



Article

Blood Parasite Diversity and Zoonotic Risk in Captive Sun-Tailed Monkeys from Gabon

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Abstract: The present study investigates the prevalence and diversity of *Plasmodium* and *Trypanosoma* infections in *Allochrocebus solatus*, a vulnerable primate species native to Gabon. Using molecular techniques like nested PCR and phylogenetic analysis, we found 34.0% infection rate for malaria parasites infection, 21.3% for *Trypanosoma* spp., and 12.8% co-infections. Additionally, *Hepaticystis* was exclusively detected among malaria parasites, while *Trypanosoma brucei brucei*, *T. vivax*, and *T. congolense* were identified. These results underscore the complex host–parasite interactions influenced by captivity and the ecological and immunological consequences of such infections, particularly the increased susceptibility associated with captivity-induced stress. This preliminary study highlights the need for ongoing surveillance to mitigate health risks in primates and prevent potential zoonotic spillovers, providing critical data for conservation efforts.



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1. Introduction

The sun-tailed monkey (*Allochrocebus solatus*) is an endemic primate species from Gabon belonging to the Cercopithecinae subfamily. First described in 1984 [1,2], its elusive behavior and cryptic appearance hinder observation in the wild, which has limited research on the species [3]. Nonetheless, *A. solatus* plays a vital ecological role in seed dispersal and forest dynamics [4,5].

A. solatus faces numerous threats, including deforestation and subsistence hunting [6,7], resulting in its classification as vulnerable on the IUCN Red List (IUCN, 2012) and its inclusion in Appendix II of CITES [5]. In Gabon, *A. solatus* is fully protected under the Forestry Code and 2011 decree.

Alongside anthropogenic pressures, *A. solatus*, like other non-human primates, is susceptible to health risks from infectious agents that may significantly impact its population dynamics [8–11], although data on these pathogens is scarce. Previous studies have explored simian immunodeficiency virus (SIV) [12] and intestinal parasite [10], highlighting an urgent need for further research on pathogens affecting this species. Despite the advances in knowledge concerning its ecology and gastrointestinal parasites, systemic

infections such as malaria and trypanosomiasis have yet to be extensively studied. The paucity of prior molecular studies on blood parasites in *A. solatus* signifies a critical gap in the existing body of research, necessitating further investigation to assess potential health risks and zoonotic implications.

It is evident that African non-human primates (NHPs), in both wild and captive settings, have been documented as significant reservoirs for various blood parasites, particularly those belonging to the *Plasmodium* and *Trypanosoma* genera [13–17]. Blood parasites identified in NHPs include several malaria species such as *Plasmodium praefalciparum*, *Plasmodium falciparum*, *Plasmodium gonderi*, *Plasmodium* DAJ-2004 (recently renamed *Plasmodium mandrillii* [18]), *Plasmodium petersi*, *Hepaticystis* spp. [13,18–22]. Additionally, *Trypanosoma* species, including *Trypanosoma brucei brucei*, *Trypanosoma vivax*, and *Trypanosoma congolense*, have been documented [16,17]. Despite the extensive research conducted on NHPs, *Allochrocebus solatus* remains unstudied in this regard, underscoring a critical gap in our understanding of its interactions with blood parasites and potential zoonotic risks.

From a “One Health” perspective, *A. solatus* may act as a reservoir for *Plasmodium* spp. and *Trypanosoma* spp., with potential implications for spillover to humans and other animals [18,23]. Given increasing habitat encroachment and human–wildlife interactions, it is crucial to understand these infections for both primate conservation and public health purposes [15]. Traditional microscopic methods for parasite detection have limitations, particularly in detecting low-level infections and differentiating closely related parasite species [24,25]. To address these challenges, this study employs nested PCR for enhanced sensitivity and specificity, while phylogenetic analysis will allow for precise identification of parasite lineages and potential zoonotic connections [15,26,27].

The present study aims to address this knowledge gap by investigating the diversity of blood parasites in captive *A. solatus*, with a focus on malaria and trypanosomiasis. By identifying species-specific health risks, this research will contribute to conservation efforts through the implementation of targeted health management strategies. This will ensure better protection of *A. solatus* and mitigate potential zoonotic threats.

2. Materials and Methods

2.1. Study Area and Sample Collection

The diversity of *Plasmodium* spp. and *Trypanosoma* spp. in *Allochrocebus solatus* (Figure 1) at the CIRMF Primatology Centre was characterized. Most individuals were born in captivity at the center, while some were confiscated from private owners by Ministry of Water and Forestry agents in collaboration with Save Gabon’s Primates NGO. During annual health checks, blood samples were collected by trained veterinarians who adhered to animal welfare standards and used EDTA tubes. They extracted approximately 2 to 7 mL of blood per animal, depending on its weight, and immediately placed the tubes in a cool box for transport to the CIRMF’s Unite de Recherches en Ecologie de la Santé (URES) laboratories, where blood samples were stored at −20 °C. A total of 47 individuals were sampled. For additional details on the study population, see Table 1.

Table 1. Summary of data characterizing the population studied.

Sex	Juvenile	Adults	Total
Males	9	12	21
Females	10	16	26
Total	19	28	47



Figure 1. Some solatus individuals from the group are living in captivity at CIRMF's Primatology Centre.

2.2. DNA Extraction and Polymerase Chain Reaction (PCR)

Total DNA was extracted from approximately 200 μ L of blood for each sample using the DNeasy Blood and Tissue Kit (Qiagen, Courtaboeuf, France) following the manufacturer's procedures.

2.3. Molecular Detection of Trypanosomes Parasites

Trypanosome detection utilized a two-step PCR with a recombinant Taq DNA polymerase kit (Life Technologies, France). The initial PCR targeted trypanosomal DNA using primers ITS1 and ITS2, amplifying regions of the 18S rRNA gene and 5.8S/28S rRNA [28]. The 25 μ L reaction included 5 μ L of DNA, 2.5 μ L of 10 $\times$ PCR buffer, 2 μ L of dNTPs (10 mM each), 2 μ L of $MgCl_2$ (50 mM), 0.24 μ L of each primer (10 μ M), 0.3 μ L of Taq polymerase, and 12.72 μ L of RNase-free water. Amplification started with an initial denaturation at 95 $^{\circ}$ C for 7 min, followed by 35 cycles of denaturation at 94 $^{\circ}$ C for 1 min, annealing at 56 $^{\circ}$ C for 1 min, and extension at 72 $^{\circ}$ C for 2 min, with a final elongation at 72 $^{\circ}$ C for 10 min. In the second step, a nested PCR was performed using primers ITS3 and ITS4 [28] in a 25 μ L reaction using 1 μ L of DNA from the first PCR product, along with the same PCR components except for 16.72 μ L of RNase-free water. This protocol included an initial 5 min of denaturation at 95 $^{\circ}$ C, then 35 cycles of denaturation at 94 $^{\circ}$ C for 30 s, annealing at 56 $^{\circ}$ C for 30 s, and extension at 72 $^{\circ}$ C for 1 min, with a final elongation at 72 $^{\circ}$ C for 10 min. PCR products were analyzed by electrophoresis on a 1.5% agarose gel in TAE buffer to confirm the presence of trypanosomal DNA. All positive samples underwent species identification through nested PCR targeting the 18S rRNA gene [29] to amplify a 2.0 kb fragment, followed by sequencing. The sequences were compared to GenBank entries for species identification.

2.4. Identification of Malaria Parasites

Malaria parasite detection followed a documented protocol [21,30]. Amplification targeted a portion of the cyt-b gene using nested PCR with two sets of primers: DW2–DW4, developed by [31], and Cytb1–Cytb2, originally designed by [32] for *P. falciparum*. Notably, these primers effectively amplify cyt-b from a broad spectrum of haemosporidian parasites in diverse hosts [21,27,33] and are suitable for taxonomic studies [34,35]. All PCR products (10 μ L) were analysed on 1.5% agarose gels in a Tris-acetate-EDTA (TAE) buffer. Sequencing was performed by Eurofins MWG.

2.5. Phylogenetic Analysis

To assess the relationship between the obtained cyt-b sequences and known haemosporidian species, a phylogenetic tree was constructed using reference sequences from various haemosporidian parasites. Phylogenetic analyses were conducted on a matrix comprising the multiple alignment of the partial Cyt-b sequences (860 nucleotides) from malaria parasites and the ITS 18S sequences (1967 nucleotides) for trypanosomes, along with reference sequences from GenBank. Alignments were performed using ClustalW (v1.8.1) within the BioEdit software (v7.0.9.0; [36]). Maximum Likelihood (ML) methods were used for tree construction, with the most appropriate ML model identified by ModelTest [37] on the Akaike Information Criterion as the General Time Reversible (GTR) model with Gamma distribution and invariant sites (GTR + Gamma + I). The highest-likelihood tree and corresponding bootstrap support values were then generated using PhyML [38,39], available on the ATGC bioinformatics platform (<http://www.atgc-montpellier.fr/>, accessed on 16 January 2025). Tree construction involved Nearest Neighbor Interchange (NNI) and Subtree Pruning Regrafting (SPR) branch swapping, with 100 bootstrap replicates to ensure robustness of the process.

2.6. Statistical Analyses

All statistical analyses were performed using the R software (version 4.3.1), developed by the R Foundation for Statistical Computing, based in Vienna, Austria. Initially, descriptive statistics were utilized to summarize the infection prevalence rates according to parasite type (*Trypanosoma* spp., *Hepaticystis* spp., and mixed infections), age class (juveniles vs. adults), sex (male vs. female), and season (wet vs. dry). To evaluate whether infection rates varied significantly across demographic and environmental factors, Pearson's Chi-square (χ^2) tests were applied to compare proportions of infected individuals between groups. The analyses included the following: (i) *Trypanosoma* spp. infection prevalence by sex, age class, and season; (ii) malaria parasites (*Hepaticystis* spp.) infection prevalence by sex, age class, and season; and (iii) mixed infection prevalence by age and sex. The significance threshold was set at $p < 0.05$ for all statistical tests.

3. Results

3.1. Detection of *Trypanosoma* spp.

Among the 47 blood samples collected from *A. solatus*, the overall prevalence of *Trypanosoma* spp. infection was 21.3% (10/47). Of the 10 individuals testing positive for *Trypanosoma* spp., 25% (7/28) were adults, while 15.8% (3/19) were juveniles. However, the difference in infection rates between adults and juveniles (7/28 vs. 3/19) was not statistically significant ($p = 0.72$). Three species of *Trypanosoma* spp. were found in this population: *Trypanosoma brucei brucei* (5/47), *Trypanosoma vivax* (3/47), and *Trypanosoma congolense* (2/47) (Figure 2).

The phylogenetic tree made from 18S rRNA sequences of *Trypanosoma* spp. shows three separate groups that correspond to *T. brucei brucei*, *T. congolense*, and *T. vivax*. Sequences obtained from *A. solatus* grouped closely with reference sequences of these species from GenBank, confirming their species. The *T. brucei brucei* clade was a strong group, with high support (>90%) from the analysis, showing that the isolates were closely related. The *T. congolense* and *T. vivax* sequences showed slightly more divergence, forming independent branches but still grouping reliably within their respective species clades. This tree shows that there are different types of *Trypanosoma* spp. infecting the same animals and highlights the genetic diversity of these parasites in monkeys.

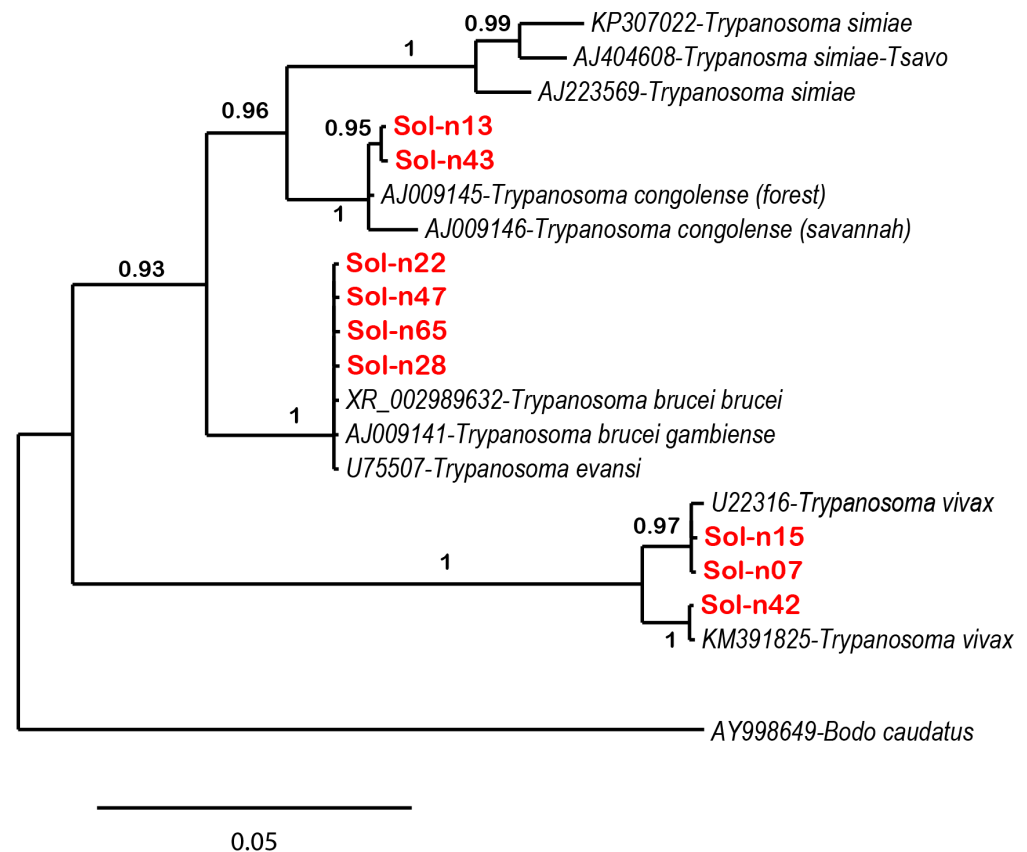


Figure 2. 18S rRNA-based phylogeny of trypanosomes from *Allochrocebus solatus*. Maximum likelihood tree with bootstrap values for 1000 replicates. 18S rRNA sequences generated in this study are in red, and Genbank sequences are accession numbers before the parasite names. Each sequence is labeled with the *A. solatus* number (e.g., Sol-n13, accession number: PV425447–PV425455).

3.2. Detection of Malaria Parasites

Malaria parasite infection prevalence detected by PCR targeting cytochrome b was 34.0% (16/47) among the analyzed blood samples. Of the 16 positive individuals, 25% (7/28) were adults, compared to 47.3% (9/19) in juveniles. However, the difference in infection rates between adults and juveniles was not statistically significant ($p = 0.13$). Only *Hepaticystis* spp. parasites were found in monkeys (Figure 3), previously identified in monkeys and bats in Africa, particularly in mandrills in Gabon.

The phylogenetic analysis based on a portion of the cytochrome b gene (875 bp) identified only *Hepaticystis* spp. was present among malaria parasites (Figure 3). The sequences obtained from *A. solatus* formed a well-supported monophyletic clade (bootstrap > 85%) that was distinct from *Hepaticystis* lineages previously reported in African bats, indicating that it was specific to its host. The *Hepaticystis* sequences from *A. solatus* grouped closely with lineages previously described in other African cercopithecine monkeys (e.g., *Mandrillus sphinx* and *Cercopithecus cephus*), suggesting that they share a common ancestor or that the parasites can cross species in the wild. The results also showed some genetic differences within the *Hepaticystis* complex, which could mean that there are hidden species or subspecies within it.

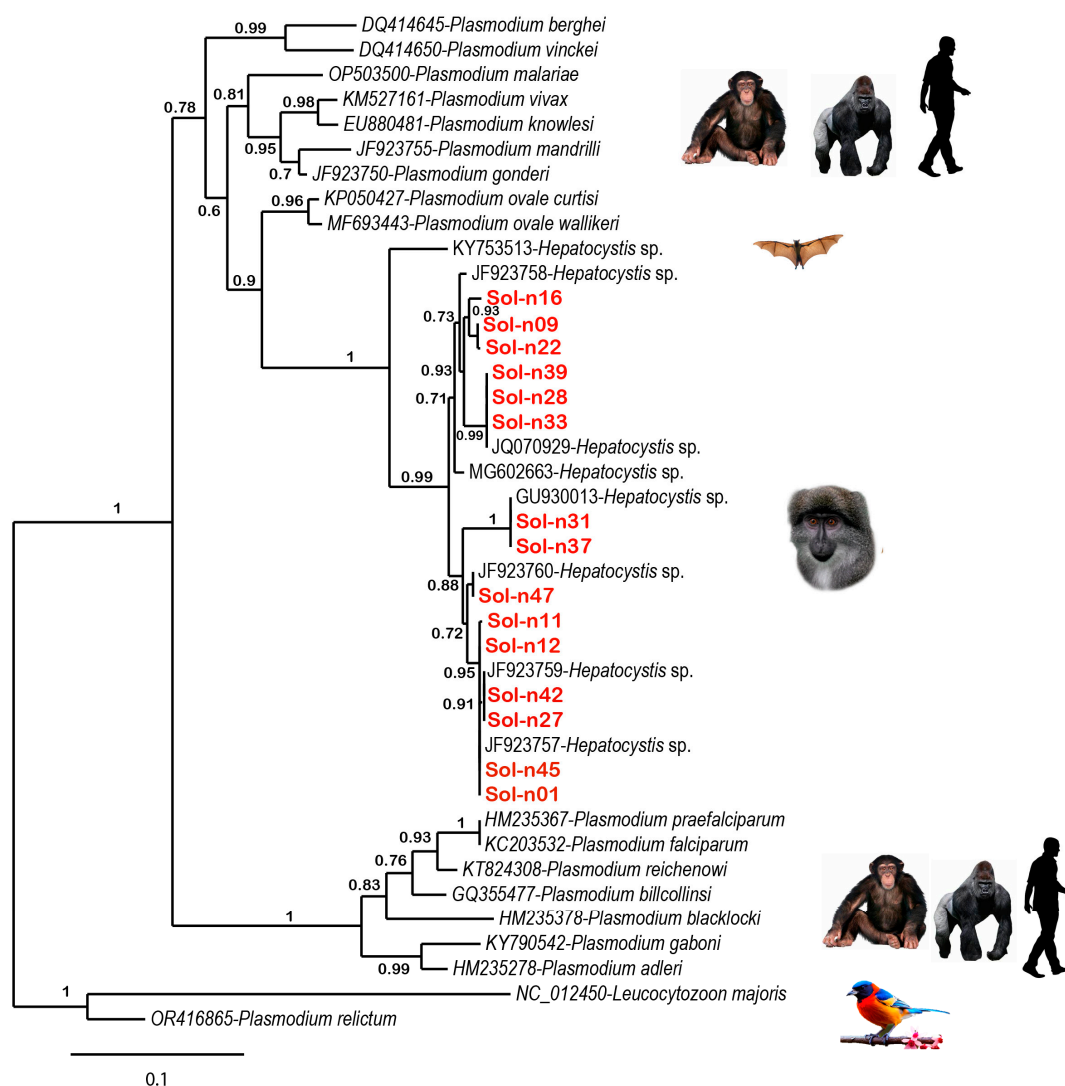


Figure 3. Phylogenetic relationships between the cytochrome b sequences of malaria parasites obtained in this study and those of known *Plasmodium* species (represented by their accession numbers). The tree was constructed using cytochrome b (cyt-b) sequences of 875 bp. Red indicates sequences from *Allochrocebus solatus*. Bootstrap values are given at each node.

3.3. Mixed Infection Prevalence

Mixed infections of *Plasmodium* and *Trypanosoma* occurred in 12.8% (6/47) of cases. The most common combinations were *Hepatocystis-Trypanosoma brucei brucei* (50%, 3/6), *Hepatocystis-Trypanosoma vivax* (33.3%, 2/6), and *Hepatocystis-Trypanosoma congolense* (16.7%, 1/6) (Table 2).

Table 2. The occurrence of mixed infection association results in infection.

Associations	Associations	Percentage
<i>Heptacystis</i> sp.- <i>Trypanosoma brucei brucei</i>	3/6	50%
<i>Heptacystis</i> sp.- <i>Trypanosoma vivax</i>	2/6	33.3%
<i>Heptacystis</i> sp.- <i>Trypanosoma congolense</i>	1/6	16.7%

4. Discussion

This study provides valuable insights into the parasitic infections affecting *Allochrocebus solatus* in captivity, highlighting the prevalence of *Trypanosoma* spp. and *Plasmodium* spp., as well as the occurrence of co-infections. These findings underscore the intricate

interactions between parasites, their host, and the conditions of captivity, offering broader implications for wildlife health and conservation efforts.

4.1. Prevalence of *Trypanosoma* spp. Infections

The prevalence of *Trypanosoma* spp. infections in solatus was 21.3%, with a higher infection rate observed in adults (25%) compared to juveniles (15.8%). Although this difference was not statistically significant ($p = 0.72$), it aligns with findings in other African primates, where adults often exhibit higher prevalence due to prolonged exposure to vectors [16,17]. The trypanosome species identified in this study (*Trypanosoma brucei*, *T. vivax*, and *T. congolense*) are principally transmitted by tsetse flies (*Glossina*), although other blood-sucking flies, such as stomoxids and tabanids, may also contribute to transmission [28,40].

The presence of *Trypanosoma* spp. in captive primates can be attributed to a combination of ecological, biological, and environmental factors. Primates held in captivity often reside in environments that remain conducive to vector proliferation, particularly when enclosures are situated near wetlands or areas with high humidity, as these factors favor the survival of *Glossina* [41,42]. Furthermore, the confinement of hosts within controlled environments increases the likelihood of host–vector interactions, thereby promoting parasite transmission [41,43]. Additionally, some infections may have been contracted prior to capture, as *Trypanosoma* spp. are known to establish chronic, asymptomatic infections that can persist undetected for extended periods without specific diagnostic tools [40,44]. Finally, natural reservoirs such as rodents and other mammals living near enclosures may sustain transmission cycles, increasing the risk of continued parasite exposure despite controlled conditions [45,46].

It is also important to consider the role of captivity stress in modulating immune responses, which may increase susceptibility to infections. Chronic stress in captive primates has been associated with elevated cortisol levels, leading to immunosuppression and reduced ability to control parasitic infections [47,48]. Earlier research on macaques and baboons has shown that stress-induced immune modulation can lead to increased parasite burdens and more severe disease progression in captive animals compared to their wild counterparts [49,50]. This suggests that stress-related immune alterations could partially explain the infection rates observed in solatus, emphasizing the need for improved management strategies to mitigate stress and enhance immune resilience in captive settings.

In captivity, consistent and prolonged exposure to vectors increases the risk of infection across age groups. Environmental factors, such as the proximity of enclosures to wetlands and the presence of unmanaged waste, create ideal conditions for vector breeding and transmission. Habitat fragmentation and human activities, as noted by Njiokou et al. [16], further exacerbate primate–vector interactions and facilitate the persistence of *Trypanosoma* spp. infections. The identification of *T. brucei brucei* in this study provides an important model for understanding zoonotic subspecies such as *T. b. gambiense*, the causative agent of human African trypanosomiasis (sleeping sickness). This underscores the need for ongoing surveillance of *Trypanosoma* spp. in captive primates to better understand their epidemiology and potential zoonotic implications.

4.2. Prevalence of *Plasmodium* Infections

The present study observed a higher prevalence of malaria parasites (34.04%) than of *Trypanosoma* spp. infections, a trend that was particularly pronounced among juveniles (47.3%) compared to adults (25%). Although this difference was not statistically significant ($p = 0.13$), it suggests that juveniles, being immunologically naive, are more vulnerable to these infections [51]. This phenomenon is consistent with observations reported in previous

studies, which indicate that adults develop partial immunity through repeated exposure, reducing their susceptibility to new infections [51,52]. The infection rates reported in this study also align with previous observations [13,14,18]. For instance, Boundenga et al. [18] documented variations in infection levels among different primate species in Gabon: 16% in *Mandrillus sphinx*, 40% in *Cercocebus torquatus*, and 75% in *Cercopithecus cephus*. A similar study in Cameroon reported a prevalence of 49.2% (144/292) in *Cercopithecus nictitans* [13]. Conversely, at the same sites in Gabon, infection rates were significantly lower for other cercopithecine species, with 0.48% (2/420) in mandrills and 0.86% (1/115) in other species [23]. These differences suggest that ecological factors, such as vector exposure or biological differences between host species, play an important role. In *A. solatus*, the high prevalence of malaria parasite infections could be linked to increased vector pressure in their natural habitat, where favorable conditions, such as high mosquito densities, elevate transmission risks.

The exclusive detection of *Hepaticystis* spp. in this population emphasizes particular host–parasite interactions. *Hepaticystis* infections have been documented in various African primates, notably *Mandrillus sphinx*, *Cercopithecus cephus*, and *C. torquatus* in Gabon [18]. In captive environments, high animal density and the presence of artificial water sources promote the proliferation of vectors [53,54]. Furthermore, it is well-documented that captivity-induced stress impairs immune function, thereby increasing host susceptibility to infections [55,56]. This stress-induced immune suppression may, therefore, provide a plausible explanation for the elevated infection rates observed in *solatus*.

Phylogenetic analysis indicates that the *Hepaticystis* spp. identified in *A. solatus* forms a monophyletic clade with lineages found in other African primates. This clade is distinct from the clade containing parasites of African bats [57]. This finding highlights the significant genetic diversity within the *Hepaticystis* genus. However, it remains uncertain whether this diversity indicates the presence of multiple taxa or a single, genetically varied species [13]. It is plausible that these distinct lineages correspond to different species. The present study, in concordance with earlier research [13,14,18,21], posits the hypothesis that diverse species of African monkeys may be infected by these disparate parasitic lineages, which appear to be unstructured with regard to host species or genus.

4.3. Co-Infections: Ecological and Immunological Implications

Co-infections involving *Hepaticystis* spp. strain and *Trypanosoma* spp. are of particular significance in regions where both parasites are endemic. These co-infections can result in complex interactions that affect disease progression, host immunity, and transmission dynamics. Research has demonstrated that the prevalence of *Plasmodium* spp. and *Trypanosoma* spp. co-infections exhibits variation [58]. The present study reports a co-infection rate of approximately 12.8% in the study population, with notable associations observed between *Hepaticystis-Trypanosoma brucei brucei* (50%) and *Hepaticystis-Trypanosoma vivax* (33.3%). These associations are frequently related to the occurrence of shared vector habitats and the presence of common exposure risks [59–61].

Such co-infections have the potential to exert a profound effect on the host. These co-infections have been demonstrated to exacerbate immunosuppressive effects, increase parasite burden, and disrupt host–pathogen interactions. Experimental studies in animal models, particularly mice, have demonstrated that co-infection with *Plasmodium berghei* and *Trypanosoma brucei* results in increased severity of both malaria and trypanosomiasis [62]. Intriguingly, *Trypanosoma brucei* infection has also been observed to offer protection to mice against subsequent malaria infection by modulating the host immune response [58,63].

The co-endemicity of malaria and trypanosomiasis suggests that these co-infections may influence the transmission dynamics of both diseases. For instance, the presence

of *Trypanosoma* spp. infections in animal reservoirs has been demonstrated to alter the vectorial capacity of *Anopheles* mosquitoes for *Plasmodium* parasites [61]. A comprehensive understanding of these interactions is imperative for the formulation of integrated disease management strategies. Further research is required to elucidate the mechanisms underlying *Plasmodium*–*Trypanosoma* co-infections and their impact on host immunity and disease transmission. Such insights are essential for predicting the effects of co-infections on host populations and for designing effective control measures in regions where both parasites are prevalent.

4.4. Implications for Public Health and Conservation

The presence of *Trypanosoma* spp. and *Plasmodium* spp. in captive *A. solatus* has significant implications for public health and conservation, particularly in regions where there is frequent interaction between humans and wildlife. While *Trypanosoma brucei brucei* and *T. vivax* primarily infect animals, there is evidence of atypical human cases that suggest potential zoonotic transmission [64,65]. Furthermore, certain *Plasmodium* species that infect non-human primates have the potential to infect humans, particularly in areas where vector control measures are deemed inadequate. Parasite spillover may occur via multiple pathways, including vector contact, environmental contamination, and direct interactions, as evidenced by documented cases of *Plasmodium knowlesi* transmission from macaques to humans in Southeast Asia [66,67]. The increasing encroachment of human habitats onto wildlife habitats, such as those of primates, has the potential to facilitate bidirectional transmission of infectious diseases, including those of primates to humans, and thus the risk of emerging infectious diseases. In order to mitigate these risks, integrated vector control strategies should be prioritized, including the modification of habitats to limit vector breeding, the use of insecticide-treated nets, and targeted spraying to disrupt transmission cycles. Additionally, continuous health monitoring programs involving both primates and human populations are essential for tracking infection dynamics and enabling early intervention. The findings emphasize the importance of *One Health* approaches, which emphasize collaboration between wildlife conservationists, public health authorities, and local communities to ensure both primate conservation and human health security.

4.5. Study Limitations and Future Directions

This study's relatively small sample size (47 individuals) may limit the generalizability of the results. Additionally, focusing solely on PCR-detectable parasites excludes other pathogens, such as viruses and bacteria, that may impact *A. solatus*. Comparative analyses with wild populations could provide a more comprehensive understanding of the effects of captivity on parasitic prevalence. Future studies should adopt a multi-pathogen approach and include larger sample sizes to address these gaps.

5. Conclusions

In conclusion, this study provides valuable preliminary data on the diversity of blood parasites circulating in captive *A. solatus* in Gabon, revealing significant infection rates by *Hepatocystis* spp. (34.0%) and *Trypanosoma* spp. (21.3%), as well as cases of co-infection in 12.8% of individuals. Although no significant age or sex differences were observed, the results highlight the complexity of host–parasite interactions in semi-captive environments and the increased susceptibility of these primates to vector-borne diseases. Given the frequent proximity of these captive colonies to human activities, our results highlight the importance of establishing systematic health surveillance of captive primates. Such surveillance not only improves animal welfare and optimizes conservation efforts but also enables early detection of pathogens with zoonotic potential.

The adoption of an integrated approach based on the *One Health* concept appears essential to anticipate and control health risks at the human–animal–environment interface. The detection of *Trypanosoma* spp. and *Hepaticystis* spp. lineages with known or suspected zoonotic capacity reinforces the need to implement unified management strategies, mobilizing the veterinary, environmental, and public health sectors. Finally, the identification of *A. solatus* as a potential reservoir host for several blood-feeding parasites calls for further research into its ecological role in transmission cycles. A better understanding of these dynamics is essential for the development of targeted and effective interventions for the conservation of endangered species and the prevention of emerging diseases.

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Institutional Review Board Statement: The study was approved by the National Ethics Committee of Gabon and approved by the Gabonese Ministries of Water and Forests, Higher Education, Scientific Research and Innovation (Approval No. AR0031/10/MENESRESI/CENAREST/CG/CST/CSAR, 10 March 2013). In addition, the study was approved by the Scientific Committee of the Centre Interdisciplinaire de Recherches Médicales de Franceville (CIRMF) to ensure compliance with the ethical principles of animal research (15 June 2021). All samples were collected in strict compliance with animal welfare standards and, in the case of domestic animals, consent was obtained from the owners prior to sampling.

Informed Consent Statement: Not applicable.

Data Availability Statement: All the data generated in this work are available in this article. In addition, the sequences obtained have been submitted to GenBank (PV425447–PV425455 for Trypanosomes parasites and PV454983–PV454997 for malaria parasites).

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

CIRMF	Centre Interdisciplinaire de Recherches Médicales de Franceville
URES	Unité de Recherches en Ecologie de la Santé
SOL	<i>Solatus</i>

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