily treated with antibiotics in the early stages of the disease. However, untreated bubonic plague has a fatality rate of 50%. Infection may progress to septicemic plague with bloodstream dissemination of the bacteria to diverse parts of the body. Secondary involvement of the lungs results in pneumonia. Pneumonic plague is of special medical significance since respiratory aerosols may serve as a source of person-to-person transmission. This can result in devastating epidemics in densely populated areas. Untreated pneumonic or septicemic plague is invariably fatal but responds to early antibiotic therapy. To ensure proper diagnosis, medical personnel should be aware of areas where the disease is enzootic. Plague is often misdiagnosed, especially when travelers or military personnel develop symptoms after returning from an enzootic area.

II. Military Impact and Historical Perspective: Epidemics of plague have been known since ancient times and have profoundly affected civilization. During the Middle Ages, Europe experienced repeated pandemics of plague. Twenty-five percent of the continent’s population died during the great pandemic of the 14th century. The last pandemic of plague originated at the close of the 19th century in northern China and spread to other continents by way of rats on steamships. Plague has been a decisive factor affecting military campaigns, weakening besieged cities or attacking armies during the Middle Ages. Severe ecological disturbances and dislocations of human populations during the Vietnam War led to outbreaks of plague. Even though plague has been declining on a worldwide basis, persistent enzootic foci can trigger the recurrence of epidemics when general sanitation and health services are disrupted by war or natural disaster. Presently, the threat of plague to military operations is intermediate year-round and countrywide with epidemic transmission unlikely (NCMI, 2019).

III. Transmission Cycle(s): Plague is a disease of rodents. It is maintained in nature among wild rodents and their fleas. This zoonotic cycle is termed sylvatic, campestral, rural, or wild. Plague and can be very complex, involving many rodent and flea species. Worldwide, over 220 species of rodents have been shown to harbor *Y. pestis*. Gerbils are important rodent reservoirs in the Middle East. Some rodents are highly susceptible to infection, resulting in high mortality. Although large numbers of dead and dying rodents are a good indication of an epizootic of plague, rodent species that are resistant to the effects of infection are more important in maintaining the zoonotic cycle. Most cases in military personnel would probably occur as a result of intrusion into the zoonotic cycle during or following an epizootic of plague in wild rodents. Domestic cats and dogs may carry infected rodent fleas into buildings or tents. Cats may occasionally transmit infection by their bites or scratches, or by aerosol when they have pneumonic plague. DWF’s should not be allowed to adopt cats or dogs as pets during military operations. The entry of wild rodents or their infected fleas into human habitations can initiate an epizootic among commensal rodents, primarily *Rattus* spp., which are highly susceptible to infection. Close association of humans and large populations of infected commensal rodents can result in an urban cycle of plague. A similar cycle can occur in military cantonments harboring large infestations of commensal rodents. The most important vector of urban plague worldwide is the Oriental rat flea, *Xenopsylla cheopis*. Plague is transmitted to humans primarily by the bite of infected fleas. Fleas often exhibit a host preference, but most species of medical importance readily pass from one host to another. A lack of absolute host specificity increases the potential for infection and transmission of pathogens. Plague may also be acquired by handling tissues of infected animals and infected humans, and by person-to-person transmission of pneumonic plague. Crushed infected fleas and flea feces inoculated into skin abrasions or mucous membranes can also cause infection. Not all flea species are competent vectors. The vector competence of the Oriental rat flea is attributed to enzymes produced by the plague bacilli that cause blood to coagulate in the flea’s digestive tract. The flea attempts to clear the blockage in its digestive tract by repeated efforts to feed. In the process, plague bacilli are inoculated into the host. Fleas may remain infective for months when temperature and humidity are favorable. *Xenopsylla cheopis* may require 2 to 3 weeks after an infective blood meal before it can transmit plague bacilli.

IV. Additional Resources:

CDC Background Plague

WHO Background Plague

Source: WHO/PED, as of 15 March 2016
Onchocerciasis (River Blindness)

I. Disease Background: This is a chronic, nonfatal disease in which adult worms form fibrous nodules in subcutaneous tissues. Adult female worms can live for 15 years and produce thousands of microfilariae that migrate through the skin, causing disfiguring skin lesions. Microfilariae invade other tissues and organs and may reach the eye, where their invasion and subsequent death cause visual disturbances and blindness. The parasite is a filarial nematode worm, *Onchocerca volvulus*. A related species, *O. fasciata*, occurs in camels, but does not infect humans.

II. Military Impact and Historical Perspective: Onchocerciasis has had a devastating impact on villages in the savanna area of West Africa. In many places over 10% of the population is blind. Because of limited exposure, the impact of Onchocerciasis would be insignificant during most military operations. The severity of disease depends on cumulative effects of repeated infection that could result in long-term health problems for continuously exposed troops. Knowledge of this could impact troop morale during an operation. Prolonged infection in an endemic area would be required to develop clinically severe disease. After infection, larvae grow into adult worms over a period of months. Microfilariae are found in the skin a year or more after the infective bite, which is usually long after military personnel have left an endemic area.

III. Transmission Cycle(s): Man is the definitive host in which *O. volvulus* multiplies. Microfilariae in human skin are ingested by vector black flies when they suck blood. In East Africa, vectors are members of the *Simulium damnosum* complex. The microfilariae transform within the black fly to an infective stage that enters the human host when the fly takes subsequent blood meals. This period of development requires 7–14 days. Humans are also the reservoir host. Onchocerciasis is not considered a zoonosis, although natural infections have been found in a spider monkey in Guatemala and a gorilla in the Congo and chimpanzees have been infected in the laboratory.

IV. Additional Resources:

CDC Background Onchocerciasis

Mosquito Vector Species Profiles
**Aedes (Aedeomyia) cumminsii**  
(Theobald, 1903)

**Bionomics:**

*Ae. cumminsii* is primarily zoophilic (cattle) but opportunistically feeds on man. This species is a floodwater mosquito with females laying eggs above the water line pending future flooding to stimulate hatching of the eggs.

**Medical Importance:**

*Ae. cumminsii* is a vector of Rift Valley fever virus (RVFV) in Africa.

**Associated Arboviruses:**

MIDV, NKOV, RVFV, SHOV, SINV, SPOV

[WRBU Species Profile](#)

---

**Aedes (Stegomyia) bromeliae**  
(Theobald, 1911)

**Bionomics:**

Immatures of *Ae. bromeliae* are found in natural containers, including treeholes, bamboo stumps, coconut shells, rockholes, palm fronds, and leaf axils. They are also found in all varieties of artificial containers and will breed indoors. This species is associated with agricultural plots, and readily feeds on humans and monkeys. (Huang, 1986).

**Medical Importance:**

*Aedes bromeliae* is a highly efficient vector of yellow fever virus (YFV).

[WRBU Species Profile](#)

---

[Back to table of contents](#)
Aedes (Aedimorphus) ochraceus (Theobald, 1901)

Bionomics

*Ae. ochraceus* is a floodwater mosquito that preferentially oviposits into the surface layer of soil in locations where future flooding is likely. This species is primarily zoophilic but will opportunistically feed on man.

**Medical Importance:**

*Ae. ochraceus* is a primary vector of RVFV in Africa.

**Associated Arboviruses:**

CHIK, DENV (1-3), WSLV

**WRBU Species Profile**

Habitat Suitability Model: MaxEnt
**Aedes (Aedimorphus) vexans** (Meigen, 1830)

**Bionomics:**

*Aedes vexans* is a floodwater mosquito with a wide global distribution. Immatures are often found in unshaded fresh water flood pools in secondary scrub, but have also been collected in ditches, swamps and rice fields. Habitats usually have little aquatic vegetation or algae. Females are night biters and are opportunistic feeders that readily take blood meals from man and cattle (Reinert 1973).

**Medical Importance:**

* Ae. vexans is capable of transmitting West Nile virus (WNV).

**Associated Arboviruses:**

BAV, BATV, CHAOV, EEEV, GETV, JBEV, JCV, KEYV, LCV, NEGV, POTV, SFV, SLEV, TAHV, TVTV, VRV, VEEV, WEEV, WNV, ZIKV, *Dirofilaria immitis*.

[WRBU Species Profile](#)

Habitat Suitability Model: MaxEnt

[Back to table of contents](#)
**Aedes (Diceromyia) furcifer**

(Edwards, 1913)

**Bionomics:**

*Aedes furcifer* and *Aedes taylori* have historically been confused, and so are often not differentiated in literature making each species’ true distribution difficult to determine. Larvae of *Ae. furcifer* are found in rot-holes in trees (Hopkins, 1952). This species is both zoophilic and anthropophilic.

**Medical Importance:**

*Ae. furcifer* is involved in the monkey-to-human and, to a lesser extent, human-to-human transmission of yellow fever (Germain, Francy, Ferrara et al., 1980), a potential vector of Dengue 2 (Jupp and Kemp 1993), and a vector of Chikungunya viruses (Diallo, Thonnong et al., 1999).

**Associated Arboviruses:**

BOUV, BUNV, CHIKV, DENV, RVFV, YFV, ZIKV

[WRBU Species Profile](#)
**Aedes (Diceromyia) taylori** Edwards, 1936

**Bionomics:**

*Aedes taylori* and *Aedes furcifer* have historically been misidentified as one another, and so are often not differentiated in literature making each species true distribution difficult to deduce. *Aedes taylori* larvae are found in tree holes, rock pools, artificial containers, and a number of other vegetative larval sites.

**Medical Importance:**

*Ae. taylori* is involved in the monkey-to-man and, to a lesser extent, man-to-man transmission of yellow fever (Germain, Francy, Ferrara et al., 1980), a potential vector of Dengue 2 (Jupp and Kemp 1993), and a vector of Chikungunya viruses (Diallo, Thommon et al., 1999).

**Associated Arboviruses:**

BOUV

**WRBU Species Profile**

---

**Aedes (Neomelaniconion) mcintoshi** Huang, 1985

**Bionomics:**

*Aedes mcintoshi* is a floodwater mosquito that is a nighttime biter. It prefers cattle, but has been seen to commonly feed on humans outdoors. In Kenya, pupae were collected in a ground pool and in a medium-sized flooded pool in Ruiru, Kitambu.

**Medical Importance:**

*Aedes mcintoshi* is a major vector of Rift Valley fever virus in East Africa. This species has also been implicated as a major vector of Wesselsbron virus and Middelburg virus in the region.

**Associated Arboviruses:**

BBKV, BUNV, NDUV, NRIV, MIDV, PGAV, RVFV, WESV


**WRBU Species Profile**

---

Back to table of contents
Aedes (Ochlerotatus) caspius  
(Pallas, 1771)

**Bionomics:**

*Aedes caspius* is a salt marsh mosquito with immatures often found in saline marshes near coastal areas. Larvae have also been found in freshwater marshes, however. This species is capable of being a major pest in large numbers as it bites both indoors and out throughout the day and night.

**Medical Importance:**

A known vector of Rift Valley Fever virus (RVFV) and West Nile Virus (WNV). Unique to *Aedes caspius*, it is one of the few mosquito species capable of transmitting the tularemia parasite, *Francisella tularensis*. Other associated pathogens include IKV, ISFV, TAHV, *Cristulospora aedis*, and *Spiroplasma sabaudiense*.


**WRBU Species Profile**

<table>
<thead>
<tr>
<th>Biting Times:</th>
<th>06:00-18:00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Host Preference:</td>
<td>Anthropophilic and zoophilic</td>
</tr>
<tr>
<td>Feeding Behavior:</td>
<td>Exophagic and endophagic</td>
</tr>
<tr>
<td>Resting Behavior:</td>
<td>Exophilic and endophillic</td>
</tr>
</tbody>
</table>

Back to table of contents
Aedes (Stegomyia) africanus (Theobald, 1901)

Bionomics:

*Aedes africanus* has a close association with forests and is reported to have a distribution from sea level to 1800 m. The species is a nighttime biter, primarily taking blood meals from primates at all levels of the forest but will readily feed on man indoors and outdoors. Larvae are found in tree holes, stump holes, bamboo stumps, cut bamboo, tree forks and artificial containers but will preferentially oviposit near ground level in natural containers. (Huang 1979).

Medical Importance:

*Ae. africanus* is one of the most important vectors of arboviruses in the Afrotropical region, and an efficient vector of yellow fever virus (YFV). Chikungunya (CHIKV) and Rift Valley fever (RVFV) viruses have also been isolated from this species (Huang, 1979). Other associated pathogens include BBKV, BOUV, BOZOV, DENV-2, and ZIKV.
**Aedes (Stegomyia) aegypti** (Linnaeus, 1762)

**Bionomics:**

*Aedes aegypti* is the best studied mosquito worldwide. Primarily found in close association with humans, *Ae. aegypti* will use any and all natural and artificial containers as larval breeding sites, but is sensitive to competition with *Aedes albopictus* in these larval sites. Away from urban areas the species tends to favor pools in river beds, tree stumps, tree holes and natural containers. The species is highly anthropophilic and will readily enter houses to feed, but some populations have been found to be zoophilic.

**Associated Pathogens:**

AINOV, AHSV, BOZO, BSQV, BUNV, CATUV, CHIKV, CHPV, CPV, CVV, DENV, EE-EV, EHDV, GROV, HPV, ILHV, ITV, IRIV, JAPV, JBEV, JOIV, KETV, KUNV, LACV, MAYV, MBGV, MCOV, MEBV, MELV, MTBV, MUCV, MVEV, NAVV, NEPV, NOLAV, NTAV, ORIV, ORUV, RESV, RVFV, SFV, SINV, TAHV, TSUV, TYUV, VEEV, VSIV, WARV, WNV, WSLV, YAOV, YF, ZEGV, ZIKV, *Plasmodium gallinaceum*, *Plasmodium lophurae*

**WRBU Species Profile**

**Dengue Vector Bionomics (Ritchie, S. 2014)**

<table>
<thead>
<tr>
<th><strong>Biting Times:</strong></th>
<th>06:00-18:00</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Host Preference:</strong></td>
<td>Primarily anthropophilic</td>
</tr>
<tr>
<td><strong>Feeding Behavior:</strong></td>
<td>Exophagic and endophagic</td>
</tr>
<tr>
<td><strong>Resting Behavior:</strong></td>
<td>Exophilic and endophillic</td>
</tr>
</tbody>
</table>

**Boosted Regression Tree Model: Habitat Suitability**


[Map of habitat suitability for Aedes aegypti]
I. Vector Surveillance and Suppression: Landing rate counts provide a quick relative index of adult abundance. The number of mosquitoes that land on an individual within a short period of time, usually one minute, is recorded. Several indices (container, house, Breteau indices) have been devised to provide a relative measure of the larval populations of *Ae. aegypti*. Adult egg-laying activity can be monitored by using black oviposition cups. Control of dengue fever is contingent upon reducing or eliminating vector populations. Ground or aerial applications of insecticidal aerosols have been relied upon to reduce adult populations during epidemics of dengue. Many vector control specialists have questioned the efficacy of ultra-low volume (ULV) adulticiding. In some outbreaks of dengue fever, ULV dispersal of insecticides has had only modest impact on adult mosquito populations. *Ae. aegypti* is a domestic mosquito that frequently rests and feeds indoors and therefore is not readily exposed to aerosols. The sides of large storage containers should be scrubbed to remove eggs when water levels are low. Water should be stored in containers with tight-fitting lids to prevent access by mosquitoes. A layer of oil will prevent mosquito eggs from hatching and will suffocate the larvae. The elimination of breeding sources, such as old tires, flowerpots, and other artificial containers, is the most effective way to reduce mosquito populations and prevent dengue outbreaks. In Singapore, passage of sanitation laws and their strict enforcement to eliminate breeding sites reduced the house index for *Ae. aegypti* larvae from 25% to 1%. Proper disposal of trash, bottles and cans at military cantonments must be rigidly enforced. The individual soldier can best prevent infection by using personal protective measures during the day when *Ae. aegypti* mosquitoes are active. Wear permethrin-impregnated uniforms and use extended-duration DEET repellent on exposed skin surfaces. For more detailed information on control strategies for Aedes aegypti, see the AFPMB Technical Guide No. 47: *Aedes Mosquito Vector Control*.

II. Reported Insecticide Resistance:


III. Vector Identification:


LUCID Pictorial Key to the Medically Important Mosquitoes of AFRICOM (WRBU)

IV. Additional Resources:


Anopheles (Cellia) arabiensis Patton, 1905

**Bionomics:**

*An. arabiensis* larvae are found in relative short duration, sunlit water pools (3–5 weeks) with high turbidity and a lack of aquatic vegetation or surface film. Chosen breeding sites appear to be associated with cattle, the preferred host. Although primarily known to occur in dry-savannah type environments, *An. arabiensis* is also found in forested areas that have been recently disturbed or cleared. Biting and resting behavior of adult female *An. arabiensis* is known to be highly variable. Adults are known to be both anthropophilic and zoophilic depending on the availability of blood meals. This species is also known to modify resting behavior when in contact with some insecticides used during Indoor Residual Spraying (IRS) control measures.

**Medical Importance:**

*An. arabiensis* is considered a secondary malaria vector in Kenya.


---

**WRBU Species Profile**

<table>
<thead>
<tr>
<th>Biting Times:</th>
<th>19:00-03:00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Host Preference:</td>
<td>Primarily zoophilic but known to be anthropophilic on occasion</td>
</tr>
<tr>
<td>Feeding Behavior:</td>
<td>Exophagic</td>
</tr>
<tr>
<td>Resting Behavior:</td>
<td>Primarily exophillic but known to be endophillic on occasion</td>
</tr>
</tbody>
</table>

---

**Habitat Suitability Model: MaxEnt**

---

[Back to table of contents]
**Anopheles (Cellia) funestus** Giles, 1902

**Bionomics:**

In most parts of its range, *An. funestus* breeds characteristically in bodies of clear water that are either large and more or less permanent, e.g. swamps (near edges if deep), weedy sides of streams, rivers, furrows or ditches, protected portions of lake shore, ponds, or water such as seepages, which are fed from underground permanent sources (Evans, 1938). It is one of the most anthropophilic mosquitoes known. *An. funestus* is also strongly endophilic, resting indoors after blood meals. The great bulk of feeding takes place inside houses after 22:00h up to dawn (Gillies and de Meillon, 1968) with peak biting between 0300 to 0500.

**Medical Importance:**

*An. funestus* is a vector of *Plasmodium vivax*, *P. falciparum* and *Wuchereria bancrofti*

**Associated Arboviruses:**

BOZOV, BUNV, BWAV, GERV, NDOV, NRIV, ONNV, ORUV, SFV, TANV, TATV


[WRBU Species Profile]

Back to table of contents
Anopheles (Cellia) gambiae Giles, 1902

Bionomics:

*Anopheles gambiae* is known as the most dangerous animal in the world. It is the dominant malaria vector in the Afrotropical region and is highly anthropophilic. *An. gambiae s.s.* has been found on sticky traps 40–240 m above the ground, giving the impression that it uses long-distance migration as a potential strategy of survival. These species occur in a great variety of types of water; the most striking are the shallow, open sun-lit pools. Females readily enter houses and bite man both indoors and outdoors starting at sunset and peaking just at dawn (Gillies and de Meillon, 1968).

Medical Importance:

*Anopheles gambiae s.s.* is the primary malaria (*P. falciparum*) vector in Africa (Gillies and de Meillon, 1968). It is also a vector of *P. gallinaceum* (chicken), *P. malariae*, *P. ovale*.

Associated Arboviruses:

AnCV, AnCPV, AgDNV, AngFV, AToV, BARV, BWAV, CVOV, ILEV, NRIV, ONNV, ORUV, TAHV, TATV, WSLV, ZIKV


WRBU Species Profile

Habitat Suitability Model: MaxEnt
**Anopheles (Cellia) moucheti** Evans, 1925

**Bionomics:**

*Anopheles moucheti* inhabits forest-edge environments, and its larvae are found along the borders of slow moving streams and large rivers or in pools or ponds in turbid water. It is a highly anthropophilic and endophilic species readily entering houses to feed (Gilles and de Meillon, 1968). *Anopheles moucheti* bites throughout the night with peak biting between midnight and dawn.

**Medical Importance:**

An important vector of malaria (*P. falciparum* & *P. praefalciparum*) where it occurs in any abundance (Gillies and de Meillon, 1968).

[WRBU Species Profile]

---

**Anopheles (Cellia) nili**

(Theobald, 1901)

**Bionomics:**

*Anopheles nili* s.s. is principally a stream breeder with larvae being found in vegetation or in dense shade along the edges of streams and large rivers. Symes (1931a) found that out of 163 collections of larvae of this species in Kenya, 139 were taken from streams. It is known to be an anthropophilic species biting man readily indoors and outdoors and frequently resting indoors by day (Gillies and de Meillon, 1968:85). Other members of this complex are known to be forest feeders who are primarily zoophilic but will opportunistically feed on humans.

**Medical Importance:**

An important vector of malaria (*Plasmodium* spp.) in many parts of West Africa.

[WRBU Species Profile]

[Back to table of contents]
Anopheles (Cellia) merus Dönitz, 1902

**Bionomics:**

*An. merus* is often found in brackish water or swamps near coastlines, and is known to use salt pans and saline pools for their inland larval habitats. *An. merus* is anthropophilic but generally opportunistic in its selection of hosts. The species is exophilic and bites between 18:00 and 06:00.

**Medical Importance:**

*An. merus* is a known vector of *Plasmodium falciparum*.

---

**WRBU Species Profile**

Habitat Suitability Model: MaxEnt

[Map showing habitat suitability model]

---

*An. merus*, image from Vector Base. Photo Credit: James Gathany, CDC

Back to table of contents
**Anopheles (Cellia) pharoensis** Theobald, 1901

### Bionomics:
Primarily a species of large vegetated swamps, but is also found along lake shores and among floating plants, such as *Pistia* and *Potamogeton*. It’s also found in reservoirs rice fields, streams, ditches, and overgrown wells. They feed from dusk to dawn with a peak at about 01:00 and will enter homes to feed (Gillies and de Meillon, 1968). *An. pharoensis* is zoophilic in Kenya but will opportunistically feed on humans. This species is thought to comprise a species complex over it’s range.

### Medical Importance:
A known vector of malaria in Egypt. In tropical Africa, it is a poor vector of malaria (*P. falciparum* and *P. vivax*) (Gillies and de Meillon, 1968).

### Associated Pathogens:
BGIV, BIRV, RVFV, SINV, *Brugia* spp.

[WRBU Species Profile](#)
Control of Anopheles spp.

I. Vector Surveillance and Suppression: Light traps are used to collect night-biting mosquitoes, but not all Anopheles spp. are attracted to light. The addition of the attractant carbon dioxide to light traps increases the number of species collected. Traps using animals, or even humans, as bait are useful for determining feeding preferences of mosquitoes collected (use of humans as bait must be conducted under approved human use protocols). Adults are often collected from indoor and outdoor resting sites using a mechanical aspirator and flashlight. Systematic larval sampling with a long-handled white dipper provides information on species composition and population dynamics, which is used when planning control measures. Malaria suppression includes elimination of gametocytes from the blood stream of the human reservoir population, reduction of larval and adult Anopheles mosquito populations, use of personal protective measures such as skin repellents, permethrin impregnated uniforms and bed nets to prevent mosquito bites, and chemoprophylaxis to prevent infection. Application of residual insecticides to the interior walls of buildings and sleeping quarters is an effective method of interrupting malaria transmission when local vectors feed and rest indoors. Nightly dispersal of ultra low volume (ULV) aerosols can reduce exophilic mosquito populations. Larvicides and biological control with predaceous fish can control larvae at their aquatic developmental sites before adults emerge and disperse. For more information about Insecticides used for mosquito control consult the AFPMB Technical Guide No. 48, Contingency Pest Management and Vector Surveillance (CAC required). Chemical control may be difficult to achieve in some areas. After decades of malaria control, many vector populations are now resistant to insecticides. Sanitary improvements, such as filling and draining areas of impounded water to eliminate breeding habitats, should be used to the extent possible. The use of bed nets impregnated with a synthetic pyrethroid, preferably permethrin, is an extremely effective method of protecting sleeping individuals from mosquito bites. Buildings and sleeping quarters should be screened to prevent entry of mosquitoes and other blood-sucking insects. The interior walls of tents and bunkers can be treated with permethrin to control resting vectors.

II. Reported Insecticide Resistance:


III. Vector Identification:

Illustrated Key to the Female Anopheles of Southwestern Asia and Egypt (Diptera: Culicidae)

LUCID Pictorial Key to the Medically Important Mosquitoes of AFRICOM


IV. Additional Resources:


Back to table of contents
**Culex (Culex) pipiens** Linnaeus, 1758

**Bionomics:**

Larvae of *Cx. pipiens* are found in numerous and variable breeding places ranging from highly polluted cesspits to clear water pools and containers. Both shaded and unshaded larval habitats are utilized by this species. Females readily feed on humans both indoors and outdoors at night, however, they typically feed almost exclusively on birds.

**Medical Importance:**

Adult *Cx. pipiens* have been found naturally infected with Sindbis virus (SINV), West Nile virus (WNV), Rift Valley fever virus (RVFV) and is a primary vector of periodic Bancroftian filariasis. Other associated arboviruses include AHSV, AI-NOV, FMV, GROV, ITV, JBEV, LACV, LMV, OCKV, ROCV, SFV, TAHV, TURV, TVTV, TYUV, VEEV, USUV

**WRBU Species Profile**

<table>
<thead>
<tr>
<th>Biting Times:</th>
<th>18:00-06:00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Host Preference:</td>
<td>Anthropophilic and zoophilic</td>
</tr>
<tr>
<td>Feeding Behavior:</td>
<td>Exophagic and endophagic</td>
</tr>
<tr>
<td>Resting Behavior:</td>
<td>Exophilic and endophillic</td>
</tr>
</tbody>
</table>

[Back to table of contents]
**Bionomics:**

Immatures of *Cx. quinquefasciatus* have been found in domestic and peri-domestic habitats with clean or polluted water. Typical larval localities are sewers, ditches, agricultural seepage pits, etc. This species is an opportunistic feeder but primarily anthropophilic. *Culex quinquefasciatus* feeds indoors and outdoors at night and will rest both indoors and outdoors as well.

Larvae can be found in bodies of water containing a high degree of organic pollution and close to human habitation. Females readily enter houses at night and preferably bite humans.

**Medical Importance:**

This species is a vector of avian malaria and a primary vector of *Wuchereria bancrofti*, Western equine encephalomyelitis (WEEV), St. Louis encephalitis (SLEV) and West Nile viruses (WNV). Other associated pathogens include AMTV, APEUV, APV, BEFV, BUNV, CHIKV, CHPV, CWV, EEEV, EHDV, GFV, INGV, JEV, KOTV, KOWV, KRIV, KUNV, MAGV, MVEV, NEPV, NTAV, ORIV OROV, PARAV, ROCV, RVFV, SFSV, SFV, STRV, TURV, USUV, VEEV, VSAV, VSIV, VSNJV, WANV, ZEGV, *P. relictum*.

**WRBU Species Profile**

Habitat Suitability Model: MaxEnt
**Bionomics:**

*Culex theileri* is an extremely adaptable and local species as it appears in large amounts in some areas but are nonexistent in others. Larvae have been found in large ground pools that are temporary or permanent, fresh, brackish, salt and foul with no sunlight distinctions. They are also seen in slow moving streams, and artificial containers like cement tanks, jars, etc. *Culex theileri* is seen to be zoophilic in some areas and anthropophilic in others. It can be common in some areas of high elevation and nonexistent in others.

Adults have been collected resting in vegetation and were attracted to human bait near sunset and to CDC traps set in secondary forests, and along edges of swamps and rivers. Larvae are reported from stagnant water (Forattini & Sallum, 1996)

**Medical Importance:**

*Culex theileri* is the principal epidemic vector of Rift Valley fever virus (RVFV) on the inland plateau of southern Africa (McIntosh, 1980). Other associated pathogens include CtFV, GERV, OCKV, SHUV, SINV, WEEV, WNV, *Dirofilaria immitis*.

**WRBU Species Profile**

*Back to table of contents*
**Culex (Culex) univittatus** Theobald, 1901

**Bionomics:**

The distribution of *Culex univittatus* has historically been misidentified as a number of similar species making some of its bionomic information and distribution doubtful. Larvae of *Cx. univittatus* are found in ground pools, marshy pools, barrow pits, stagnant drains and streams, canals and shallow wells. Females feed primarily on birds and mammals but will opportunistically feed on humans. Feeding times range from sunset to sunrise with a peak around midnight.

**Medical Importance:**

*Cx. univittatus* is known to vector West Nile (WNV) and Sindbis (SINV) viruses. Other associated pathogens include BAGV, OLIV, SPOV, USUV, WEEV, WSLV, *Wuchereria bancrofti*.

---

**WRBU Species Profile**

<table>
<thead>
<tr>
<th>Biting Times:</th>
<th>18:00–06:00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Host Preference:</td>
<td>Anthropophilic and zoophilic</td>
</tr>
<tr>
<td>Feeding Behavior:</td>
<td>Exophagic and endophagic</td>
</tr>
<tr>
<td>Resting Behavior:</td>
<td>Exophilic and endophillic</td>
</tr>
</tbody>
</table>

---

Habitat Suitability Model: MaxEnt

---

*Back to table of contents*
**Bionomics:**

The feeding habits of *Culex bitaeniorhynchus* vary on region from feeding primarily on birds to other animals to ferociously attacking humans. Larvae of *Cx. bitaeniorhynchus* are restricted to permanent and temporary ground water habitats including swamps, rice fields, marshes and more containing filamentous green algae (*Spirogyra*) which acts as the primary food source of the immatures. Adults often feed after sunset and continue throughout the night.

**Medical Importance:**

Adult *Cx. bitaeniorhynchus* have been found naturally infected with *Wuchereria bancrofti* and is considered a potential vector of Sindbis virus (SINV). Other associated pathogens include AINOV, AKAV, BATV, DENV-3, GETBV, JBEV, KAIV, MVEV, RVFV, SAGV, UMBV, *Brugia malayi* and *Plasmodium relictum*.

<table>
<thead>
<tr>
<th>Biting Times:</th>
<th>18:00–06:00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Host Preference:</td>
<td>Anthropophilic and zoophilic</td>
</tr>
<tr>
<td>Feeding Behavior:</td>
<td>Exophagic and endophagic</td>
</tr>
<tr>
<td>Resting Behavior:</td>
<td>Exophilic and endophillic</td>
</tr>
</tbody>
</table>

**WRBU Species Profile**
Control of *Culex* spp.


**II. Reported Insecticide Resistance:**


**III. Vector Identification:**


LUCID Pictorial Key to the Medically Important Mosquitoes of East Africa, AFRICOM (WRBU)

**IV. Additional Resources:**


**Mansonia (Mansonioides) uniformis** (Theobald, 1901)

**Bionomics:**

*Mansonia uniformis* has a wide distribution, is highly exophagic and zoophilic (cattle) but will readily bite man and other mammals. Its peak feeding time is between 19:00-20:00 after sunset. Immatures are found in open swamps unshaded by trees and have been found in rice fields as well. Larvae use specially modified siphons to pierce stems and roots of aquatic vegetation to obtain air. (Wharton, 1962).

**Medical Importance:**


---

**WRBU Species Profile**

---

[Back to table of contents]
Sand Fly Vector Species Profiles
Sand Flies of Kenya

I. General Information: Adult sand flies rest during the daytime in dark, humid, protected areas, such as rodent burrows, rock crevices and caves. The preparation of military bunkered ground positions in desert areas provides additional protected daytime resting sites for phlebotomine sand flies. In urban areas, sand fly adults often rest in dark, cool, humid corners of inhabited human and animal structures. Abandoned structures and their vegetative overgrowth often become attractive wild rodent habitats and foci of rural CL. Vegetation is important as a sugar source for both male and female sand flies. Sugar is required for females developing parasite infections. Eggs are developed after a blood meal and are deposited in dark, humid, protected areas. They develop into minute caterpillar-like larvae that feed on mold spores and organic debris. The larvae go through four instars and then pupate near larval feeding sites. Development from egg to adult is 30 to 45 days, depending on feeding conditions and environmental temperatures. Phlebotomine sand fly eggs, larvae and pupae have seldom been found in nature, although exhaustive studies and searches have been made. The adult female has been observed to spread eggs around rather than ovipositing in single egg laying sites. The larvae are believed to be widely distributed in endemic environments but are probably below the ground surface in termite mounds, rodent burrows or other tunnels where temperature, humidity and mold growth provide ideal growing conditions. Because of their minute and delicate nature, larvae have seldom been collected in the wild. The dusk to dawn movement of adults is characterized by flight just above the ground surface to avoid wind. Adult sand flies generally do not travel great distances, and most flights are believed to be less than 100 meters. The females fly in a low hopping flight just above the ground in search of rodent hosts. Both male and female sand flies seek plant sugars from local vegetation. Sand fly habitats in Kenya range from semi-arid areas in the Eastern Province to the caves surrounding Mt. Elgon in the Rift Valley Province to the Coast Province. While there are a number of perennial species in Kenya, rainfall plays a large role in breeding, larval survivability, and distribution. Multiple *Leishmania* spp. are transmitted throughout Kenya. Vector sand flies have short flight ranges. Their dusk to dawn flights coincide with the nomadic activity of peoples of the region, who often travel at night to avoid the extreme heat of daytime hours. Areas with some vegetation, and cliffs, rock outcroppings, or other geologic formations that allow for suitable hiding places and daytime resting sites are important habitats. Exact information on reservoirs and vectors will require more extensive study in many countries of the region. Vast areas of these countries remain unsurveyed for vectors and disease. When searches are made, sand fly vectors are often found in areas where they were previously unknown.

II. Vector Surveillance and Suppression: Sand flies may be collected by a variety of methods. Light traps used for mosquito collection should be modified with fine mesh screens because the small size of phlebotomine sand flies allows them to pass through normal mosquito netting. Sticky traps prepared with paper and vegetable or plant oil are useful and may be placed near rodent burrows, rock crevices, building debris, in and around buildings or constructed military earthworks, and in local vegetation where sand flies are likely to rest during daylight hours. The sticky paper trap is also useful where light traps are either unavailable or their use is limited due to night security measures. Aspirator collections by trained personnel from sand fly resting sites are useful but labor intensive. Identification requires a microscope and some training; however, with some experience, sorting and identification by color and size is quite accurate using minimal magnification. For proper species identification, laboratory microscopes having 100X magnification are required or molecular barcoding. Sand flies are susceptible to most pesticides, and residual insecticide spraying of grounds/structures (inside and outside walls) of encampment areas, coupled with barrier spraying of 200 m of territory surrounding encampment sites, is effective. When the use of organophosphates or other insecticides is impractical due to the combat situation or other operational requirements, personal protective measures (proper wearing of permethrin-treated uniforms and skin repellents) will provide nearly complete protection. Normal mosquito bed nets and screening are ineffective because of the ability of sand flies to crawl through the mesh. Commanders must inform troops of the risks of infection and monitor the proper wearing of uniforms and skin repellents. Since small desert rodents are often the normal hosts of sand flies, selection of encampment sites without vegetation or rock outcroppings that enhance rodent haborage is important. Cleanup and removal of garbage and debris that encourage rodent haborage are necessary for longer periods of occupation. Where combat situations outweigh selection and cleanup, residual insecticide spraying will greatly reduce sand fly prevalence. Again, proper wearing of treated uniforms and use of skin repellents will suffice where other control measures cannot be used to reduce sand fly incidence. Pets must be strictly prohibited because any small desert rodent and/or local dog may be infected with cutaneous or visceral leishmaniasis and other infectious diseases. For more information on control strategies for sand flies consult the AFPMB Technical Guide No. 49: Sand Flies (Diptera: Psychodidae: Phlebotominae): Significance, Surveillance, and Control in Contingency Operations.

III. Vector Identification:

LUCID Pictorial Key to the Medically Important Sand Flies of AFRICOM (WRBU)

IV. Additional Resources:

ECDC Phlebotomines


**Phlebotomus (Larroussius) orientalis** (Parrot, 1936)

**Bionomics:** *Phlebotomus orientalis* has been associated and found in with woodlands in north-eastern Kenya. The type habitat of *Ph. orientalis* is prairie/mountain vegetation.

**Medical Importance:** *Phlebotomus orientalis* is a known vector of *Leishmania donovani* (or *L. archibaldi*) is the main human-biter in the Acacia-Balonites forests of Sudan (Hoogstraal & Heyne-man, 1969; Killick-Kendrick, 1990). This species is the proven vector of visceral leishmaniasis in Kenya (Anjili, 2011).


Habitat Suitability Model: MaxEnt

[Image of a map showing habitat suitability model for *Phlebotomus orientalis*.]
**Phlebotomus (Larroussius) guggisbergi** Kirk & Lewis, 1952

**Bionomics:** This species has been found in the caves around Mt. Elgon in the Rift Valley Province. The type habitat of *Phlebotomus guggisbergi* is steppe.

**Medical Importance:** A large species found in caves and among trees; enters houses where it bites humans. Proven vector of *L. tropica*.


**Phlebotomus (Larroussius) pedifer** Lewis, Mutinga and Ashford, 1972

**Bionomics:** This species has been found in the caves around Mt. Elgon in the Rift Valley Province. The type habitat of *Ph. pedifer* is mountainous vegetation and broadleaf forests. *Phlebotomus pedifer* is anthropophilic.

**Medical Importance:** This species readily bites humans and is a proven vector of cutaneous leishmaniasis (*Leishmania aethiopica*) in Ethiopia and Kenya (Ashford *et al.*, 1973; Kaddu, 1986; WHO, 1981). Females of this species can be confused for the non-vector *P. elgonensis* (= *P. aculeatus*) but they can be distinguished by isoenzyme analysis (Rogo *et al.*, 1988).

Back to table of contents
Phlebotomus (Phlebotomus) duboscqi Neveu-Lemaire, 1906

**Bionomics:** *Phlebotomus duboscqi* is found in a small focus in Baringo district, Rift Valley Province. This species is exophagic and breeds year round. It will opportunistically feed on humans in areas of plentiful vegetation and animal burrows (Anjili, 2011).

**Medical Importance:** This species is a proven vector of *Leishmania major* in Senegal and Kenya and suspected vector throughout the Sahel region of Africa (Dedet *et al.*, 1979; Killick-Kendrick, 1990).

---


---

Habitat Suitability Model: MaxEnt

---

Back to table of contents
**Phlebotomus (Synphlebotomus) martini** Parrot, 1936

**Bionomics:** *Phlebotomus martini* has been captured widely throughout the semi-arid parts of the Rift Valley and Eastern Provinces in Kenya (Anjili, 2011). This species has been shown to feed on dogs, rabbits, goats, cattle and humans and is a perennial breeder. The type habitat of *Ph. martini* is desert vegetation.

**Medical Importance:** A vector of *Leishmania donovani* in Kenya (Adler, 1964; Bray, 1972; Killick-Kendrick, 1990; Southgate, 1977) and perhaps in Ethiopia (south-west) (Fuller et al., 1979). *Phlebotomus martini* is reported to be the only one of three closely related species found in all areas of Kenya endemic for visceral leishmaniasis (Killick-Kendrick, 1990: 80); also see *P. celiae* (The females of *P. celiae, P. martini* and *P. vansomerenae* are morphologically indistinguishable and, therefore, their vectorial roles are not fully known (Killick-Kendrick, 1990; Lewis, 1982).

Tick Vector Species Profiles
Ixodidae (hard) ticks
# Ixodidae (hard) ticks

**Bionomics (General):** Vector ticks, and hence their diseases, tend to be more urban than rural in distribution because they are associated with hosts found in urban areas. *Rhipicephalus sanguineus* (the brown dog tick), in particular, tends to be more concentrated in urban areas, where its canine hosts are abundant. Many of the tick species in Kenya are active year-round. After feeding, females drop from the host and oviposit.

**Medical Importance:** *Hyalomma rufipes* & *H. truncatum* are considered the primary tick vectors of CCHF, however, multiple other species, such as *Amblyomma variegatum* and *R. pulchellus* are capable of transmission. Their importance depends heavily on host preference. *H. dromedarii* and *H. impeltatum* are primarily enzootic vectors. *R. sanguineus*, the brown dog tick, is a suspected zoonotic vector of CCHF and will feed on humans whenever close associations occur. Enzootic infection in dogs, rodents and other animals is usually subclinical. Transmission to humans is by bite of infected ticks. Contamination of breaks in the skin or mucous membranes with crushed tissues or feces of infected ticks can also lead to infection. Several species of Ixodid ticks transmit *Coxiella burnetii* and other viruses and parasites to animals but are not an important source of human infection.

**Vector Identification:**


**Additional Resources:**


<table>
<thead>
<tr>
<th>Species Name</th>
<th>Medical Importance</th>
<th>Bionomics</th>
<th>Host Preference</th>
<th>Additional Information</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Hyalomma impeltatum</em> Schulze &amp; Schlottke, 1930</td>
<td>Crimean-Congo Hemorrhagic Fever (CCHF)</td>
<td>This species is usually a two-host tick that lives in scattered foci of semi-desert, savanna, and steppe biotopes.</td>
<td>Camels, cattle, rodents, hares, ground birds and other large domestic animals</td>
<td>In <em>Hyalomma</em> ticks, the number of eggs laid is variable, ranging from hundreds in rodent burrows to thousands on open ground or vegetation. Eggs usually hatch within 30 days.</td>
</tr>
</tbody>
</table>

[Back to table of contents]
**Rhipicephalus pulchellus** (Gerstacker, 1873) (Zebra tick)

<table>
<thead>
<tr>
<th>Bionomics</th>
<th>This species is a three-host tick</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Importance</td>
<td>Tick typhus, Crimean-Congo Hemorrhagic Fever (CCHF)</td>
</tr>
<tr>
<td>Host Preference</td>
<td>Domestic cattle, sheep, camels, goats, and wild zebras, black rhinoceroses, elands, and gemsbok</td>
</tr>
<tr>
<td>Oviposition</td>
<td><em>Rhipicephalus</em> ticks, lay hundreds of eggs, generally in the dens of host animals, especially dogs.</td>
</tr>
</tbody>
</table>

*Back to table of contents*
*Amblyomma variegatum* Fabricius 1794

<table>
<thead>
<tr>
<th>Bionomics</th>
<th>This species is a three-host tick.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Importance</td>
<td>Crimean-Congo Hemorrhagic Fever (CCHF)</td>
</tr>
<tr>
<td>Host Preference</td>
<td>Sheep and cattle, and occasionally humans.</td>
</tr>
</tbody>
</table>

![Amblyomma variegatum](Photo credit WRBU)

**Habitat Suitability: Ticks of Kenya**

*Amblyomma variegatum*

![Habitat Suitability Map](image)

Back to table of contents
**Hyalomma truncatum** Koch, 1844

**Bionomics**
This species is usually a two-host tick that is found in floodplains in semi-deserts and steppes, or vegetated hillsides and mountain-sides are preferred habitats.

**Medical Importance**
Crimean-Congo Hemorrhagic Fever (CCHF)

**Host Preference**
Cattle, camels and sheep, immature stages tend to parasitize ground-feeding birds.

**Oviposition**
In *Hyalomma* ticks, the number of eggs laid is variable, ranging from hundreds in rodent burrows to thousands on open ground or vegetation. Eggs usually hatch within 30 days.

*Hyalomma truncatum* Photo credit AFPMB

---

**Habitat Suitability: Ticks of Kenya**

*Hyalomma truncatum*

---

Back to table of contents
**Hyalomma rufipes** Koch, 1844

<table>
<thead>
<tr>
<th>Bionomics</th>
<th>This species is a two-host tick.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Importance</td>
<td>Tick typhus, Crimean-Congo Hemorrhagic Fever (CCHF)</td>
</tr>
<tr>
<td>Host Preference</td>
<td>Cattle, sheep, goats, horses and wild ungulates.</td>
</tr>
<tr>
<td>Oviposition</td>
<td>In Hyalomma ticks, the number of eggs laid is variable, ranging from hundreds in rodent burrows to thousands on open ground or vegetation. Eggs usually hatch within 30 days.</td>
</tr>
</tbody>
</table>

**Habitat Suitability: Ticks of Kenya**

*Hyalomma rufipes*

![Map showing habitat suitability for *Hyalomma rufipes* in Kenya](image)
**Haemaphysalis leachi** (Audouin, 1826)

Note: *H. leachi* is recognized as a species complex.

<table>
<thead>
<tr>
<th>Bionomics</th>
<th>This species is a three-host tick.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Importance</td>
<td>Tick typhus</td>
</tr>
<tr>
<td>Host Preference</td>
<td>Domestic dogs and wild carnivores</td>
</tr>
<tr>
<td>Oviposition</td>
<td><em>Ha. leachi</em> can lay 5000 eggs that hatch within 2 to 9 weeks.</td>
</tr>
</tbody>
</table>

**Habitat Suitability: Ticks of Kenya**

*Haemaphysalis leachi*

![Habitat Suitability Map](image)
**Hyalomma dromedarii** Koch, 1844

<table>
<thead>
<tr>
<th>Bionomics</th>
<th>This species may be either a two- or three-host tick.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Importance</td>
<td>Crimean-Congo Hemorrhagic Fever (CCHF)</td>
</tr>
<tr>
<td>Host Preference</td>
<td>Camels, cattle, goats, dogs, small mammals, lizards and occasionally humans</td>
</tr>
<tr>
<td>Oviposition</td>
<td>In <em>Hyalomma</em> ticks, the number of eggs laid is variable, ranging from hundreds in rodent burrows to thousands on open ground or vegetation. Eggs usually hatch within 30 days.</td>
</tr>
</tbody>
</table>

**Rhipicephalus sanguineus** (Latreille, 1806) (Brown Dog Tick)

<table>
<thead>
<tr>
<th>Bionomics</th>
<th>This species is a three-host tick that is prevalent in urban areas because of its close association with dogs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Importance</td>
<td>Crimean-Congo Hemorrhagic Fever, Boutonneuse Fever</td>
</tr>
<tr>
<td>Host Preference</td>
<td>Dogs but also feeds on camels, gerbils and, occasionally, humans.</td>
</tr>
<tr>
<td>Oviposition</td>
<td><em>Rhipicephalus</em> sp. ticks, lay hundreds of eggs, generally in the dens of host animals, especially dogs.</td>
</tr>
<tr>
<td>Questing Behavior</td>
<td>Adult <em>Rhipicephalus</em> sp. are passive in their host-questing activity (rarely moving more than 2 m)</td>
</tr>
</tbody>
</table>
Flea Vector Species Profiles
**Xenopsylla cheopis** (Rothschild, 1903)

**Bionomics:** *Xenopsylla cheopis*, also known as the Oriental rat flea, occurs primarily where commensal rodents are found, particularly *Rattus norvegicus*. Hosts, as well as the primary vector, are more widely distributed in urban areas. *X. cheopis* may occur sporadically in villages, when rats are present, or in highlands, associated with gerbils. The distribution of this flea is determined by the distribution of its hosts, primarily *R. rattus*, *R. norvegicus*, *Mus musculus*, *Meriones* spp. and *Psammomys* spp. (gerbils). Adult fleas feed exclusively on blood and utilize blood protein for egg production. After feeding on a rodent, the female Oriental rat flea lays several (2 to 15) eggs. Several hundred eggs may be laid during the entire life span. Oviposition most often occurs on the hairs of the host, although the eggs drop off and hatch in the nest or its environment. In locally humid environments, such as rodent burrows, eggs may hatch in as little as 2 days. Larvae live in the nest and feed on dried blood, dander, and a variety of organic material. They grow rapidly when temperature exceeds 25°C and the relative humidity is greater than 70%. The larval stages can be completed in as little as 14 days (at 30 to 32°C), or as long as 200 days when temperatures drop below 15°C or when nutrition is inadequate. Mature larvae pupate in cocoons, loosely attached to nesting material. Adult emergence from pupae may occur in as little as 7 days or as long as a year and is stimulated by carbon dioxide or host activity near the cocoon. Adult fleas normally await the approach of a host rather than actively search for one. They feed on humans when people and rodents live close together, but humans are not a preferred host. However, if rat populations decline suddenly due to disease or rat control programs, fleas readily switch to feeding on humans. The life span of adult *X. cheopis* is relatively short compared to that of other flea species.

**Medical Importance:**

*X. cheopis* is considered a primary vector of plague and murine typhus.

*Xenopsylla cheopis* Female, Photo credit J. Stoffer WRBU

[Back to table of contents]
Control of *Xenopsylla cheopis* (Rothschild, 1903)

I. Vector Surveillance and Suppression: The methods of flea surveillance depend upon the species of flea, the host, the ecological situation, and the objective of the investigation. Fleas can be collected from hosts or their habitat. The relationship of host density to flea density should be considered in assessing flea populations. It has been common practice for years to use a flea index (average number of fleas per host), especially in studies of rodent fleas. For *X. cheopis*, a flea index > 1.0 flea per host is considered high. The flea index has many limitations, since only adults are considered and then only while they are on the host. Fleas are recovered by combing or brushing the host or by running a stream of carbon dioxide through the fur while holding the host over a white surface. Flea abundance in the environment can be determined by counting the number of fleas landing or crawling in one minute on the lower parts of the legs of the observer. The trouser legs should be tucked into the socks to prevent bites. Flea populations can also be estimated by placing a white cloth on the floor in buildings or on the ground in rodent habitat and counting the fleas that jump onto the cloth. Various flea traps have been devised. Some use light or carbon dioxide as an attractant. Use of a modified Tullgren apparatus, based of the Berlase funnel, sifting and flotation of rodent nesting materials and dust and debris from infested buildings are effective methods of collecting fleas from the environment. Serologies of wild carnivores are sensitive indicators of enzootic plague. Control of enzootic plague over large areas is not feasible. Control efforts should be limited to foci adjacent to urban areas, military encampments, or other areas frequented by military personnel. If possible, cantonment sites should not be located in wild rodent habitat. Fleas quickly leave the bodies of dead or dying rodents in search of new hosts. Consequently, flea control must always precede or coincide with rodent control operations. Application of insecticidal dusts to rodent burrows is effective in reducing flea populations, but it is very labor intensive. Baiting with formulations that rodents carry to their dens or with baits containing systemic insecticides that kill fleas when they feed, has been effective but may pose environmental risks. Urban plague control requires that rodent runs, harborage and burrows be dusted with an insecticide labeled for flea control and known to be effective against local fleas. Insecticide bait stations can also be used. Rat populations should be suppressed by well planned and intensive campaigns of poisoning and concurrent measures to reduce rat harborage and food sources. Buildings should be rat-proofed to the extent possible to prevent rats from gaining entry. Insecticides recommended for flea control are listed in AFPMB Technical Guide No. 24, Contingency Pest Management Guide. Military personnel, especially those involved in rodent control, should use the personal protective measures discussed in AFPMB Technical Guide No. 36: Personal Protective Techniques Against Insects and Other Arthropods of Military Significance. Active immunization with a vaccine of killed bacteria confers protection against bubonic plague (but not pneumonic plague) in most recipients for several months. Booster injections are necessary every six months. Vaccination should not be relied upon as the sole preventive measure. For more detailed information consult the AFPMB Technical Guide No. 40: Methods for Trapping and Sampling Small Mammals for Virologic Testing.

II. Vector Identification:


CDC Pictorial Keys to Arthropods, Reptiles, Birds, and Mammals of Public Health Significance: Fleas

III. Additional Resources:

Flea Morphology (BYU, Fleas of the World)

Note: Fleas and tissues from suspected reservoirs or humans may be submitted for plague analysis to the Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Vector-borne Infectious Diseases, P.O. Box 2087, Foothills Campus, Fort Collins, Colorado 80522. Contact Centers for Disease Control and Prevention at (970) 221-6400 for additional information.
Other Vector Species Profiles
**Glossina (Morsitans) pallidipes** Austen, 1903

(Tsetse fly)

**Bionomics:** Tsetse flies are obligate blood-sucking dipterans of medical importance due to their ability to carry and transmit Trypanosomiasis, or sleeping sickness. They resemble house flies and typically have a size range of 8 mm to 17 mm. They’re easily identifiable while resting through two characteristics: one wing is rested directly on top of the other and they have a lengthy proboscis which extends directly forward and is attached through a unique appendage to the bottom of their head. Female flies contain a uterus in which a single egg hatches. This larva then feeds on the milk glands of the tsetse fly for 10-15 days when the fly deposits the now larger, third instar larva into soil. After burrowing into the ground, the larva pupates almost immediately and emerges into an adult fly three weeks or longer later. Both sexes of tsetse flies exclusively take blood meals to feed. One female can only produce eight-ten larva in ten to twelve day intervals, which allows their populations to be controlled relatively easily. Tsetse flies readily feed on humans as well as both wild and domestic animals, all of which are reservoirs for sleeping sickness parasites (trypanosomes). *Glossina* spp. are highly mobile insects who can travel up to 1 km per day making accurate modeling somewhat difficult. The bites of tsetse flies can be very painful and often result in a reaction on the bite site known as a trypanosomal chancre. The species that transmit sleeping sickness generally breed under trees near watering holes such as rivers and lakes, or in an open woodland when feeding off of larger animals. Tsetse flies are more active as biters during midday hours and their activity typically declines after sunset. Members of the *Morsitans*, or savanna, and *Palpalis*, or riverine groups, contain the most medically important species. *G. palpalis* and *G. morsitans* or the most significant vectors throughout the whole of Africa, however, *G. pallidipes*, a member of the *Morsitans* subgenus, is the most significant in Kenya.

**Medical Importance:** Members of the *Morsitans* group are the primary vectors of Trypanosomiasis in Kenya. *Glossina (Morsitans) pallidipes* is a member of this group and is the most significant vector of sleeping sickness in Kenya.

**Vector Surveillance and Suppression:** The most effective efforts of control have typically been those centered on their environment such as the removal of wild game, clearing of forest land, and burning to prevent brush growth. More typical methods of spraying insecticides and trapping may lower local populations, but are not feasible means of eliminating the vectors as a whole. Population screening for Trypanosomiasis parasites is one of the main control strategies used as humans are significant disease reservoirs. Permethrin and other repellent have not been shown to be very effective against tsetse flies but they would aid in the prevention of Trypanosomiasis spread through mechanical means (other fly bites). Military personnel should minimize their contact with tsetse flies by wearing protective clothing, avoiding bush and woodland, and inspecting vehicles before they enter as tsetse flies are attracted to the movement and dust caused by vehicles in motion.

**Vector Identification:**


Training Manual for Tsetse Control Personnel

**Additional Resources:**

- WHO Tsetse Fly Profile
- Britannica Tsetse Fly Profile
- BBC Tsetse Fly and Sleeping Sickness
- CDC Prevention and Control
- Vector Base
- Tsetse Fly Module

[Back to table of contents]
**Simulium damnosum** s.l. (Black fly)

**Bionomics:** After a bloodmeal, female black flies lay eggs on emergent vegetation along streams, or on logs and rocks that are splashed with water. Several masses of 150 to 500 eggs may be laid over a life span of 3 to 4 weeks. Eggs hatch in 2 to 3 days at temperatures of 25 to 30°C. Using caudal suckers and silken threads, black fly larvae attach to rocks in swift flowing streams, generally in mountainous areas of 300 to 1,200 m. They require relatively clean streams with high oxygen content. Larvae feed on small crustaceans, protozoa, algae, bacteria, and decaying bits of plants and animals suspended in the water. They progress through 6 to 9 (often 7) instars, and pupate 7 to 12 days after hatching, depending on temperature. Pupae are found in streams for about 1 to 2 weeks prior to emergence of adults. *Simulium damnosum* complex vectors are fierce biters that emerge in large numbers during the rainy season. Many generations can be produced as long as streams are flowing. Females often circle in swarms around the lower extremities of human hosts. They are persistent biters that feed primarily outside and during the day. Enorgo-ment usually requires only a few minutes. Bites may cause extreme irritation and itching in human or animal hosts. In sensitive persons, black fly bites can cause an acute allergic response. These flies are anthropophilic but also feed on cattle. Black flies are exophilic and not noted for entering human structures. After feeding, black flies fly to nearby shaded sites or protective vegetation. Black flies are strong fliers that can travel many kilometers (5 to 10 km or more) from their home streams. It is estimated that strong winds could easily carry them an additional 5 to 10 km from their breeding sites. Because most suitable streams flow primarily during the rainy season, the seasonal distribution of black flies is usually short. Historically, species from the *S. damnosum* complex in Kenya have been found in all geographic zones of Kenya where flowing water is present, namely throughout perennial river-systems found around Mt. Kenya and in the western portion of the country. Identification of species within the *S. damnosum* complex requires chromosomal analysis or molecular barcoding since they are morphologically identical.

**Medical Importance:** Members of the *Simulium damnosum* complex are the primary vectors of Onchocerciasis (river blindness).

**Vector Surveillance and Suppression:** Control can rarely be achieved by directly attacking the adult black fly. Adults are susceptible to insecticides but are usually too widely dispersed for insecticidal spraying or fogging to achieve more than very temporary local control. Black fly populations are most concentrated in the immature aquatic stages. Control measures have been directed against black fly larvae with great success. Black fly larvae are susceptible to very low doses of many insecticides, including the biological control agent *Bacillus thuringiensis* (BTI). Aerial larviciding is usually necessary to treat rivers with extensive tributary systems. Reducing contact between black flies and military personnel is best achieved by using personal protective measures, such as wearing protective clothing and headgear and applying repellents. Control efforts eradicated *Simulium neavai*, the historical primary vector of Onchocerciasis in Kenya.

**Vector Identification:**


**Additional Resources:**


Back to table of contents
**Pediculus humanus** Linnaeus, 1758

**Bionomics:** Human lice spend their entire life cycle (egg, 3 nymphal stages and adult) on the host. Eggs of body lice are attached to clothing at a rate of about 5 eggs per female per day. At 29 to 32°C, eggs hatch in 7 to 10 days. The maximum time eggs can survive unhatched is 3 to 4 weeks, which is important when considering the survival of lice in infested clothing and bedding. A blood meal is required for each of the 3 nymphal molts and for egg production in adults. The nymphal stages are passed in 8 to 16 days. Louse populations have the potential to double every 7 days. Adults live about 2 weeks and feed daily. Infestations of lice cause considerable irritation and scratching, which may lead to skin lesions and secondary infections. Body lice are commonly found in the seams and folds of clothing. Lice tolerate only a narrow temperature range and will abandon a dead host or one with a body temperature of 40°C or above. This contributes to the spread of lice and louse-borne disease. Human lice can survive without a host for only a few days.

**Medical Importance:** Human lice are known vectors of louse-borne relapsing fever and epidemic typhus.

**Vector Surveillance and Suppression:** Surveillance for body lice consist of examining individuals and their clothing for lice or nits (eggs). Body louse infestations have declined with higher standards of living, although infestations are still common in some populations. Military personnel should avoid close personal contact with infested persons and their belongings, especially clothing and bedding. Dry cleaning or laundering clothing or bedding in hot water (55°C for 20 minutes) will kill eggs and lice. Control of epidemics requires mass treatment of individuals and their clothing with effective insecticides. The permethrin-treated uniform is extremely effective against lice. Since lice cannot survive away from the human host, application of insecticides to buildings, barracks or other living quarters is not necessary.

**Vector Identification:**

University of Florida, Entomology and Nematology, Featured Creatures: Body Louse

CDC Pictorial Keys to Arthropods, Reptiles, Birds, and Mammals of Public Health Significance: Lice

**Additional Resources:**

CDC: Pediculosis Background

Personal Protective Measures

**Field Uniform:** Personal protective measures are the first line of defense against arthropod-borne disease and, in some cases, may be the only protection for deployed military personnel. Proper wearing of the uniform and appropriate use of repellents can provide high levels of protection against blood-sucking arthropods. The uniform fabric provides a significant mechanical barrier to mosquitoes and other blood-sucking insects. Therefore, the uniform should be worn to cover as much skin as possible if weather and physical activity permit. Proper wearing of the field uniform is essential to minimize skin exposure (Figure 2-1). If the risk of heat stress is a factor in a particular environment, common sense or advice from medical or Preventive Medicine personnel should dictate when the following recommendations are not practical:

1. Tuck pant legs into boots or into socks. This forces non-flying pests, such as ticks, chiggers, stinging ants and spiders, to climb up the outside of the pant legs, thus decreasing access to the skin and increasing the likelihood of their being seen.

2. Roll sleeves down and close the collar to help protect the arms and neck from arthropod attack. This is especially important in malaria-endemic regions when Anopheles species bite from dusk until dawn.

3. It is difficult for arthropods to bite through the uniform fabric unless it is pulled tightly against the skin. Therefore, the uniform should be worn loosely, with an undershirt worn underneath the coat to act as an added barrier. The undershirt should be tucked into the pants to decrease access by crawling arthropods at the waistline. Mosquitoes can easily bite through tight-fitting material such as that used for the combat uniform.

4. The field cap and its brim help protect the head and face. Some biting insects tend to avoid the shaded area of the face under the cap's brim.

5. Uniforms that are treated with permethrin provide protection only on the covered portion of the body. Mosquitoes will still readily feed on the hands, neck and head. It is essential to apply an approved insect repellent to exposed body surfaces. Re-application is advised according to the label.

For more information on personal protective measures, consult **AFPMB Technical Guide No. 36:** *Personal Protective Measures Against Insects and Other Arthropods of Military Significance.*
Personal Protective Measures

When personnel are operating in tick-infested areas, they should tuck their pant legs into their boots to prevent access to the skin by ticks, chiggers, and other crawling arthropods. They should also check themselves frequently for ticks and immediately remove any that are found. If a tick has attached, seek assistance from medical authorities for proper removal or follow these guidelines from TIM 36,

1. Grasp the tick’s mouthparts where they enter the skin, using pointed tweezers.

2. Pull out slowly and steadily with gentle force. a. Pull in the reverse of the direction in which the mouthparts are inserted, as you would for a splinter. b. Be patient – The long, central mouthpart (called the hypostome) is inserted in the skin. It is covered with sharp barbs, sometimes making removal difficult and time consuming. c. Many hard ticks secrete a cement-like substance during feeding. This material helps secure their mouthparts firmly in the flesh and adds to the difficulty of removal. d. It is important to continue to pull steadily until the tick can be eased out of the skin. e. Do not pull back sharply, as this may tear the mouthparts from the body of the tick, leaving them embedded in the skin. If this happens, don’t panic. Embedded mouthparts are comparable to having a splinter in your skin. However, to prevent secondary infection, it is best to remove them. Seek medical assistance if necessary. f. Do not squeeze or crush the body of the tick because this may force infective body fluids through the mouthparts and into the wound. g. Do not apply substances like petroleum jelly, fingernail polish remover, repellents, pesticides, or a lighted match to the tick while it is attached. These materials are either ineffective or, worse, may agitate the tick and cause it to salivate or regurgitate infective fluid into the wound site. If tweezers are not available, grasp the tick’s mouthparts between your fingernails, and remove the tick carefully by hand. Be sure to wash your hands especially under your fingernails -- to prevent possible contamination by infective material from the tick.

3. Following removal of the tick, wash the wound (and your hands) with soap and water and apply an antiseptic.

4. Save the tick in a jar, vial, small plastic bag, or other container for identification should you later develop disease symptoms. Preserve the tick by either adding some alcohol to the jar or by keeping it in a freezer. Storing a tick in water will not preserve it. Identification of the tick will help the physician’s diagnosis and treatment, since many tick-borne diseases are transmitted only by certain species.

5. Discard the tick after one month; all known tick-borne diseases will generally display symptoms within this time period. Newly developed repellents provide military personnel with unprecedented levels of protection. An aerosol formulation of permethrin (NSN 6840-01-278-1336) can be applied to the uniform according to label directions, but not to the skin. This will impart both repellent and insecticidal properties to the uniform material that will be retained through numerous washings. An extended formulation lotion of N, N-diethyl-m-toluamide (deet) (NSN 6840-01-284-3982) has been developed to replace the 2 oz. bottles of 75% deet in alcohol. This lotion contains 33% active ingredient. It is less irritating to the skin, has less odor and is generally more acceptable to the user. A properly worn Battle Dress Uniform (BDU) impregnated with permethrin, combined with use of extended duration deet on exposed skin, has been demonstrated to provide nearly 100% protection against a variety of blood-sucking arthropods. This dual strategy is termed the DoD INSECT REPELLENT SYSTEM. In addition, permethrin may be applied to bednets, tents, and other field items as appropriate. Complete details regarding these and other personal protective measures are provided in TIM 36, Personal Protective Techniques Against Insects and Other Arthropods of Military Significance (1996).

For more information on personal protective measures, consult AFPMB Technical Guide No. 36: Personal Protective Measures Against Insects and Other Arthropods of Military Significance.

Back to table of contents
# Table of Arboviruses & Parasites

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Arbovirus</th>
<th>Abbreviation</th>
<th>Arbovirus</th>
</tr>
</thead>
<tbody>
<tr>
<td>AINOV</td>
<td>Aino Virus</td>
<td>AgDNV</td>
<td>Anopheles gambiae Denso-</td>
</tr>
<tr>
<td>AnCV</td>
<td>Anopheles C Virus</td>
<td>AngFV</td>
<td>Anopheles gambiae Fla-</td>
</tr>
<tr>
<td>AToV</td>
<td>Anopheles Totivirus</td>
<td>AHSV</td>
<td>African Horseickness Virus</td>
</tr>
<tr>
<td>AMTV</td>
<td>Arumowot Virus</td>
<td>APEUV</td>
<td>Apeu Virus</td>
</tr>
<tr>
<td>APV</td>
<td>Agua Preta Virus</td>
<td>BAGV</td>
<td>Bagaza Virus</td>
</tr>
<tr>
<td>BAV</td>
<td>Banna Virus</td>
<td>BARV</td>
<td>Barur Virus</td>
</tr>
<tr>
<td>BBKV</td>
<td>Babanki Virus</td>
<td>BEFV</td>
<td>Bovine Ephemeral Fever Virus</td>
</tr>
<tr>
<td>BGIV</td>
<td>Bangui Virus</td>
<td>BIRV</td>
<td>Birao Virus</td>
</tr>
<tr>
<td>BOUV</td>
<td>Boubouí Virus</td>
<td>BOZOV</td>
<td>Bozo Virus</td>
</tr>
<tr>
<td>BSQV</td>
<td>Bussuquara Virus</td>
<td>BUNV</td>
<td>Bunyamwera Virus</td>
</tr>
<tr>
<td>BWAV</td>
<td>Bwamba Virus</td>
<td>CHAOV</td>
<td>Chaoyang Virus</td>
</tr>
<tr>
<td>CHIKV</td>
<td>Chikungunya Virus</td>
<td>CHPV</td>
<td>Chandipura Virus</td>
</tr>
<tr>
<td>CPV</td>
<td>Coastal Plains Virus</td>
<td>CTFV</td>
<td>Colorado Tick Fever Virus</td>
</tr>
<tr>
<td>CVOV</td>
<td>Calovo Virus</td>
<td>CVV</td>
<td>Cache Valley Virus</td>
</tr>
<tr>
<td>CWV</td>
<td>Cape Wrath Virus</td>
<td>DENV 1-3</td>
<td>Dengue Virus 1, Dengue Virus 2, Dengue Virus 3</td>
</tr>
<tr>
<td>EEEV</td>
<td>Easter Equine Encephalomyelitis Virus</td>
<td>EHDV</td>
<td>Epizootic Hemorrhagic Disease of Deer Virus</td>
</tr>
<tr>
<td>FMV</td>
<td>Fort Morgan Virus</td>
<td>GERV</td>
<td>Germiston Virus</td>
</tr>
<tr>
<td>GFV</td>
<td>Gabek Forest Virus</td>
<td>GROV</td>
<td>Guaroa Virus</td>
</tr>
<tr>
<td>HPV</td>
<td>Hart Park Virus</td>
<td>IKV</td>
<td>Issyk-Kul Virus</td>
</tr>
<tr>
<td>ILEV</td>
<td>Ilesha Virus</td>
<td>ILHV</td>
<td>Ilheus Virus</td>
</tr>
</tbody>
</table>

[Back to table of contents]
# Table of Arboviruses & Parasites

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Arbovirus</th>
<th>Abbreviation</th>
<th>Arbovirus</th>
</tr>
</thead>
<tbody>
<tr>
<td>INGV</td>
<td>Inwavuma Virus</td>
<td>ISFV</td>
<td>Isfahan Virus</td>
</tr>
<tr>
<td>ITV</td>
<td>Israel Turkey Meningoencephalitis Virus</td>
<td>IRIV</td>
<td>Irituia Virus</td>
</tr>
<tr>
<td>JAPV</td>
<td>Japanaut Virus</td>
<td>JBEV</td>
<td>Japanese B Encephalitis Virus</td>
</tr>
<tr>
<td>JCV</td>
<td>Jamestown Canyon Virus</td>
<td>JEV</td>
<td>Japanese Encephalitis Virus</td>
</tr>
<tr>
<td>JOIV</td>
<td>Joinjakaka Virus</td>
<td>KAIIV</td>
<td>Kaikalur Virus</td>
</tr>
<tr>
<td>KEYV</td>
<td>Keystone Virus</td>
<td>KOTV</td>
<td>Kotonkan Virus</td>
</tr>
<tr>
<td>KOWV</td>
<td>Kowanyama Virus</td>
<td>KRIV</td>
<td>Kairi Virus</td>
</tr>
<tr>
<td>KUNV</td>
<td>Kunjin Virus</td>
<td>LACV</td>
<td>La Crosse Virus</td>
</tr>
<tr>
<td>LCV</td>
<td>Lake Clarendon Virus</td>
<td>LMV</td>
<td>Las Maloyas Virus</td>
</tr>
<tr>
<td>MAGV</td>
<td>Maguari Virus</td>
<td>MALV</td>
<td>Malakai Virus</td>
</tr>
<tr>
<td>MBGV</td>
<td>Marburg Virus</td>
<td>MCOV</td>
<td>Marco Virus</td>
</tr>
<tr>
<td>MEBV</td>
<td>Mount Elgon bat Virus</td>
<td>MIDV</td>
<td>Middleburg Virus</td>
</tr>
<tr>
<td>MTBV</td>
<td>Marituba Virus</td>
<td>MUCV</td>
<td>Mucambo Virus</td>
</tr>
<tr>
<td>MVEV</td>
<td>Murray Valley Encephalitis Virus</td>
<td>NAVV</td>
<td>Navarro Virus</td>
</tr>
<tr>
<td>NDOV</td>
<td>Nyando Virus</td>
<td>NDUV</td>
<td>Ndumu Virus</td>
</tr>
<tr>
<td>NEGV</td>
<td>Negishi Virus</td>
<td>NEPV</td>
<td>Nepuyo Virus</td>
</tr>
<tr>
<td>NKOV</td>
<td>Nkolbisson Virus</td>
<td>NOLAV</td>
<td>Nola Virus</td>
</tr>
<tr>
<td>NRIV</td>
<td>Nigari Virus</td>
<td>NTAV</td>
<td>Ntaya Virus</td>
</tr>
<tr>
<td>OCKV</td>
<td>Ockelbo Virus</td>
<td>OLIV</td>
<td>Olifantsylei Virus</td>
</tr>
<tr>
<td>ONNV</td>
<td>O’nyong-nyong Virus</td>
<td>ORIV</td>
<td>Oriboca Virus</td>
</tr>
</tbody>
</table>

[Back to table of contents]
# Table of Arboviruses & Parasites

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Arbovirus</th>
<th>Abbreviation</th>
<th>Arbovirus</th>
</tr>
</thead>
<tbody>
<tr>
<td>OROV</td>
<td>Oropuche Virus</td>
<td>ORUV</td>
<td>Orungo Virus</td>
</tr>
<tr>
<td>PARAV</td>
<td>Para Virus</td>
<td>PGAV</td>
<td>Pongola Virus</td>
</tr>
<tr>
<td>PGAV</td>
<td>Pongola Virus</td>
<td>POTV</td>
<td>Potosi Virus</td>
</tr>
<tr>
<td>PUCV</td>
<td>Puchong Virus</td>
<td>RESV</td>
<td>Restan Virus</td>
</tr>
<tr>
<td>ROCV</td>
<td>Rocio Virus</td>
<td>RRV</td>
<td>Ross River Virus</td>
</tr>
<tr>
<td>RVFV</td>
<td>Rift Valley Fever Virus</td>
<td>SAGV</td>
<td>Sagiyama Virus</td>
</tr>
<tr>
<td>SFSV</td>
<td>Sandfly Fever Sicilian Virus</td>
<td>SFV</td>
<td>Semliki Forest Virus</td>
</tr>
<tr>
<td>SHOV</td>
<td>Shokwe Virus</td>
<td>SHUV</td>
<td>Shuni Virus</td>
</tr>
<tr>
<td>SINV</td>
<td>Sindbis Virus</td>
<td>SLEV</td>
<td>Saint Louis Encephalitis Virus</td>
</tr>
<tr>
<td>SPOV</td>
<td>Spondweni Virus</td>
<td>STRV</td>
<td>Stratford Virus</td>
</tr>
<tr>
<td>TAHV</td>
<td>Tahyna Virus</td>
<td>TANV</td>
<td>Tanga Virus</td>
</tr>
<tr>
<td>TSUV</td>
<td>Tsuruse Virus</td>
<td>TURV</td>
<td>Turlock Virus</td>
</tr>
<tr>
<td>TVTV</td>
<td>Trivittatus Virus</td>
<td>TYUV</td>
<td>Tyuleniy Virus</td>
</tr>
<tr>
<td>UMBV</td>
<td>Umbre Virus</td>
<td>USUV</td>
<td>Usutu Virus</td>
</tr>
<tr>
<td>VFV</td>
<td>Virgin River Virus</td>
<td>VEEV</td>
<td>Venezuelan Equine Encephalitis Virus</td>
</tr>
<tr>
<td>VSAV</td>
<td>Vesicular Stomatitis, Alagoas Serotype Virus</td>
<td>VSIV</td>
<td>Vesicular Stomatitis, Indiana Serotype Virus</td>
</tr>
<tr>
<td>VSNJV</td>
<td>Vesicular Stomatitis, New Jersey Serotype Virus</td>
<td>WANV</td>
<td>Wanowrie Virus</td>
</tr>
<tr>
<td>WNV</td>
<td>West Nile Virus</td>
<td>WEEV</td>
<td>Western Equine Encephalomyelitis Virus</td>
</tr>
<tr>
<td>WSLV</td>
<td>Wesselsbron Virus</td>
<td>YAOV</td>
<td>Yaounde Virus</td>
</tr>
<tr>
<td>YATAV</td>
<td>Yata Virus</td>
<td>YFV</td>
<td>Yellow Fever Virus</td>
</tr>
<tr>
<td>ZEGV</td>
<td>Zegla Virus</td>
<td>ZIKV</td>
<td>Zika Virus</td>
</tr>
</tbody>
</table>

[Back to table of contents](#)
# Table of Arboviruses & Parasites

<table>
<thead>
<tr>
<th>Parasite</th>
<th>Associated Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Brugia malayi</em></td>
<td>Lymphatic filariasis</td>
</tr>
<tr>
<td><em>Brugia patei</em></td>
<td>Filariasis</td>
</tr>
<tr>
<td><em>Dirofilaria immitis</em></td>
<td>Dirofilariasis</td>
</tr>
<tr>
<td><em>Francisella tularensis</em></td>
<td>Tularemia</td>
</tr>
<tr>
<td><em>Plasmodium falciparum</em></td>
<td>Human malaria</td>
</tr>
<tr>
<td><em>Plasmodium lophurae</em></td>
<td>Malaria</td>
</tr>
<tr>
<td><em>Plasmodium ovale</em></td>
<td>Human malaria</td>
</tr>
<tr>
<td><em>Plasmodium relictum</em></td>
<td>Malaria</td>
</tr>
<tr>
<td><em>Brugia pahangi</em></td>
<td>Filariasis</td>
</tr>
<tr>
<td><em>Cristulospora aedis</em></td>
<td>Microsporidiosis</td>
</tr>
<tr>
<td><em>Dirofilaria repens</em></td>
<td>Dirofilariasis</td>
</tr>
<tr>
<td><em>Plasmodium cynomolgibastienelii</em></td>
<td>Malaria</td>
</tr>
<tr>
<td><em>Plasmodium gallinaceum</em></td>
<td>Malaria</td>
</tr>
<tr>
<td><em>Plasmodium malariae</em></td>
<td>Human malaria</td>
</tr>
<tr>
<td><em>Plasmodium praefalicparum</em></td>
<td>Malaria</td>
</tr>
<tr>
<td><em>Plasmodium vivax</em></td>
<td>Human malaria</td>
</tr>
<tr>
<td><em>Wuchereria bancrofti</em></td>
<td>Lymphatic/Bancroftian filariasis</td>
</tr>
</tbody>
</table>

[Back to table of contents]
# Bionomics Table: Mosquito Vectors of Kenya

<table>
<thead>
<tr>
<th>Species Name</th>
<th>Vector Status</th>
<th>Biting Times</th>
<th>Host Preference</th>
<th>Feeding Behavior</th>
<th>Resting Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Anopheles</em> (<em>Cel.</em>) <em>arabiensis</em></td>
<td>Malaria</td>
<td>19:00–03:00</td>
<td>Primarily zoophilic but known to be Anthropophilic on occasion</td>
<td>Exophagic</td>
<td>Primarily exophillic but known to be endophillic on occasion</td>
</tr>
<tr>
<td><em>Aedes</em> (<em>Adm.</em>) <em>cumminsii</em></td>
<td>RVFV</td>
<td>No data</td>
<td>Primarily zoophilic but opportunistically anthropophilic</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td><em>Aedes</em> (<em>Stg.</em>) <em>bromeliae</em></td>
<td>YFV</td>
<td>No data</td>
<td>Anthropophilic and zoophilic</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td><em>Aedes</em> (<em>Stg.</em>) <em>aegypti</em></td>
<td>DENV, CHIKV, ZIKV</td>
<td>06:00–18:00</td>
<td>Primarily anthropophilic</td>
<td>Exophagic and endophagic</td>
<td>Exophillic and endophillic</td>
</tr>
<tr>
<td><em>Culex</em> (<em>Cux.</em>) <em>pipiens</em></td>
<td>WNV, SINV, Bancroftian filariasis</td>
<td>18:00–06:00</td>
<td>Anthropophilic and zoophilic</td>
<td>Exophagic and endophagic</td>
<td>Exophillic and endophillic</td>
</tr>
<tr>
<td><em>Culex</em> (<em>Cux.</em>) <em>univittatus</em></td>
<td>WNV, SINV</td>
<td>18:00–06:00</td>
<td>Anthropophilic and zoophilic</td>
<td>Exophagic and endophagic</td>
<td>Exophillic and endophillic</td>
</tr>
<tr>
<td><em>Culex</em> (<em>Cux.</em>) <em>quinquefasciatus</em></td>
<td>WNV</td>
<td>18:00–06:00</td>
<td>Primarily anthropophilic and zoophilic</td>
<td>Exophagic and endophagic</td>
<td>Exophillic and endophillic</td>
</tr>
<tr>
<td><em>Culex</em> (<em>Ocu.</em>) <em>bitaeniorhynchus</em></td>
<td>SINV</td>
<td>18:00–06:00</td>
<td>Anthropophilic and zoophilic</td>
<td>Exophagic and endophagic</td>
<td>Exophillic and endophillic</td>
</tr>
<tr>
<td><em>Aedes</em> (<em>Och.</em>) <em>caspius</em></td>
<td>WNV</td>
<td>06:00–18:00</td>
<td>Anthropophilic and zoophilic</td>
<td>Exophagic and endophagic</td>
<td>Exophillic and endophillic</td>
</tr>
<tr>
<td><em>Aedes</em> (<em>Adm.</em>) <em>ochraceus</em></td>
<td>RVFV</td>
<td>No data</td>
<td>Primarily zoophilic but opportunistically anthropophilic</td>
<td>No data</td>
<td>No data</td>
</tr>
</tbody>
</table>

[Back to table of contents](#)
# Bionomics Table: Mosquito Vectors of Kenya

<table>
<thead>
<tr>
<th>Species Name</th>
<th>Vector Status</th>
<th>Biting Times</th>
<th>Host Preference</th>
<th>Feeding Behavior</th>
<th>Resting Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Aedes (Adm.) vexans</em> (Meigen, 1830)</td>
<td>WNV, WEEV, EEEV, SLEV</td>
<td>Night</td>
<td>Anthropophilic and zoo-philic</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td><em>Aedes (Dic.) furcifer</em> (Edwards, 1913)</td>
<td>YFV, DENV-2, CHIKV</td>
<td>No data</td>
<td>Anthropophilic and zoo-philic</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td><em>Aedes (Dic.) taylori</em> Edwards, 1936</td>
<td>YFV, DENV-2, CHIKV</td>
<td>No data</td>
<td>Anthropophilic and zoo-philic</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td><em>Aedes (Stg.) africanus</em> (Theobald, 1901)</td>
<td>YFV, CHIKV, RVFV</td>
<td>Night</td>
<td>Primarily zoophilic but opportunistically anthropophilic</td>
<td>Exophagic and endophagic</td>
<td>No data</td>
</tr>
<tr>
<td><em>Aedes (Neo.) mcintoshi</em> Huang, 1985</td>
<td>RVFV, WSLV, MIDV</td>
<td>Night</td>
<td>Primarily zoophilic but opportunistically anthropophilic</td>
<td>Exophagic</td>
<td>No data</td>
</tr>
<tr>
<td><em>Anopheles (Cel.) funestus</em> Giles, 1900</td>
<td>Malaria</td>
<td>22:00–06:00</td>
<td>Anthropophilic</td>
<td>Endophagic</td>
<td>Endophilic</td>
</tr>
<tr>
<td><em>Anopheles (Cel.) gambi-ae</em> s.l.</td>
<td>Malaria</td>
<td>18:00–06:00</td>
<td>Anthropophilic</td>
<td>Primarily endophagic</td>
<td>Endophilic</td>
</tr>
<tr>
<td><em>Anopheles (Cel.) moucheti</em> Giles, 1923</td>
<td>Malaria</td>
<td>18:00–06:00</td>
<td>Anthropophilic</td>
<td>Endophagic</td>
<td>Endophilic</td>
</tr>
<tr>
<td><em>Anopheles (Cel.) nili</em> s.l.</td>
<td>Malaria</td>
<td>18:00–06:00</td>
<td>Anthropophilic and zoo-philic</td>
<td>Exophagic and endophagic</td>
<td>Endophilic</td>
</tr>
<tr>
<td><em>Anopheles (Cel.) merus</em> Donitz, 1902</td>
<td>Malaria</td>
<td>18:00–06:00</td>
<td>Primarily anthropophilic but opportunistically zoophilic</td>
<td>No data</td>
<td>Exophilic</td>
</tr>
</tbody>
</table>

[Back to table of contents]
## Bionomics Table: Mosquito Vectors of Kenya

<table>
<thead>
<tr>
<th>Species Name</th>
<th>Vector Status</th>
<th>Biting Times</th>
<th>Host Preference</th>
<th>Feeding Behavior</th>
<th>Resting Preference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Anopheles (Cel.) pharoensis s.l.</em></td>
<td>Malaria</td>
<td>18:00–06:00</td>
<td>Primarily zoophilic but opportunistically anthropophilic</td>
<td>No data</td>
<td>No data</td>
</tr>
<tr>
<td><em>Culex (Cux.) theleri</em></td>
<td>RVFV</td>
<td>No data</td>
<td>Anthropophilic and zoophilic</td>
<td>Exophagic and endophagic</td>
<td>No data</td>
</tr>
<tr>
<td>(<em>Theobald, 1903</em>)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Mansonia (Mnd.) uniformis</em></td>
<td>RVFV, <em>Wuchereria malayi</em></td>
<td>19:00–20:00</td>
<td>Primarily zoophilic but opportunistically anthropophilic</td>
<td>Exophagic</td>
<td>No data</td>
</tr>
<tr>
<td>(<em>Theobald, 1901</em>)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*RVFV*: Rift Valley Fever Virus
## Bionomics Table: Tick Vectors of Kenya

<table>
<thead>
<tr>
<th>Species Name</th>
<th>Vector Status</th>
<th>Life Cycle</th>
<th>Host Preference</th>
<th>Oviposition</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Amblyomma variegatum</em> Fabr. 1794</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF)</td>
<td>This species is a three-host tick.</td>
<td>Sheep and cattle, and occasionally humans</td>
<td>No data</td>
</tr>
<tr>
<td><em>Hyalomma dromedarii</em> Koch, 1844</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF)</td>
<td>This species may be either a two- or three-host tick.</td>
<td>Camels, cattle, goats, dogs, small mammals, lizards and occasionally humans</td>
<td>In <em>Hyalomma</em> ticks, the number of eggs laid is variable, ranging from hundreds in rodent burrows to thousands on open ground or vegetation. Eggs usually hatch within 30 days.</td>
</tr>
<tr>
<td><em>Hyalomma truncatum</em> Koch, 1844</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF)</td>
<td>This species is usually a two-host tick that is found in floodplains in semi-deserts and steppes, or vegetated hillsides and mountainsides are preferred habitats.</td>
<td>Cattle, camels and sheep, immature stages tend to parasitize ground-feeding birds</td>
<td>Same as <em>H. dromedarii</em></td>
</tr>
<tr>
<td><em>Rhipicephalus sanguineous</em> (Latreille, 1806)</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF), Boutonneuse Fever</td>
<td>This species is a three-host tick that is prevalent in urban areas because of its close association with dogs.</td>
<td>Dogs but also feeds on camels, gerbils and, occasionally, humans</td>
<td><em>Rhipicephalus</em> sp. ticks, lay hundreds of eggs, generally in the dens of host animals, especially canines.</td>
</tr>
<tr>
<td><em>Haemaphysalis leachi</em> (Audouin, 1826) (Species complex including <em>Ha. elliptica</em> Koch, 1844)</td>
<td>Tick typhus</td>
<td>This species is a three-host tick.</td>
<td>Domestic dogs and wild carnivores</td>
<td><em>Ha. leachi</em> can lay 5000 eggs that hatch within 2 to 9 weeks.</td>
</tr>
<tr>
<td><em>Hyalomma impeltatum</em> Schulze &amp; Schlottke, 1930</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF)</td>
<td>This species is usually a two-host tick that lives in scattered foci of semi-desert, savanna, and steppe biotopes.</td>
<td>Camels, cattle, rodents, hares, ground birds and other large domestic animals</td>
<td>Same as <em>H. dromedarii</em></td>
</tr>
<tr>
<td><em>Rhipicephalus pulchellus</em> (Gerstacker, 1873)</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF), Tick typhus</td>
<td>This species is a three-host tick.</td>
<td>Domestic cattle, sheep, camels, goats, and wild zebras, black rhinoceroses, elands, and gemsbok</td>
<td><em>Rhipicephalus</em> sp. ticks, lay hundreds of eggs, generally in the dens of host animals, especially canines.</td>
</tr>
<tr>
<td><em>Hyalomma rufipes</em> Koch, 1844</td>
<td>Crimea-Congo Hemorrhagic Fever (CCHF), Tick typhus</td>
<td>This species is a two-host tick.</td>
<td>Cattle, sheep, goats, horses and wild ungulates.</td>
<td>Same as <em>H. dromedarii</em></td>
</tr>
</tbody>
</table>

[Back to table of contents]
References


References


References


References


