

Tsetse Flies and African Trypanosomiasis: Biology, Epidemiology, and Integrated Vector Control

Atul Khajuria¹, Mahesh Gaba^{2,*}

Abstract

Tsetse flies (Glossina spp.) are hematophagous dipterans confined to sub-Saharan Africa and serve as cyclical vectors of human African trypanosomiasis (HAT) and African animal trypanosomiasis (AAT), with major implications for human health, livestock productivity, and rural development.¹ An integrated understanding of tsetse biology, parasite transmission, climate- and land-use-driven distribution shifts, and the comparative performance of control tools – especially insecticides and sterile insect technique (SIT) – is essential for designing sustainable elimination strategies. HAT (sleeping sickness) is caused by Trypanosoma brucei gambiense in West and Central Africa and T. b. rhodesiense in East and Southern Africa, transmitted by infected tsetse flies. Historically, HAT has caused devastating epidemics, but coordinated vector control, improved diagnosis, and therapeutics have reduced reported cases to historically low levels, and elimination as a public health problem is now considered achievable. At the same time, AAT caused by T. congolense, T. vivax, and other trypanosomes remains a major constraint on cattle and other livestock, undermining food security and rural economies. From a microbiological perspective, HAT provides a paradigm of extracellular protozoan infection with sophisticated antigenic variation; from a public health perspective, it illustrates the challenges of vector-borne disease control under climate change, land-use transformation, and constrained health systems.

Keywords: African animal trypanosomiasis, glossina, human African trypanosomiasis, sterile insect technique, tsetse fly, vector control

INTRODUCTION

Tsetse flies (*Glossina* spp.) are hematophagous Diptera confined to sub-Saharan Africa and are the cyclical vectors of African trypanosomes causing human African trypanosomiasis (HAT, sleeping sickness) in humans and African animal trypanosomiasis (AAT, nagana) in livestock [1–3]. HAT remains a neglected tropical disease of major public health importance, historically responsible for large epidemics with high mortality in rural African populations. Large-scale control efforts, including improved case detection, safer drugs, and strengthened vector control, have led to a marked decline in reported HAT cases and a transition from epidemic to low-incidence, focal transmission in most endemic countries [2, 3]. In contrast, AAT continues to impose a substantial burden on livestock production through anemia, weight loss, reduced fertility, and death in cattle and other domestic animals, thereby constraining mixed crop–livestock systems and limiting agricultural development [4].

*Author for Correspondence

Mahesh Gaba
E-mail: atulkhajuria83@gmail.com

¹Dean, Allied & Health Care Sciences, Rayat Bahra Professional University, Bohan, Hoshiarpur, Punjab, India

²Faculty, Medical Laboratory Science, PCTE Group of Institutes, Jhanda, Ludhiana, Punjab, India

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The biology of *Glossina*, particularly adenotrophic viviparity, low reproductive rate, and close association with specific habitats and vertebrate hosts, shapes both transmission dynamics and control options [1, 5]. Environmental change – including deforestation, agricultural expansion, and climate warming – is modifying tsetse distribution, potentially shrinking some traditional foci while creating new areas of suitability, especially at higher elevations [6–10]. At the same time, resource and health-system constraints threaten the sustainability of existing gains.

Against this background, an integrated understanding of tsetse taxonomy and ecology, parasite biology, clinical features and management of HAT, and the performance and cost-effectiveness of available control tools – ranging from insecticide-based methods to sterile insect technique within area-wide strategies – is essential for planning long-term elimination and maintaining tsetse-free zones where feasible (Table 1) [1–3, 5, 11].

BIOLOGY AND TAXONOMY OF TSETSE FLIES

Classification and Species Groups

Tsetse flies belong to the family Glossinidae, genus *Glossina*, and are divided into three main species groups: *morsitans* (savanna), *palpalis* (riverine), and *fuscus* (forest). *Palpalis* group species (e.g., *G. palpalis*, *G. fuscipes*) are the principal vectors of *T. b. gambiense*, whereas *morsitans* group species (e.g., *G. morsitans*, *G. pallidipes*) are associated with *T. b. rhodesiense* and important AAT foci [2, 4].

Morphology and Reproductive Biology

Adult tsetse are medium-sized flies (6–15 mm) with a forward-pointing proboscis and wings that fold scissor-like completely over each other at rest [1, 5]. Both sexes are obligate blood feeders on a range of wild and domestic vertebrates. Tsetse is notable for adenotrophic viviparity: females retain a single developing larva in the uterus, nourished by specialized milk glands, and deposit a fully developed third-instar larva, which pupates in soil [5].

Each female produces roughly one larva every 9–10 days, resulting in low reproductive output but high larval survival, a life-history trait that underpins the effectiveness of population-level interventions targeting adult flies [5]. Pupae require several weeks for development, depending on temperature and humidity, before emergence of adults [1, 5].

Symbiotic Microbiota

Tsetse harbor a characteristic microbiota, including the obligate mutualist *Wigglesworthia*, the commensal *Sodalis*, and often the reproductive symbiont *Wolbachia*. *Wigglesworthia* is essential for larval development and adult immunity; its loss leads to reduced fecundity and increased susceptibility to infection. *Sodalis* has been associated in some studies with higher susceptibility to trypanosome infection, suggesting that microbiome manipulation could modulate vector competence [12–14].

Table 1. Key biological and taxonomic features of tsetse flies.

Feature	Description
Taxonomy	Diptera, Glossinidae, <i>Glossina</i> spp. (<i>morsitans</i> , <i>palpalis</i> , <i>fuscus</i> groups).
Distribution	Sub-Saharan Africa, ~7–10 million km ² infested.
Feeding	Both sexes obligate blood feeders on wildlife, livestock, humans.
Reproduction	Adenotrophic viviparity, single larva nourished by milk glands.
Development	Larva in soil → pupa → adult; generation time weeks–months (climate-dependent).
Symbionts	<i>Wigglesworthia</i> , <i>Sodalis</i> , <i>Wolbachia</i> ; influence fecundity and vector competence.

TRYPANOSOMES AND HUMAN AFRICAN TRYPANOSOMIASIS

Parasite Life Cycle and Immune Evasion

Ingested bloodstream trypomastigotes transform to procyclic forms in the tsetse midgut, migrate to the proventriculus and salivary glands, differentiate to epimastigotes, and finally to infective metacyclic trypomastigotes that are inoculated with saliva during subsequent blood meals. In the mammalian host, trypanosomes remain extracellular in blood and lymph, later invading the central nervous system (CNS) and causing meningoencephalitis in late-stage disease [2, 3].

African trypanosomes evade adaptive immunity through antigenic variation of variant surface glycoproteins (VSGs), generating waves of parasitemia as each variant is cleared by host antibodies and replaced by another. This mechanism complicates vaccine development and underlies the chronicity of *T. b. gambiense* infection [15].

Clinical Stages

HAT is classically staged as [2, 3].

- *Stage 1 (Hemolymphatic)*: parasite largely confined to blood and lymph, with intermittent fever, headache, malaise, pruritus, and lymphadenopathy.
- *Stage 2 (Meningoencephalitic)*: CNS involvement with sleep–wake disturbance, neuropsychiatric symptoms, motor and endocrine dysfunction.
- *T. b. gambiense* causes a chronic disease, often progressing over months to years, whereas *T. b. rhodesiense* causes an acute illness with rapid progression to severe systemic and neurological manifestations (Table 2).

Table 2. Comparison of *T. b. gambiense* and *T. b. rhodesiense* HAT.

Parameter	<i>T. b. gambiense</i>	<i>T. b. rhodesiense</i>
Geography	West/Central Africa	East/Southern Africa.
Reservoir	Mainly humans (anthroponotic)	Livestock and wildlife (zoonotic).
Course	Chronic, months–years	Acute, weeks–months.
Stage 1	Intermittent fever, pruritus, posterior cervical lymphadenopathy	High fever, chancre, myocarditis, less marked lymphadenopathy.
Stage 2	Sleep disturbance, behavioral change, endocrine and neurologic signs	Rapid CNS involvement, severe systemic disease, high mortality if untreated.

EPIDEMIOLOGY, CLIMATE, AND LAND-USE-DRIVEN DISTRIBUTION CHANGES

Historical Burden and Current Trends

HAT caused large epidemics in the early and mid-20th century but strengthened surveillance and vector control have reduced reported case numbers dramatically in recent decades. Current hotspots for gambiense HAT are localized foci in Central Africa, notably the Democratic Republic of Congo, whereas rhodesiense HAT is reported from East and Southern African ecosystems with wildlife–livestock–human interfaces [3, 16]. Tsetse infestation covers much of sub-Saharan Africa, but actual disease risk is focal, dictated by local tsetse species, host availability, and health-system performance.

Climate Change and Tsetse Distribution

Tsetse flies are sensitive to temperature and humidity; mechanistic and statistical models show that climate warming can reduce suitability in overheated lowlands while increasing suitability at higher elevations and in some previously cooler areas. In Zimbabwe, a mechanistic model fitted to long-term data suggested marked declines in low-altitude tsetse populations since about 2000 but stable or increasing suitability in cooler, higher-altitude zones [6].

Species distribution and logistic models in Kenya and Tanzania indicate that under warming scenarios, potential tsetse habitat may shift into densely populated highland areas, raising concerns about future overlap between human settlement and vector distribution. Machine-learning approaches also highlight possible contraction in some traditionally endemic areas and emergence of new risk zones where the climate becomes more favorable [7, 8].

Land-Use Change and Habitat Modification

Tsetse requires suitable woody vegetation, shade, and hosts; extensive clearing for agriculture and settlement fragments or destroys this habitat, causing local declines in fly density [9]. In Lambwe Valley (Kenya), tsetse control and subsequent expansion of cultivation around a protected area led to sharp reductions of tsetse and near disappearance of sleeping sickness from settled zones, though flies persisted within the undisturbed park (Table 3).

Across several countries, conversion of woodland to cropland and overhunting of wildlife have been linked to contraction of tsetse range, particularly for savanna species [9, 10]. At the same time, reforestation, bush encroachment, or protected wildlife areas can maintain or recreate suitable habitat despite regional warming, producing a mosaic of shrinking and emerging foci [6, 9, 10].

Table 3. Climate and land-use influences on tsetse distribution.

Driver	Effect on tsetse	Examples
Rising temperature	Reduced suitability in hottest lowlands; expansion into cooler highlands	Declining low-altitude tsetse; increased suitability above ~1000 m.
Rainfall/vegetation	Moderate rainfall and woody cover favor tsetse; aridity/deforestation reduce habitat	Landscape–climate models in Kenya.
Agricultural expansion	Cropland replaces woodland and wildlife, lowering tsetse density	Lambwe Valley gradients of clearing.
Protected areas	Maintain habitat and hosts, allowing persistent foci	Wildlife parks, riparian corridors.

DIAGNOSIS AND TREATMENT OF HAT

Diagnosis

Parasitological confirmation remains central. In gambiense HAT, low parasitemia necessitates concentration methods such as microhematocrit centrifugation, mini–anion exchange chromatography, and buffy-coat examination [17]. In Rhodesiense HAT, higher parasite loads often allow direct microscopy of blood films or lymph node aspirates. Cerebrospinal fluid examination for trypanosomes and white-cell count is required for staging, which guides therapy [3].

Serologic screening (card agglutination tests and rapid diagnostic tests) is widely used for gambiense HAT to select suspects for confirmatory parasitology, especially in mobile screening campaigns. Molecular tools, such as PCR offer high sensitivity and subspecies differentiation, particularly valuable in research and for surveillance, but remain limited in routine field use [15, 17].

Treatment

Historically, suramin and pentamidine were used for early-stage disease, and melarsoprol, an arsenic-based drug with serious toxicity, for late-stage disease. For gambiense HAT, nifurtimox–eflornithine combination therapy (NECT) and an all-oral fexinidazole regimen now provide safer, more practical alternatives, especially for late-stage disease. Fexinidazole facilitates outpatient treatment in selected patients and eases logistical constraints in remote settings. For rhodesiense HAT, suramin remains recommended for early stages, with melarsoprol still widely used for CNS involvement owing to lack of validated alternatives; new oral regimens remain a research priority [3, 11].

TSETSE CONTROL STRATEGIES

Insecticide-Based Methods

Conventional tools include odor-baited traps and insecticide-treated cloth targets, which exploit tsetse host-seeking behavior and visual attraction to reduce adult populations [5, 8]. Insecticide-treated cattle act as live baits; flies are killed while feeding, reducing transmission of both HAT and AAT. Aerial spraying (sequential aerosol technique) can rapidly suppress tsetse in certain habitats.

Costing work in Uganda estimated approximate control costs per km² for isolated populations as lowest for insecticide-treated cattle, moderate for traps/targets, and higher for aerial spraying, with variation by ecology and logistics. Benefit–cost analyses of insecticide-treated cattle showed favorable benefit–cost ratios, highlighting strong returns in livestock-oriented settings.

Sterile Insect Technique (SIT)

SIT is an autocidal method in which mass-reared male tsetse are sterilized with ionizing radiation and released over target areas so that wild females mate with sterile males and produce no viable offspring. Because females typically mate only once per reproductive cycle and tsetse reproduction is slow, repeated releases of competitive sterile males over several generations can drive the wild population towards elimination, particularly when initial densities are already low [18, 19].

SIT is most effective as part of area-wide integrated pest management (AWIPM): conventional insecticide-based suppression reduces populations to low levels, after which SIT is used to push towards

local eradication and maintain tsetse-free status [19]. The Unguja (Zanzibar) project, which combined suppression with SIT, achieved eradication of *Glossina austeni* and durable elimination of AAT on the island, illustrating the potential of this approach under favorable ecological and managerial conditions (Table 4) [19].

Table 4. Comparison of major tsetse control tools.

Tool	Mode of action	Advantages	Limitations
Traps/targets	Visual/odor attraction + insecticide	Species-specific, relatively low cost, community-run	Maintenance-dependent; less efficient at very low density.
Insecticide-treated cattle	Flies killed while feeding on treated animals	Controls tsetse and ticks; high benefit–cost in livestock areas	Requires cattle, compliance; resistance risk.
Aerial spraying (SAT)	ULV insecticide sprayed over large areas	Rapid suppression over extensive zones	Higher cost; environmental/operational constraints.
SIT	Release of sterilized males	Species-specific, non-polluting, powerful at low density	High technical and capital requirements; best in isolated populations.

Cost-Effectiveness of SIT Versus Insecticides

Economic analyses indicate that insecticide-based approaches are generally more cost-effective for initial control and suppression, especially when tsetse densities are high, because costs scale primarily with area rather than population size [20, 21]. In contrast, SIT tends to be more expensive upfront but can become cost-effective, or even economically superior, when applied to already suppressed, low-density and ecologically isolated populations with elimination as the explicit goal [19, 21].

A comparative costing study for Uganda concluded that, for short- to medium-term horizons, purely insecticide-based elimination (e.g., aerial spraying with or without targets) could be cheaper in some scenarios; however, over longer horizons, once maintenance, environmental impacts, and import-dependent insecticide supply were factored in, inclusion of SIT within an AW-IPM strategy could provide better economic performance in suitable areas [20, 21].

Control and suppression programs in large endemic areas, therefore, rely mainly on insecticide-based tools, whereas elimination programs in ecologically isolated or well-delimited populations may benefit from phased strategies that integrate suppression with SIT despite higher technical demands [19, 21, 22].

Sustaining Tsetse-Free Zones: Operational and Public Health Challenges

Sustaining tsetse-free zones is feasible but requires ecological isolation, robust surveillance, and enduring political and financial commitment. Island or ecologically isolated populations, such as Unguja or Senegal’s Niayes region, offer favorable conditions because reinvasion risk is low and barrier systems are easier to maintain. On the mainland, tsetse populations are often connected via riverine or wildlife corridors, so maintaining tsetse-free status demands continuous monitoring and peripheral “barriers” using traps, targets, or insecticide-treated animals [23–27].

Post-eradication land-use change presents both opportunity and risk. Removal of trypanosomiasis risk permits expansion of livestock and cultivation, but unregulated agricultural expansion, deforestation, and overgrazing can degrade natural resources and alter wildlife communities. For planners, tsetse control should be embedded within broader land-use planning and environmental safeguards to avoid long-term ecological damage [10, 23, 28–30].

Social and behavioral factors also matter. As disease incidence declines, communities and funders may perceive the problem as solved, leading to reduced support for maintaining traps, targets, treated cattle, and surveillance [31–34]. Migration into newly “safe” areas can increase human and livestock densities without concurrent strengthening of health and veterinary services, increasing vulnerability should tsetse re-invent (Table 5).

Table 5. Key challenges in sustaining tsetse-free zones.

Domain	Challenges
Ecological	Reinvasion via rivers and wildlife corridors; connected mainland populations.
Operational	Need for long-term entomological and disease surveillance; maintaining barriers; high technical requirements for AW-IPM and SIT.
Economic	Declining funding post-success; high fixed costs for mass-rearing and monitoring; reliance on imported insecticides.
Land use	Post-control agricultural expansion, deforestation, overgrazing; risk of degradation.
Social/behavioral	Reduced community engagement once disease declines; unmanaged migration into former risk areas.

CONCLUSION

Tsetse flies (*Glossina* spp.) are hematophagous Diptera confined to sub-Saharan Africa and are the cyclical vectors of African trypanosomes causing human African trypanosomiasis (HAT, sleeping sickness) in humans and African animal trypanosomiasis (AAT, nagana) in livestock. HAT remains a neglected tropical disease of major public health importance, historically responsible for large epidemics with high mortality in rural African populations. Large-scale control efforts, including improved case detection, safer drugs, and strengthened vector control, have led to a marked decline in reported HAT cases and a transition from epidemic to low-incidence, focal transmission in most endemic countries. In contrast, AAT continues to impose a substantial burden on livestock production through anemia, weight loss, reduced fertility, and death in cattle and other domestic animals, thereby constraining mixed crop–livestock systems and limiting agricultural development.

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