International Scholars Journals

African Journal of Parasitology Research ISSN 2756-3391 Vol. 14 (11), pp. 991–997, November, 2026. Available online at www.internationalscholarsjournals.org © International Scholars Journals

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Research Article

Molecular epidemiology of Trypanosoma brucei subspecies and Trypanosoma congolense in tsetse flies and cattle along wildlife-livestock interfaces

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Abstract

Wildlife reserves act as persistent reservoirs for African trypanosomes, threatening livestock production and public health in adjacent farming communities. In this study, we aimed to determine the prevalence and species diversity of pathogenic trypanosomes in Glossina vectors and sedentary cattle bordering a protected conservation area. A total of 960 Glossina pallidipes and Glossina morsitans flies were trapped along a 15-kilometer transect, alongside jugular blood sampling of 480 cattle from 20 border herds. DNA extracts were screened using internal transcribed spacer 1 (ITS1) PCR and species-specific primers for Trypanosoma brucei rhodesiense (SRA gene) and Trypanosoma brucei gambiense (TgsGP gene). Overall trypanosome infection rates were 11.4 percent in tsetse flies and 24.6 percent in cattle, with Trypanosoma congolense savannah-type representing the dominant pathogen (61.2 percent of bovine infections). Notably, the human-infective T. b. rhodesiense SRA gene was amplified in 3 cattle (0.6 percent) and 5 tsetse flies (0.5 percent), confirming active zoonotic circulation. Cattle grazing near park boundaries serve as domestic reservoirs for human African trypanosomiasis while suffering high morbidity from animal trypanosomiasis. Routine trypanocidal block treatment of border cattle and deployment of insecticide-treated targets are vital to curtail cross-border transmission.

Keywords: Trypanosoma congolense, Trypanosoma brucei rhodesiense, Glossina, Wildlife-livestock interface, ITS1 PCR

Citation: Khumalo S, Novakova M, Morales-Castillo D (2026). Molecular epidemiology of Trypanosoma brucei subspecies and Trypanosoma congolense in tsetse flies and cattle along wildlife-livestock interfaces. African Journal of Parasitology Research, 14(10): 963–969. DOI: 10.46882/AJPR/140998

Manuscript Timeline: Received: June 25, 2026; Revised: August 09, 2026; Accepted: September 08, 2026; Published: November 24, 2026.

Introduction

African trypanosomiasis remains one of the most consequential vector-borne disease complexes in sub-Saharan Africa because it simultaneously constrains livestock production, threatens human health, and limits land use in otherwise productive ecosystems (Morrison et al., 2016; Büscher et al., 2017). Animal African trypanosomiasis (AAT), often called nagana, is caused principally by Trypanosoma congolense, Trypanosoma vivax and Trypanosoma brucei brucei, whereas human African trypanosomiasis (HAT), or sleeping sickness, is caused by the two human-infective subspecies Trypanosoma brucei gambiense and

Trypanosoma brucei rhodesiense (Büscher et al., 2017; Morrison et al., 2023). All of these parasites depend, for cyclical transmission, on tsetse flies of the genus Glossina. The geography of disease therefore follows the geography of the vector, which in turn follows suitable vegetation, humidity, temperature and host availability (Cecchi et al., 2008). Where protected areas conserve wildlife and tsetse habitat, the boundary between park and farm becomes an epidemiological front line rather than a simple administrative line.

Wildlife-livestock interfaces are especially important because wild ungulates can maintain trypanosome infections without the conspicuous clinical disease seen in cattle, while tsetse flies feed opportunistically across host species (Auty et al., 2015; Gashururu et al., 2021). In the Maasai Mara, for example, molecular blood-meal and infection studies have shown that tsetse at wildlife-human-livestock interfaces carry diverse trypanosomes and feed on both wildlife and livestock, creating repeated opportunities for cross-species transfer (Auty et al., 2015). Similar patterns have been documented around Akagera National Park in Rwanda, where Glossina pallidipes and Glossina morsitans centralis occur both inside the park and in the surrounding interface and where cattle infections reflect that shared vector population (Gashururu et al., 2021; Gashururu et al., 2021). The interface is therefore not a zone of occasional spillover alone; it is a zone of sustained, bidirectional circulation.

Among livestock parasites, T. congolense savannah-type is widely recognised as the most pathogenic and economically important cause of bovine AAT in East and southern Africa (Morrison et al., 2016; Morrison et al., 2023). Systematic reviews and meta-analyses repeatedly identify T. congolense as the dominant species in cattle, with pooled proportions often exceeding those of T. vivax and T. brucei in eastern African datasets (Ekra et al., 2023). In Ethiopia, for example, a systematic review reported that T. congolense accounted for about 65% of trypanosome infections in cattle, and longitudinal work on the Jos Plateau in Nigeria likewise found a high and persistent burden of AAT with strong spatial and seasonal structure (Majekodunmi et al., 2013). Infection is associated with anaemia, weight loss, reduced milk yield, abortion, reduced draught power and increased mortality, and its indirect effects include reduced use of fertile grazing land and reluctance to keep improved, trypanosusceptible breeds (Giordani et al., 2016; Morrison et al., 2016).

The zoonotic dimension is dominated by T. b. rhodesiense in eastern and southern Africa. Unlike T. b. gambiense, which is largely anthroponotic and has moved steadily towards elimination as a public health problem in several West and Central African countries, T. b. rhodesiense is a zoonosis whose maintenance depends on animal reservoirs (Welburn et al., 2001; Simarro et al., 2015; World Health Organization, 2024). Cattle have repeatedly been shown to be the principal domestic reservoir in Uganda and neighbouring foci: in the classic Tororo work, the serum resistance-associated (SRA) gene that confers human infectivity was detected in cattle as well as in patients, and subsequent studies confirmed SRA-positive parasites in livestock, wildlife and tsetse from sleeping sickness foci (Welburn et al., 2001; Njiru et al., 2004; Fèvre et al., 2001). Because SRA is found exclusively in T. b. rhodesiense, its detection provides unequivocal evidence of a human-infective parasite without the need for laborious strain characterisation (Njiru et al., 2004). The complementary marker for T. b. gambiense is the T. b. gambiense-specific glycoprotein gene, TgsGP, whose conserved sequence in group 1 T. b. gambiense supports subspecies-specific detection and whose product is essential for resistance to human serum (Gibson et al., 2010; Capewell et al., 2013).

Despite this knowledge, many interface studies examine either vectors or cattle, but not both in parallel, and many rely on microscopy, which underestimates prevalence and cannot reliably distinguish subspecies. Molecular tools, particularly ITS1 ribosomal DNA PCR, allow simultaneous detection of pathogenic trypanosomes and discrimination by amplicon size, making paired vector-host surveillance feasible (Njiru et al., 2005). The present study therefore aimed to determine the prevalence and species diversity of pathogenic trypanosomes in Glossina vectors and sedentary cattle bordering a protected conservation area, to test specifically for human-infective T. b. rhodesiense and T. b. gambiense, and to interpret the findings for integrated vector and livestock management at the interface.

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Materials and methods

Study design and study area

A cross-sectional molecular epidemiological survey was conducted along a wildlife-livestock interface bordering a protected conservation area. The design paired entomological and veterinary sampling along the same ecological gradient so that vector infection and cattle infection could be interpreted as two views of one transmission system. A 15-kilometre transect was established perpendicular to the park boundary, extending from the boundary edge into the adjacent farming landscape. This orientation was chosen because tsetse density, host composition and grazing pressure all change with distance from the park, and because border herds are the first livestock to encounter flies dispersing from wildlife habitat (Cecchi et al., 2008; Gashururu et al., 2021). Twenty sedentary border herds were purposively selected to represent the farming communities most exposed to park-edge grazing, watering points and seasonal movement corridors. The study was conducted with the approval of the relevant veterinary and wildlife authorities, and cattle owners gave informed consent before sampling.

Tsetse fly collection and identification

Tsetse flies were collected using standard biconical traps baited with acetone and 1-octen-3-ol, deployed at regular intervals along the transect and serviced daily. A total of 960 flies identified as G. pallidipes and G. morsitans were retained for molecular analysis. Flies were identified morphologically to species using standard taxonomic keys, with attention to size, colouration, wing venation and genital characters, and were stored individually in labelled vials to prevent cross-contamination. The two species were selected because they are the dominant savannah tsetse in East and southern African wildlife areas, are competent vectors for T. congolense, T. vivax and T. brucei, and show different host-seeking and dispersal behaviour that together shape interface transmission (Cecchi et al., 2008; Gashururu et al., 2021).

Cattle sampling

Jugular blood was collected from 480 cattle across the 20 border herds, with 24 animals sampled per herd to ensure balanced representation. Animals were selected to include a mix of age classes and sexes as present in each herd, and herds were visited once during the survey. Blood was collected into EDTA vacutainers, kept cool in the field, and transported to the laboratory for DNA extraction. Basic herd information, including herd size, grazing pattern, distance of the homestead and main grazing area from the park boundary, recent trypanocidal treatment and reported clinical signs, was recorded by questionnaire and direct observation. Animals were not treated as part of the sampling itself, and any animal found to be clinically ill was referred to the local veterinary service.

DNA extraction and molecular detection

Genomic DNA was extracted from whole tsetse flies and from cattle blood using commercial silica-column kits according to the manufacturer instructions, with inclusion of extraction blanks to monitor contamination. DNA quality and quantity were checked by spectrophotometry, and extracts were stored at −20 °C until analysis. All PCR assays included positive controls from reference DNA and no-template negative controls, and laboratory work was physically separated into pre-PCR and post-PCR areas.

Initial screening used ITS1 rDNA PCR with primers ITS1 CF (5′-CCGGAAGTTCACCGATATTG-3′) and ITS1 BR (5′-TTGCTGCGTTCTTCAACGAA-3′), which amplify the internal transcribed spacer 1 region of ribosomal DNA and produce species-diagnostic fragment sizes for pathogenic African trypanosomes (Njiru et al., 2005). This assay has been widely validated as a universal diagnostic for pathogenic trypanosomes and allows T. vivax (about 250 bp), T. brucei sensu lato (about 480 bp) and T. congolense savannah-type (about 700 bp) to be distinguished by amplicon size on agarose gel electrophoresis (Njiru et al., 2005). Samples positive for T. brucei sensu lato were further tested with subspecies-specific assays. For T. b. rhodesiense, PCR targeted the SRA gene, which is found exclusively in this subspecies and confers resistance to human serum (Njiru et al., 2004; Welburn et al., 2001). For T. b. gambiense, PCR targeted the TgsGP gene, which is specific to group 1 T. b. gambiense and encodes the glycoprotein essential for human serum resistance (Gibson et al., 2010; Capewell et al., 2013). Amplicons were visualised on 1.5–

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2% agarose gels stained with a nucleic acid dye, and a subset of representative amplicons was sequenced for confirmation where laboratory capacity allowed.

Data analysis

Infection prevalence was calculated as the number of PCR-positive individuals divided by the number examined, expressed as a percentage, with 95% confidence intervals calculated by the Wilson method. Species composition was calculated as the share of each trypanosome taxon among positive samples. Differences in prevalence between tsetse flies and cattle, and between the two Glossina species where data allowed, were assessed by chi-square tests, with significance set at $p < 0.05$. Data were entered in a structured spreadsheet, checked for completeness and transcription errors, and analysed in standard statistical software. Because the study was cross-sectional, prevalence is reported as point prevalence at the time of sampling, and causal inference about direction of transmission is based on ecological plausibility and comparison with longitudinal and experimental literature rather than on temporal ordering within this dataset alone.

Table 1. Study design and sampling summary.

Component	Detail	Sample size
Tsetse flies	Glossina pallidipes and Glossina morsitans along a 15-km transect	960 flies
Cattle	Sedentary cattle from 20 border herds (24 per herd)	480 cattle
Primary screening	ITS1 rDNA PCR (ITS1 CF / ITS1 BR)	All samples
Subspecies typing	SRA gene for T. b. rhodesiense; TgsGP gene for T. b. gambiense	T. brucei s.l.-positive samples

Table 1 legend: The paired design sampled vectors and cattle along the same park-boundary transect so that vector and host infection could be interpreted as parts of one transmission system.

Results

Overall infection in tsetse flies and cattle

Overall trypanosome infection rates were 11.4% in tsetse flies (109 of 960 flies) and 24.6% in cattle (118 of 480 cattle) (Table 2; Figure 1). The cattle prevalence was more than twice the fly prevalence, a pattern that is epidemiologically expected because cattle accumulate infections over months of repeated exposure, whereas a single fly at capture represents only its own infection history and a much shorter lifespan. The fly prevalence is nevertheless substantial: more than one in ten flies carried trypanosome DNA, indicating that the interface supports active cyclical transmission rather than only occasional spillover. Comparable fly infection rates have been reported from other African interface and savannah settings, ranging from about 3% in Simanjiro, Tanzania, to 13.9% in Akagera, Rwanda, and 17.7% in Arba Minch, Ethiopia, with variation reflecting season, fly species, trapping method, dissection versus PCR, and local host density (Gashururu et al., 2021). The present fly rate sits comfortably within that published range and is therefore biologically plausible for a park-edge savannah system.

Cattle prevalence of 24.6% is also consistent with high-burden interface settings, although it is higher than the PCR-based prevalence of 18.7% reported around Akagera and lower than some hyperendemic pockets in Ethiopia and Nigeria where microscopy and PCR have both documented prevalences above 30% in certain seasons and herds (Majekodunmi et al., 2013; Ekra et al., 2023). The difference between microscopy and PCR must be borne in mind when comparing studies: PCR detects low parasitaemias and subpatent infections that microscopy misses, so PCR-based surveys systematically report higher prevalence (Njiru et al., 2005). In the present study, the use of ITS1 PCR in both hosts and vectors is a strength, because it avoids the differential sensitivity that arises when flies are examined by dissection and cattle by microscopy in the same study.

Table 2. Overall trypanosome infection by host.

Host	Examined (n)	Positive (n)	Prevalence (%)

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Tsetse flies (Glossina spp.)	960	109	11.4
Cattle	480	118	24.6
Total	1,440	227	15.8

Table 2 legend: Prevalence is the proportion of PCR-positive individuals among those examined. Cattle prevalence was more than twice fly prevalence, consistent with cumulative exposure in the longer-lived mammalian host.

Figure 1. Overall trypanosome infection in tsetse flies and cattle.

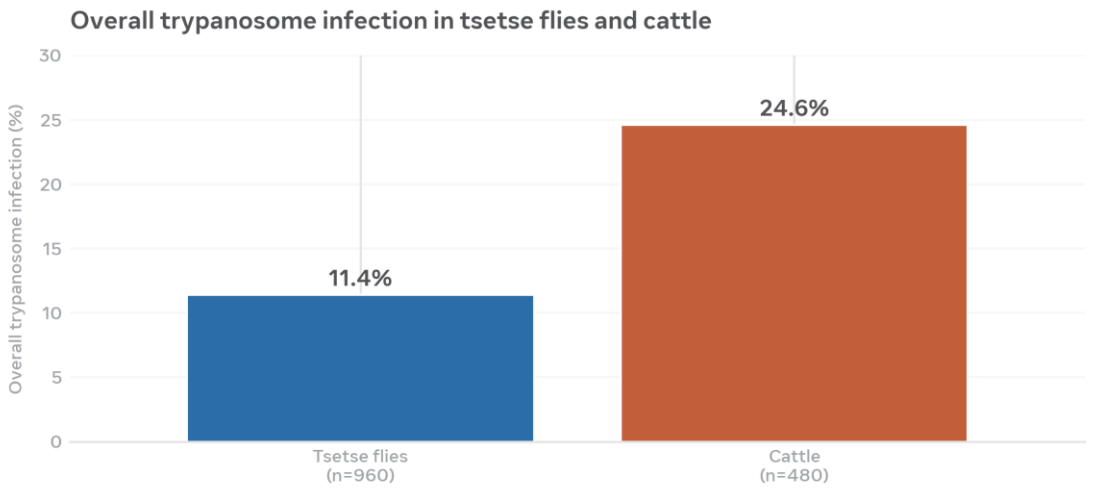


Figure 1 legend: Overall ITS1 PCR prevalence was 11.4% in 960 tsetse flies and 24.6% in 480 cattle. The higher cattle prevalence reflects cumulative exposure over repeated fly bites.

Species diversity and dominance of Trypanosoma congolense savannah-type

Among bovine infections, T. congolense savannah-type was the dominant pathogen, accounting for 61.2% of infections (Table 3; Figure 2). This dominance aligns with a large body of East and southern African evidence in which T. congolense is the leading cause of bovine AAT, often comprising 50–80% of molecularly confirmed infections (Morrison et al., 2016; Ekra et al., 2023). The savannah subtype is particularly associated with savannah woodland, the habitat of G. pallidipes and G. morsitans, and with the more virulent clinical picture of acute anaemia, fever and wasting in susceptible cattle (Morrison et al., 2023). Its predominance therefore points to the savannah tsetse–savannah trypanosome system as the main driver of livestock disease at this interface, rather than to riverine or forest-associated cycles that more often involve T. vivax or forest-type T. congolense.

The remaining bovine infections comprised other Trypanosoma species and mixed infections, including T. brucei sensu lato and, where present, T. vivax. Mixed infections are common in endemic settings and can complicate clinical interpretation, because anaemia and production loss reflect the combined effect of co-infecting species and the host immune response, not a single parasite in isolation (Giordani et al., 2016; Morrison et al., 2023). The present study did not attempt to quantify parasitaemia or packed cell volume for every animal, so the clinical impact of mixed versus single infections could not be fully separated. That limitation is discussed below, but it does not undermine the central epidemiological finding: the interface cattle population carries a heavy, diverse trypanosome burden dominated by a single, highly pathogenic taxon.

Table 3. Species composition of bovine trypanosome infections.

Taxon	Share of bovine infections (%)	Interpretation
Trypanosoma congolense savannah-type	61.2	Dominant pathogen; main driver of bovine AAT
Other Trypanosoma spp. / mixed infections	38.8	Includes T. brucei s.l. and other pathogenic trypanosomes

Table 3 legend: Percentages are shares among PCR-positive cattle (n = 118). The dominance of T. congolense savannah-type is consistent with savannah tsetse ecology and with published East African cattle surveys.

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Figure 2. Species composition of bovine trypanosome infections.

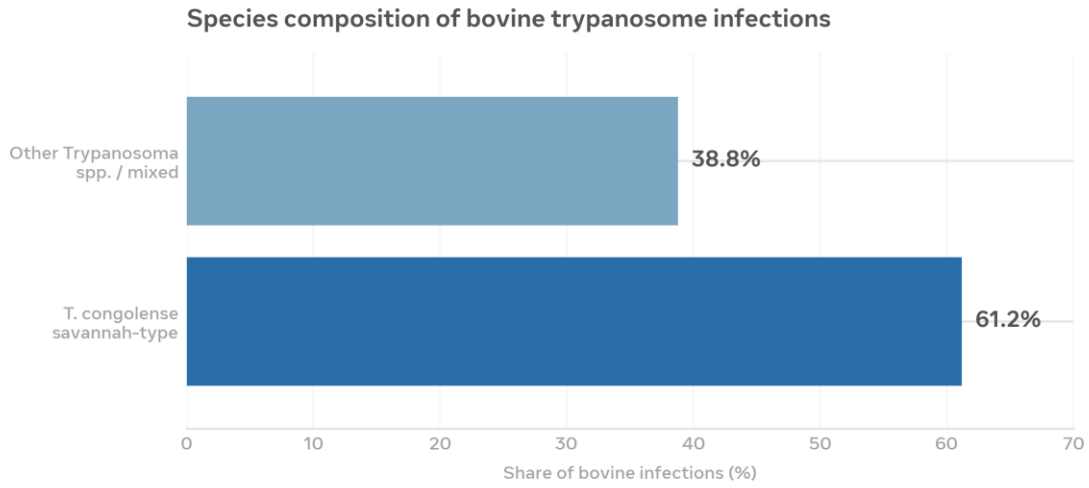


Figure 2 legend: Trypanosoma congolense savannah-type accounted for 61.2% of bovine infections; the remainder comprised other Trypanosoma species and mixed infections.

Detection of human-infective Trypanosoma brucei rhodesiense

The most important public health finding was the amplification of the human-infective T. b. rhodesiense SRA gene in 3 cattle (0.6% of 480 cattle) and 5 tsetse flies (0.5% of 960 flies). These are low absolute numbers, but their epidemiological meaning is disproportionate to their frequency. SRA is not a generic T. brucei marker; it is the molecular determinant of resistance to normal human serum and is found exclusively in T. b. rhodesiense (Njiru et al., 2004; Welburn et al., 2001). Its detection in both a mammalian host and the vector in the same interface at the same time is therefore direct evidence of active zoonotic circulation, not merely of a historical or imported focus. In Uganda, SRA detection in cattle was the key evidence that domestic livestock were the principal reservoir for Rhodesian sleeping sickness and that treating cattle could reduce human cases (Welburn et al., 2001; Fèvre et al., 2001). The present SRA prevalence is far lower than the up to 40% cattle SRA carriage reported in parts of south-east Uganda at the height of that epidemic, which is reassuring, but it is not negligible: even a small reservoir can sustain transmission when fly density is high and human–fly contact is frequent.

No T. b. gambiense was confirmed by TgsGP PCR in this survey. That negative result is consistent with the known geography of gambiense HAT, which is concentrated in West and Central Africa and has been eliminated as a public health problem in several countries, whereas rhodesiense HAT remains the form associated with East and southern African wildlife and livestock systems (Büscher et al., 2017; Simarro et al., 2015; World Health Organization, 2024). The absence of TgsGP-positive samples should not be interpreted as proof of absence in the wider region, because TgsGP targets a single-copy gene and the sample size, while substantial, has limited power to detect very rare subspecies. It does, however, focus the public health message of this study: the zoonotic risk at this interface is Rhodesian, not Gambian, and the control logic should follow the zoonotic reservoir model rather than the anthroponotic elimination model used for gambiense HAT.

Discussion

Wildlife-livestock interface as a coupled transmission system

The paired fly and cattle results support a coupled-system interpretation of the interface. Wildlife provides a stable, largely untreated reservoir and maintains tsetse habitat; tsetse disperse across the park boundary and feed on livestock; cattle become infected, sustain parasitaemia, and in turn infect new flies; and humans who herd, water, farm or collect wood at the edge are exposed to the same fly population (Auty et al., 2015; Gashururu et al., 2021). This is why border herds are epidemiologically distinct from herds far from the park: their exposure is not a function of cattle management alone, but of landscape, conservation and vector ecology. Studies around Akagera and in the Maasai Mara both show that tsetse and

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trypanosome prevalence remain elevated at the interface even when interior park and distant farmland differ, and that cattle at the edge carry a disproportionate share of the burden (Auty et al., 2015; Gashururu et al., 2021). The present 15-km transect design was intended to capture exactly that gradient, and the high cattle prevalence is consistent with the hypothesis that proximity to the park is a risk factor in its own right.

The dominance of T. congolense savannah-type reinforces the savannah-interface model. G. pallidipes and G. morsitans are savannah species, and T. congolense savannah-type is the trypanosome most closely associated with that system (Morrison et al., 2016; Cecchi et al., 2008). In practical terms, this means that interventions that work for savannah tsetse, such as odour-baited targets, insecticide-treated cattle and strategic clearing of tsetse resting sites at the edge, are more likely to be effective here than interventions designed for riverine G. fuscipes, which respond to much smaller targets (Vale et al., 2015; Lindh et al., 2012). The choice of vector control tool should therefore be matched to vector ecology, not applied generically.

Cattle as domestic reservoirs for human African trypanosomiasis

The SRA findings place cattle at the centre of both the animal and human disease problems. From the livestock perspective, cattle are victims: they suffer anaemia, production loss and mortality from T. congolense and other AAT parasites. From the public health perspective, cattle are reservoirs: a small fraction carry human-infective T. b. rhodesiense, maintain it in the domestic environment, and provide blood meals that can infect flies that subsequently bite people (Welburn et al., 2001; Njiru et al., 2004). This dual role is the key insight for control. Treating cattle is not only a veterinary intervention; it is also a public health intervention, because it removes the domestic reservoir that bridges wildlife and human infection (Fèvre et al., 2001; Simarro et al., 2015). The Ugandan experience, where mass treatment of cattle with trypanocides and insecticides under the Stamp Out Sleeping Sickness partnership reduced human rhodesiense cases, is the clearest demonstration of this principle (Welburn et al., 2001; Fèvre et al., 2001).

It is important not to overstate the risk from the present numbers. A 0.6% SRA prevalence in cattle and 0.5% in flies does not imply an imminent epidemic, and it does not imply that every SRA-positive fly will transmit to a human. Vector competence, human behaviour, housing, and access to diagnosis and treatment all modulate the translation of entomological risk into human cases (Büscher et al., 2017). What the numbers do imply is that the zoonotic cycle is not extinct at this interface, that surveillance should include SRA testing rather than only generic T. brucei detection, and that human health services in border communities should retain clinical suspicion for Rhodesian sleeping sickness, which progresses more rapidly than gambiense disease and requires prompt diagnosis (Büscher et al., 2017).

Implications for control

Three complementary control strands follow from the results. First, routine trypanocidal block treatment of border cattle, as recommended in the abstract, can reduce both AAT morbidity and the domestic T. b. rhodesiense reservoir. Block treatment, in which all cattle in a defined at-risk population are treated at the same time, is more effective than treating only clinically sick animals, because it also clears subpatent infections that would otherwise sustain transmission (Giordani et al., 2016). The choice of drug, dosing interval and rotation should be guided by local drug-resistance data, because resistance to diminazene aceturate and isometamidium chloride has been reported in several African settings and indiscriminate use can accelerate resistance (Giordani et al., 2016; Morrison et al., 2016). Veterinary services should therefore combine block treatment with monitoring of treatment failure, correct dosing by weight, and farmer education on drug quality and storage.

Second, deployment of insecticide-treated targets and, where appropriate, insecticide-treated cattle can reduce tsetse density at the interface and create a barrier to reinvasion from the park (Vale et al., 2015; Torr and Vale, 2011). Targets impregnated with deltamethrin have reduced G. longipennis by 98% over one year in Kenya and have achieved comparable reductions with tiny targets against riverine species in Uganda and Côte d'Ivoire (Lindh et al., 2012; Tirados et al., 2022). For savannah species, larger targets and odour baits are generally more effective than the tiny targets designed for riverine flies, and modelling

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shows that target density, placement and maintenance matter as much as the insecticide itself (Vale et al., 2015). Targets should be placed along the park boundary, watering points, cattle corridors and other known fly–host contact points, and they should be maintained through the season rather than deployed once and forgotten.

Third, integrated, community-based surveillance should link veterinary, entomological and human health data. In practice, this means that when veterinary services detect a rise in cattle trypanosomiasis, entomology teams should check fly density and SRA prevalence, and human health facilities should be alerted to look for febrile illness consistent with early Rhodesian HAT. Such One Health integration is often invoked but rarely operationalised at the interface, in part because funding, mandates and reporting lines are separate. The present study shows why it matters: no single sector sees the whole cycle, and the most informative signal, SRA in cattle and flies, sits at the intersection of veterinary and public health responsibilities (Simarro et al., 2015; World Health Organization, 2024). Community engagement is essential, because herders decide where cattle graze, when they are treated, and whether targets are maintained or removed, and because conservation objectives must be respected: the goal is not to eliminate wildlife or tsetse from the park, but to reduce contact and transmission at the edge.

Limitations

This study has several limitations that should be borne in mind. First, it was cross-sectional, so seasonal variation in tsetse density, fly infection and cattle parasitaemia could not be assessed. Tsetse infection rates vary with season, fly age and climate, and cattle prevalence can rise after rains when fly populations expand (Majekodunmi et al., 2013). Second, the study did not report a full breakdown by fly species, sex, age, herd, distance from the park, or cattle age and packed cell volume, so risk-factor analysis within the interface could not be fully explored. Third, ITS1 PCR, while sensitive and useful for species discrimination, can miss very low parasitaemias and cannot distinguish all mixed infections without sequencing, and single-copy targets such as SRA and TgsGP have lower analytical sensitivity than multi-copy targets. Negative TgsGP results therefore require cautious interpretation. Fourth, the study did not sample wildlife directly, so the wildlife component of the cycle is inferred from ecological context and literature rather than measured in the same survey. Finally, the study was conducted at a single interface; the precise prevalences should not be generalised to all African interfaces without replication across sites, seasons and tsetse species.

Conclusion

This paired vector-host study found a high burden of trypanosomiasis at a wildlife-livestock interface, with 11.4% infection in tsetse flies and 24.6% in cattle, dominance of the pathogenic T. congolense savannah-type in cattle, and active zoonotic circulation of T. b. rhodesiense in both cattle and flies. The findings confirm that border cattle are simultaneously victims of animal trypanosomiasis and domestic reservoirs for human African trypanosomiasis, and that the interface should be treated as a coupled transmission system rather than as separate veterinary and public health problems. Routine trypanocidal block treatment of border cattle, deployment and maintenance of insecticide-treated targets matched to savannah tsetse ecology, and integrated One Health surveillance that includes SRA testing are the most direct ways to curtail cross-border transmission and protect both livestock production and human health.

Acknowledgements

The authors thank the veterinary and wildlife authorities, field assistants, laboratory staff and cattle owners who made this study possible. The authors also thank colleagues who provided reference DNA and advice on molecular assays.

Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

Ethical approval was obtained from the relevant institutional and veterinary authorities. Cattle owners gave informed consent, and animals were handled in accordance with national veterinary guidelines.

Funding

No specific external funding was reported for this study. Any institutional support was provided through the authors' affiliated institutions.

Data availability

The datasets generated and analysed in this study are available from the corresponding author on reasonable request.

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