



Bat phylogeny and geographic location, rather than bat individual characteristics, explains the pattern of trypanosome infection in Europe



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ABSTRACT

Understanding the drivers of parasite susceptibility provides valuable information, such as how parasites spread, what conditions favour their transmission, and what host characteristics make infections more likely. It can also reveal co-evolutionary dynamics and adaptation strategies between hosts and parasites. In this study, we investigated the infection patterns of several bat species across Europe by trypanosome parasites. We used phylogenetic generalised linear mixed models to investigate whether geographic location, individual characteristics (sex, body mass and body size) or species affect trypanosome infection. Additionally, we examined whether infection patterns were influenced by host phylogeny (similar prevalence among genetically close species) and tested for a cophylogenetic signal between bats and trypanosomes. Our results show that individual characteristics were poor predictors of trypanosome infection, whereas host phylogeny and geographic location significantly explained variation in infection. We also found a cophylogenetic congruence between bat species and trypanosome lineages, but this was primarily driven by the association between the bent-winged bat (*Miniopterus schreibersii*) and its trypanosome lineages. Overall, host phylogeny emerges as the main determinant of trypanosome infection in bats. These findings suggest that the probability of infection is governed by deterministic factors rather than random encounters between bats and their trypanosome parasites. However, the high host specificity and absence of a strong cophylogenetic signal indicate that random host switching, rather than co-speciation, is the dominant mechanism shaping bat-trypanosome associations.

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1. Introduction

Understanding why some individuals are parasitised while others remain uninfected has attracted the attention of many researchers since the emergence of ecological parasitology (Poulin, 2021). Indeed, individuals do not all have the same weight when it comes to parasite transmission (Streicker et al., 2013), and one of the only universal laws of parasite ecology is that a few hosts harbour large numbers of individuals of a given parasite species, while most hosts harbour few or none (Poulin, 2021, 2007). In this context, identifying the most vulnerable hosts and

the ecological and evolutionary factors that drive heterogeneity in infection risk can help predict the dynamics or future outbreaks of parasites, as well as provide information on where new parasites might emerge (Stephens et al., 2016).

Several studies have attempted to identify the factors involved in infection risks. These investigations have shown that exposure of hosts to their pathogens varies with environmental conditions (Parratt et al., 2016). For example, a commonly observed trend is an increase in pathogen transmission and prevalence in urban areas (Brearley et al., 2013; Gottdenker et al., 2014). At the individual host level, the susceptibility of infection varies with microclimate, resource availability, or parasite virulence but also with host individual characteristics (Penczykowski et al., 2016; Tack et al., 2012). For example, several host specific features, such as sex and body condition have been associated with infection risk.

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The prevalence and intensity of parasite infection are often higher in males, with numerous exceptions (Christe et al., 2007; Wesolowska, 2022; Zuk and McKean, 1996). There is also an overall negative relationship between infection and body condition in wildlife, although there is heterogeneity between the effects found in different studies and publication bias towards negative associations (Sánchez et al., 2018).

The susceptibility to infection between two individuals may also be related to host species (Christe et al., 2003). Within a local community, vulnerability to a pathogen is not randomly distributed among species (Clark and Clegg, 2017; Parker et al., 2015; Poulin, 1995), and several works showed that host phylogeny can predict disease spillover and prevalence (Barrow et al., 2019; Parker et al., 2015; Streicker et al., 2010). In addition, there is a decline in the likelihood of a pathogen infecting different host species as phylogenetic distance increases (Gilbert and Webb, 2007; Wells et al., 2015), and phylogeny regularly explains patterns of infection susceptibility (Farrell et al., 2013). This leads to a variation in the ability of parasites to invade and persist in the hosts they encounter, from one host species to another (Combes, 1991). As there is a requirement for parasites to be adapted to the host environment, the parasites of a host may undergo evolutionary changes if hosts are subject to evolutionary changes (Page, 2003). As a result, parasites are likely to co-evolve with their host and ultimately show a co-speciation signal and should preferentially or exclusively exploit that host or closely related species. Although there are examples of co-speciation in the literature (Megía-Palma et al., 2018; Page, 2003; Pérez et al., 2019), examples remain relatively rare and are probably overestimated (de Vienne et al., 2013).

Infection risk of vector-borne parasites is shaped not only by host susceptibility but also by the ecology of the vectors involved in transmission. Factors such as vector abundance, host-vector contact rates, habitat use, and vector specificity contribute significantly to heterogeneity in infection patterns (Cator et al., 2020; de Angeli Dutra et al., 2022; Ferraguti et al., 2018; Ferro e Silva et al., 2018; Rizzoli et al., 2019). As a result, understanding variation in infection prevalence requires consideration of both host traits and ecological drivers of exposure.

Bats (Order Chiroptera) show a tendency to harbour zoonotic parasites to a greater extent than ecologically analogous taxonomic groups, such as rodents (Luis et al., 2013). Identifying the factors that modulate the risk of infection and understanding their relative importance within this mammalian order may help to limit the risk of emerging zoonoses in domestic animals and humans, while preserving the ecosystem services provided by these species, including arthropod consumption, seed dispersal, pollination, and many other benefits to agriculture (Ghanem and Voigt, 2012; Kunz et al., 2011; Maas et al., 2016). Several studies have focused on the factors that could explain why bats are an important reservoir of pathogens. First, their immune system may differ from that of most other mammals, as an indirect effect of evolutionary adaptations for sustained flight (Irving et al., 2021). There is also the presence of specific physiological mechanisms in these species that induce an excellent balance between enhanced host defence responses and immune tolerance (Irving et al., 2021; Morales et al., 2025). The ecology of bats, such as their roosting behaviour, also make them excellent hosts for parasites (Adhikari et al., 2020; Calisher et al., 2006; Irving et al., 2021; Luis et al., 2013).

In this study, we investigated the infection patterns of the kinetoplastid flagellated *Trypanosoma* parasites in several bat species across Europe. The vectors of bat-associated trypanosomes remain largely unknown, but ecological data suggest that they are transmitted by hematophagous insects such as cimicid and polyctenid bugs and nycteribiid bat flies (Austen and Barbosa, 2021; Barros et al., 2019; Lima et al., 2012). Bat trypanosomes are closely related

to trypanosomes infecting other mammals, including humans, such as *Trypanosoma cruzi*, responsible for Chagas disease in South America (Clément et al., 2020; Hamilton et al., 2012; Kostygov et al., 2021; Lima et al., 2012). This study may therefore be a further step in the understanding of the evolutionary relationships between mammals and these important parasites. Although there are only a few studies on the virulence of these parasites, trypanosome infection does not appear to be pathogenic for bats (Austen and Barbosa, 2021). This is important from an epidemiological perspective, as not succumbing to infection maximises reservoir competence (Austen and Barbosa, 2021).

A central question in disease ecology is whether host-parasite associations arise from deterministic processes, such as host traits, evolutionary history, or ecological context, or whether they are predominantly shaped by random interactions and stochastic exposure. Here, we explicitly test the hypothesis that *Trypanosoma* infection patterns in bats are not random but instead structured by predictable factors, including host species identity, geographic location, individual characteristics (sex, body mass, and size), or phylogenetic relatedness. If infection is driven by deterministic processes, we expect consistent variation in prevalence across species or locations and a phylogenetic signal in host-parasite associations. We also test for a cophylogenetic signal between bats and their *Trypanosoma* parasites to evaluate whether host-parasite evolution has been shaped primarily by co-speciation or by random host switching.

2. Material and methods

2.1. Field sampling

In 2015, a total of 607 adult individuals representing 21 bat species from the families Vespertilionidae, Miniopteridae and Rhinolophidae were surveyed in 35 sampling sites in five European countries (Fig. 1). Bats were captured in a variety of locations, including caves, forests, buildings, and bat boxes, using harp traps, mist nets or hand nets. All handling of animals followed the guidelines of the American Society of Mammalogy (Sikes et al., 2011). Identification of each bat was performed using traditional morphological keys and, in cases of uncertainty, confirmed by genetic analysis of the cytochrome *b* (*cytb*) gene (Irwin et al., 1991). For each bat, data on forearm length (to the nearest 0.01 mm), weight (to the nearest 0.1 g) and sex were documented. Blood samples, ranging from 5 to 20 μ L depending on bat species and size, were collected in heparinised tubes through a small venipuncture in the uropatagium using sterile needles. Blood samples were then stored either in tubes on ice or on dry filter paper (Whatman MM) until molecular analysis. Bats were released after ensuring that the bleeding had stopped.

2.2. Parasite screening and bat genotyping

Total DNA was extracted from blood using the standard DNeasy Blood & Tissue Kit (Qiagen). A nested PCR (n-PCR) protocol was used to determine the infection status of each bat by trypanosomes. A 561 bp fragment of the 18S small-subunit rRNA gene was amplified. TRYF-TRYR primers were used for the first PCR and SSUF-SSUR primers for the n-PCR, following the protocol by Noyes et al. (1999). The second gene consisted of 786 bp of the glycosomal glyceraldehyde phosphate dehydrogenase (gGAPDH) gene and was obtained using the pairs of primers G5-G3 for the first PCR round and G1-G4A for the second (Hamilton et al., 2004). N-PCR products were run on a 1.5 % agarose gel made with TBE buffer, stained with ethidium bromide and visualised under UV light. The PCR and n-PCR were performed for each sample in two inde-

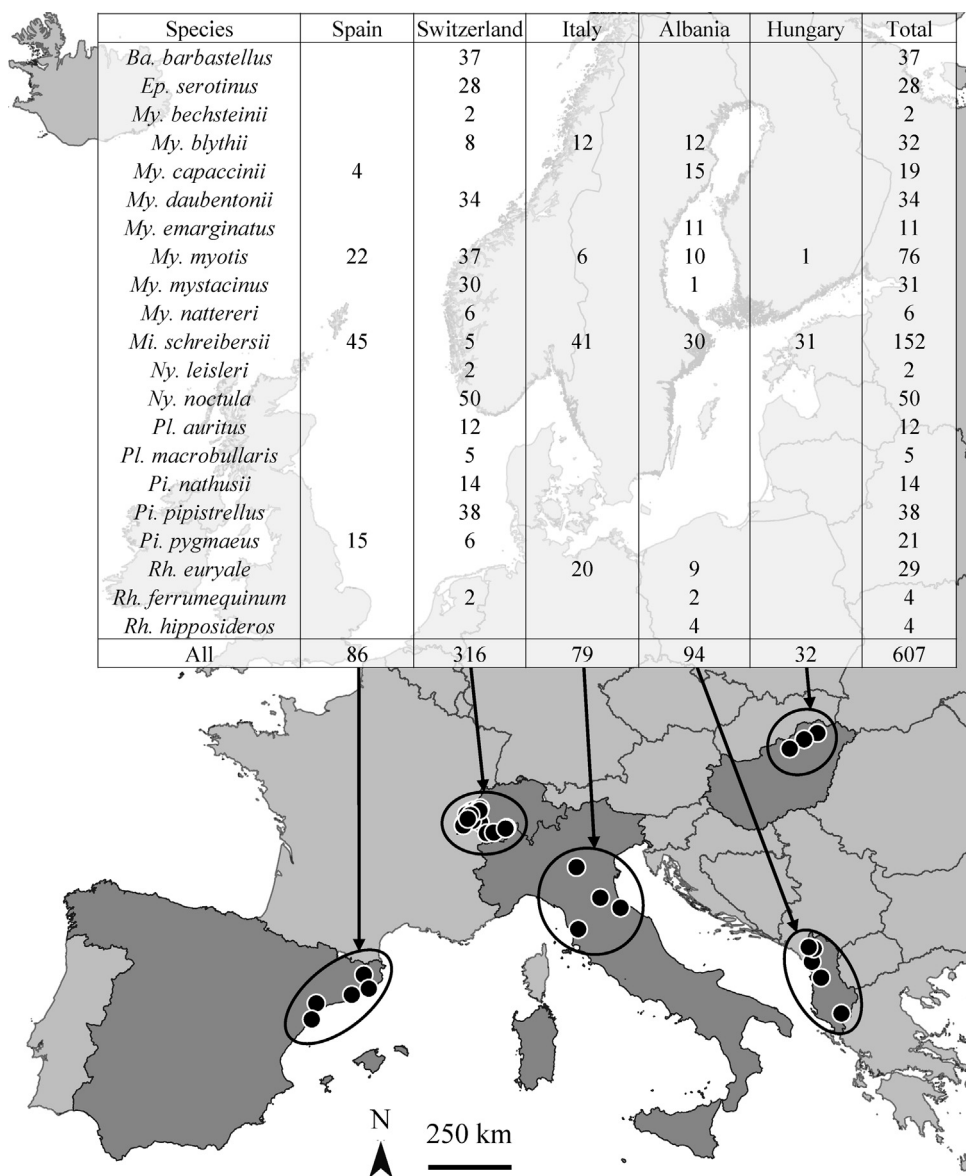


Fig. 1. Sampling sites and number of bats captured per species in each country across Europe. *Ba.* = *Barbastella*, *Ep.* = *Eptesicus*, *Mi.* = *Miniopterus*, *My.* = *Myotis*, *Ny.* = *Nyctalus*, *Pi.* = *Pipistrellus*, *Pl.* = *Plecotus*, *Rh.* = *Rhinolophus*.

pendent replicates. Sequences of 18S rRNA and gGADPH gene were obtained by Sanger sequencing of PCR fragments. All trypanosome sequences used in this study were previously published in Clément et al. (2020) and were identified through BLAST comparison against GenBank entries. For the *Trypanosoma* phylogeny, we used concatenated sequences of SSU rRNA and gGADPH genes. Only samples with both gene regions amplified from the same individual were included, to avoid combining sequences from different genotypes. No mixed *Trypanosoma* infections were detected in our samples. We extracted *cytb* gene sequences for each bat species from GenBank (Supplementary material S1).

To prevent and monitor potential contamination, all laboratory work was conducted in separate pre-PCR and post-PCR areas. Work surfaces and equipment were regularly decontaminated using DNA removal reagents and UV irradiation. DNA extraction and PCR setup were carried out using aerosol-resistant filter tips. Negative controls were systematically included during extractions (blank) and PCR (no-template control). All negative controls remained consistently free of amplification, indicating the absence of contamination during molecular procedures.

For both trypanosome and bat nucleotide sequences, alignments were performed using the MUSCLE algorithm and phylogenetic trees were constructed with the neighbour joining (p-distance) method available in MEGA X. We also constructed a phylogenetic tree with the maximum likelihood (Kimura 2-parameter model) method to consider a potential effect of the method of constructing the phylogenies. However, since we obtained the same results, we will therefore present only the analysis with the neighbour joining method in the result section.

2.3. Phylogenetic generalised linear mixed models

We ran phylogenetic generalised linear mixed models, implemented in the R package ‘phyr’, to test the effects of several explicative factors on the presence of trypanosomes in a bat accounting for phylogenetic relationships among host species. We used bat size (forearm length) and weight as proxies for body condition. Indeed, these two measurements are partially representative of the environmental conditions experienced by an individual during its growth (bat size) or during the sampling period (bat

weight (Sánchez et al., 2018)). Sex (female, male), bat size and weight, country and latitude were included as fixed factors. The sampling site, bat species and bat phylogeny were included as random factors. The significance of each factor was tested using a model comparison approach and a likelihood ratio test (LRT), between a model with all explicative variables and a model without the variable of interest but all the others. We initially tested whether the phylogenetic structure of infection varied across geographic sites by including a nested phylogenetic random effect. However, this interaction was not statistically significant (LR value = 0.51, $p = 0.16$) and was therefore not retained in the final models presented hereafter. The prevalence of trypanosomes was calculated from raw data for each bat package species and mapped along the phylogenetic tree using the R package ‘phytools’.

2.4. Host-parasite cophylogeny

We used a Procrustean Approach to Cophylogeny (PACo), available in the R package ‘paco’, to test the congruency of the trypanosome phylogeny with the bat phylogeny (Hutchinson et al., 2017). PACo is a global fit method that quantifies the degree of similarity between two topologies without explicitly testing evolutionary hypotheses such as co-speciation or host-switching and assumes that the parasite phylogeny is dependent on the host phylogeny. The resulting Procrustes sum-of-squares and p -value were established by 10'000 permutations using the ‘r0’ method, which permutes the host-parasite associations explicitly following the assumption that the parasite phylogeny tracks the evolution of the host. To test the robustness of the result, we used a jackknife analysis to ensure that a significant cophylogenetic signal was not due to only one host species. We also extracted individual PACo residuals to evaluate the strength of phylogenetic congruence at the level of specific host-parasite associations, with larger residuals indicating greater phylogenetic incongruence and potential host-switching events.

In addition to the PACo analysis, we used ParaFit to test both the global cophylogenetic structure and the significance of individual links (ParaFitLink1 statistic) based on 999 permutations. Given that the default $\alpha = 0.05$ threshold for ParaFitLink1 may be anti-conservative, we applied a stricter significance threshold of $p < 0.02$ following Balbuena et al. (2013).

2.5. Data Accessibility

All *Trypanosoma* sequences used in this study have been previously published and are available in GenBank (see Clément et al., 2020). No new sequences were generated. The R code, alignment files, and associated datasets used for the cophylogenetic analyses are publicly available via Mendeley Data (DOI: <https://doi.org/10.17632/jvhx2c3g79.1>).

3. Results

Overall, 164 bats (27 %) were infected with trypanosomes. Bat sex, size and weight, as well as latitude and bat species, were not predictors of bat infection status (Table 1). There were significant differences in infection prevalence between countries and across species phylogeny (Table 1). The prevalence of trypanosomes was lower in Spain compared to Italy and Switzerland. It was intermediate in Albania and Hungary (Fig. 2). Species had very heterogeneous levels of infection but are homogeneous when grouped by genus. *Nyctalus*, *Pipistrellus*, *Eptesicus* and *Miniopterus* have high prevalence (>20 %, ranging from 26 % to 53 %), whereas *Plecotus*, *Rhinolophus* and *Barbastella* have low prevalence (<20 %, ranging from 0 % to 20 %). Within the genus *Myotis*, species had heterogeneous trypanosome prevalence, with *My. blythii*, *My. myotis* and

Table 1
Effect of the different factors estimated by model comparison on bat infection by trypanosomes. Each factor was tested by comparing the model with all factors within the model except the tested factor. ‘LR value’ corresponds to the value of the likelihood ratio test.

	Trypanosome infection	
	LR value	p
Sex	1.22	0.27
Bat size	1.25	0.26
Bat weight	1.12	0.29
Country	17.61	p < 0.0001
Latitude	2.60	0.11
Sampling site	2.23	0.017
Bat species	0	1
Bat phylogeny	44.29	p < 0.0001

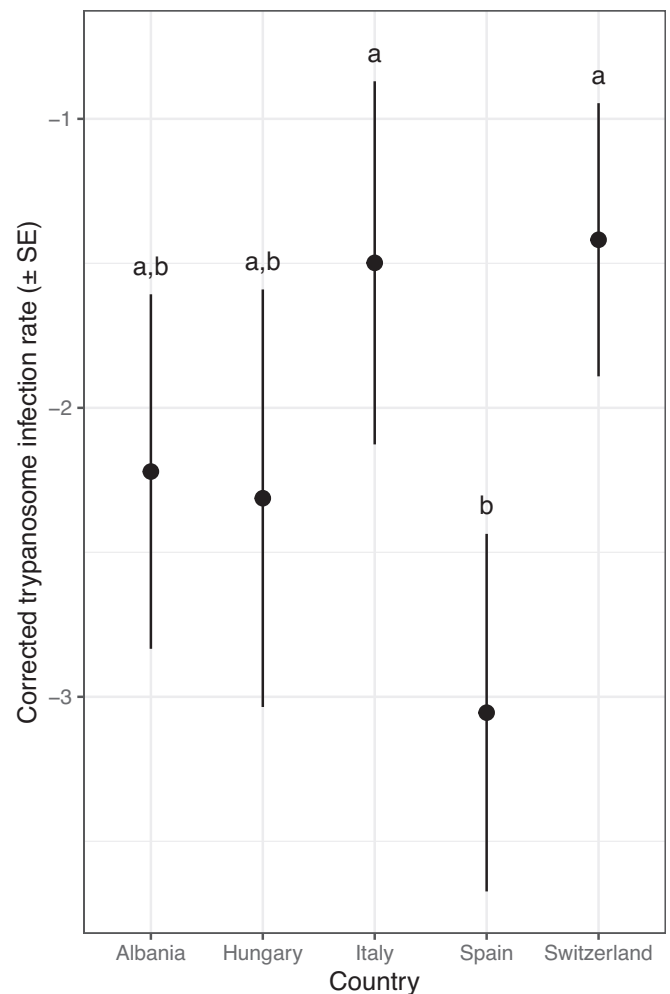


Fig. 2. Mean corrected trypanosome infection rate (±SE) of bat species by country. The prevalence is measured by the residuals of the phylogenetic generalised linear mixed model containing the random factors ‘sampling site’, ‘bat species’ and ‘bat phylogeny’, which allowed controlling for non-independence of the data. Means not sharing any letter are significantly different ($p < 0.05$).

My. mystacinus having prevalence above 25 % (28 %, 33 %, 26 %, respectively), while the other species, including *My. bechsteini*, *My. capaccinii*, *My. daubentonii*, *My. emarginatus* and *My. nattereri* have prevalence below 10 % (ranging from 0 % to 9 %, Fig. 3). Finally, we found a significant effect of the sampling site, indicating that different localities resulted in different trypanosome prevalence in bats (Table 1).

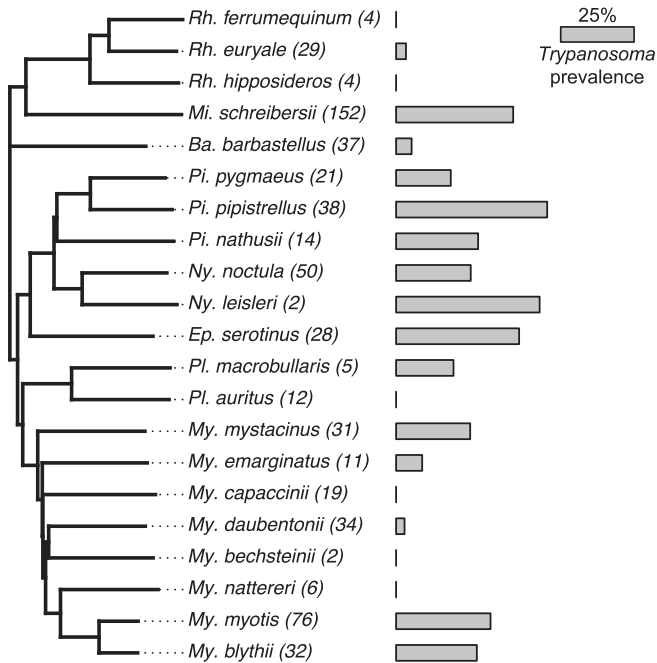


Fig. 3. Trypanosome infection rates across bat phylogeny. The number of bats captured is indicated in brackets. Ba. = *Barbastella*, Ep. = *Eptesicus*, Mi. = *Miniopterus*, My. = *Myotis*, Ny. = *Nyctalus*, Pi. = *Pipistrellus*, Pl. = *Plecotus*, Rh. = *Rhinolophus*.

We found some degree of host specificity in trypanosomes, as infections were associated with one to six different bat species out of the 21 captured in our sample, depending on the trypanosome lineage (Fig. 4). The cophylogenetic analysis indicated some congruence between the host and parasite phylogenies (PACo $m_{XY}^2 = 0.27$; global $P = 0.004$). However, jackknife analysis revealed that this significant result was primarily driven by the inclusion of *Miniopterus schreibersii* in the dataset (PACo $m_{XY}^2 = 0.22$; global $P = 0.072$). Removing any other bat species individually did not alter the overall significance of the phylogenetic congruence (see Supplementary material S2). The ParaFit global test did not detect significant phylogenetic congruence (ParaFitGlobal =

0.00029; $P = 0.581$). All individual links between hosts and parasites have high squared residuals values, except the link *Mi. schreibersii*-*T new sp1 EU*, and were non-significant with the ParaFitLink1 test (Table 2). Overall, there is no strong evidence for a cophylogenetic signal, particularly when the data are restricted to the family Vespertilionidae with lineages of *Trypanosoma dionisii*. One lineage, referred to as *T new sp1*, follows the nomenclature introduced by Clément et al. (2020), where it was described as a close relative of *T. livingstonei* and detected in *Mi. schreibersii* in Europe.

4. Discussion

In this study, we investigated the influence of host life history traits and phylogeny, as well as geographic location, on the probability of trypanosome infection in European bats. We also assessed the presence of a cophylogenetic signal between bats and trypanosomes. We found that host life history traits were poor predictors of trypanosome infection, while host phylogeny and geographic location (i.e., country and sampling site) explained a large proportion of the variation in infection. We also found a cophylogenetic congruence between bat species and trypanosome lineage, but this is only due to the link between *Mi. schreibersii* and its trypanosomes (*T dio EU1* and *T new sp1 EU*). All these results suggest that the probability of infection is under strong deterministic control and does not depend on a random encounter between bats and their trypanosome parasites.

Body condition, as measured by the size and weight of individuals, does not appear to be related to the infectious status of bats. Similarly, the weight-to-forearm ratio showed no significant association (data not shown). Studies on the pathogenicity of trypanosomes in bats, though limited, have not shown significant effects on host health (Linhardt et al., 2022, 2020; Lord and Brooks, 2014). One possible explanation is that these infections are chronic, with minimal impact on the host, suggesting that host defences may not be specifically directed against the parasite. This could explain the lack of difference in infection status between individuals in good and poor body condition. Alternatively, body condition might not be a reliable indicator of trypanosome infection. The infection costs could manifest in other life-history traits, such as survival, reproductive output or immunological paramete-

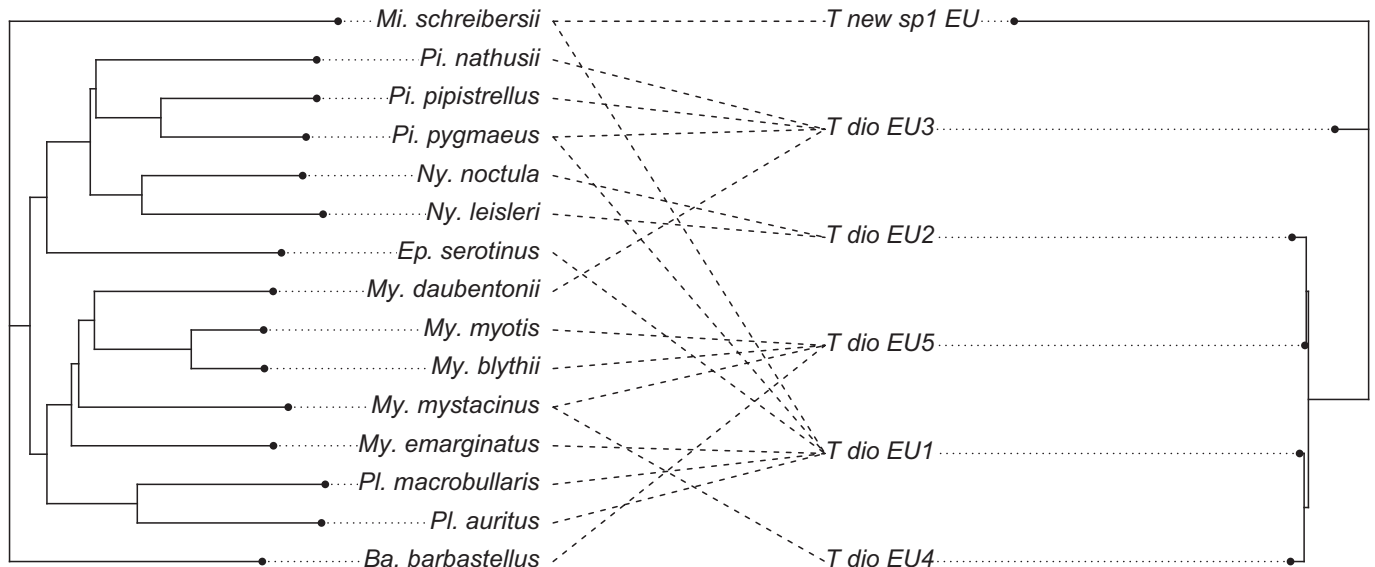


Fig. 4. Tanglegram of associations between 15 bat species and six trypanosome lineages. We removed the unparasitised bat species or bat species with unidentified trypanosome lineages from the phylogeny. T dio = *Trypanosoma dionisii*. Ba. = *Barbastella*, Ep. = *Eptesicus*, Mi. = *Miniopterus*, My. = *Myotis*, Ny. = *Nyctalus*, Pi. = *Pipistrellus*, Pl. = *Plecotus*.

Table 2
PACo residuals and ParaFit test results for individual bat–*Trypanosoma* associations. PACo residuals reflect the degree of phylogenetic incongruence for each host–parasite link, with higher values (>0.1) suggesting potential host-switching events. ParaFit p-values are based on 999 permutations. The α level of ParaFitLink1 to 0.05 is generally considered anti-conservative; significant associations are thus considered when $p < 0.02$, following Balbuena et al. (2013).

Bat species	<i>Trypanosoma</i> lineage	PACo Residual	ParaFitLink1 p-value
<i>Mi. schreibersii</i>	<i>T new sp1 EU</i>	0.045	0.508
<i>Mi. schreibersii</i>	<i>T dio EU1</i>	0.183	0.973
<i>Pi. nathusii</i>	<i>T dio EU3</i>	0.117	0.650
<i>Pi. pipistrellus</i>	<i>T dio EU3</i>	0.107	0.702
<i>Pi. pygmaeus</i>	<i>T dio EU3</i>	0.101	0.208
<i>Pi. pygmaeus</i>	<i>T dio EU1</i>	0.129	0.197
<i>Ny. noctula</i>	<i>T dio EU2</i>	0.119	0.650
<i>Ny. leisleri</i>	<i>T dio EU2</i>	0.129	0.637
<i>Ep. serotinus</i>	<i>T dio EU1</i>	0.129	0.785
<i>My. daubentonii</i>	<i>T dio EU3</i>	0.121	0.784
<i>My. myotis</i>	<i>T dio EU5</i>	0.103	0.059
<i>My. blythii</i>	<i>T dio EU5</i>	0.104	0.323
<i>My. mystacinus</i>	<i>T dio EU5</i>	0.114	0.032
<i>My. mystacinus</i>	<i>T dio EU4</i>	0.106	0.807
<i>My. emarginatus</i>	<i>T dio EU1</i>	0.114	0.714
<i>Pl. macrobullaris</i>	<i>T dio EU1</i>	0.138	0.561
<i>Pl. auritus</i>	<i>T dio EU1</i>	0.136	0.620
<i>Ba. barbastellus</i>	<i>T dio EU5</i>	0.143	0.363

ters (e.g., Schneberger et al., 2014) which were not investigated in this study.

We also found no significant differences in infection status between males and females, although females may be more exposed. Indeed, cimicid bugs (*Cimex lectularius*, *Cimex pipistrelli*) have been identified as potential vectors of trypanosome parasites in Europe (Cavazzana et al., 2010; Gardner and Molyneux, 1988; Paterson and Woo, 1984), with transmission generally occurring through contamination via infected faeces or consumption of infected bugs (Austen and Barbosa, 2021). Although precise data are lacking, infection of bats is most likely to occur in maternity colonies, so females should be more exposed to trypanosome infection than males. Moreover, it has been shown that the prevalence and intensity of bat ectoparasites are higher in females than in males, due to sex-specific social and spatial aggregation on infection strategies (Christe et al., 2007). However, Linhart et al. (2020) also found no difference in trypanosome infection levels between males and females and suggested that bats may be infected as juveniles or by vertical transmission from mother to offspring, explaining the lack of difference between sexes. There is yet no evidence that this is the case for the studied species in our sample area, and further studies focusing on the vectors are needed to determine their identity and the main transmission mechanism of the parasite between hosts and vectors.

Our results highlight spatial variation in the probability of infection, with heterogeneity between both sampling sites and countries. These differences are not related to a broader biogeographic pattern, such as the latitude of the sampling site. Indeed, prevalence is significantly higher in Switzerland and Italy than in Spain. Linhart et al. (2020) have already shown a difference in prevalence between countries, with a significantly higher prevalence in the Czech Republic compared to Bulgaria and Poland. This difference between countries, and even between sampling sites within countries, could have several possible explanations. First, there could be spatial variability in host susceptibility. Our data do not support this hypothesis because there is no significant effect of the sampling sites or the country on bat body condition (data not shown). However, we only have measurements of body condition, and it would be necessary to incorporate more direct measurements of individual immunity to completely exclude this hypothesis.

A second explanation could be related to the spatial variation in the presence of the insect vector. *C. pipistrelli* and *C. lectularius* use different types of habitats, with a large preference for human buildings for *C. lectularius* (Balvín et al., 2014). There is still a gap in our knowledge of the insect vector and its ecology. Further studies should investigate the role of ectoparasites in driving the distribution of trypanosomes in bats, considering regional differences in seasonality and bat species phenology. Finally, anthropogenic disturbance and habitat fragmentation may play significant roles in the observed variation in infection patterns among sampling sites and countries (Nunes et al., 2017; Pulosof et al., 2012; Vicente-Santos et al., 2023; Warmuth et al., 2023). These disturbances can directly or indirectly affect both vector and host populations, offering a straightforward explanation for differences in parasite prevalence. Disturbed habitats could lead to higher pathogen prevalence due to increased stress, altered host immunity, or changes in vector dynamics (Selmann et al., 2017).

Despite the lack of significant effect of individual traits, latitude, or bat species, the probability of being infected by trypanosomes varies significantly across the bat phylogeny. The apparent contradiction between the non-significant effect of bat species identity and the highly significant effect of bat phylogeny on infection probability arises because the effect of bat species becomes non-significant once phylogeny is included in the model. In other words, the phylogenetic random effect captures the variation in infection risk across species due to shared evolutionary history. This suggests that bat susceptibility, host–parasite specificity, exposure or a combination of the three may be conserved across the time scale of bat diversification. In avian malaria models, host infection status is regularly explained by host phylogeny (e.g., Barrow et al., 2019; de Angeli Dutra et al., 2023), but the bat–trypanosome system is not yet fully understood (Tsague et al., 2024). Faria et al. (2013) showed that host genetic distance is the main driver of host shifts for rabies between bats, whereas similarities in roost structures had no predictive power. Parasites and pathogens may be inherited from a shared ancestor of related hosts, or they may move and exploit new hosts. However, they tend to establish more successfully in species that are closely related to their original host (Engelstädter and Fortuna, 2019), as they are often better adapted both immunologically and morphologically to complete their life cycle within closely related species (Pinheiro et al., 2016). There are, however, exceptions to this pattern. For instance, in our study, no co-phylogenetic signal was observed when analysing bat species within the family Vespertilionidae, suggesting that other factors, such as ecological drivers, might drive host–parasite associations in this group. Additionally, while evolutionary history often constrains the adaptation of parasites to distant hosts, naïve hosts (i.e., distantly related species) can sometimes exhibit higher susceptibility due to a lack of immunological adaptations to the parasite (Sorci, 2013). This highlights the importance of considering both phylogenetic and ecological factors in studying host–parasite systems, as the drivers of susceptibility and host shifts may differ across lineages and contexts.

Our results also show a parasite specificity, with only 3 parasitised host species out of the total 21 bat species per parasite lineage on average. Bat species largely differ in their ecological and social aspects, their diet and roosting behaviour (Dietz et al., 2009). *Mi. schreibersii* is a strict cave-dwelling species whereas *Pi. pipistrellus* and *Pi. pygmaeus* are often roosting in human buildings or crevices. *Nyctalus* are migratory with a preference for roosts in mature trees and Mouse-eared bats (*My. myotis/blythii*) use different types of roosts depending on the geographic location. These large differences in roosting preferences indicate that most species never or rarely mix (except for *Mi. schreibersii* and *My. myotis*), making it difficult for the parasite to switch to a new host, leading

to the high host specificity observed in this study. However, host specificity could also be influenced by vector specificity. For instance, *C. lectularius* and *C. pipistrelli* are known to be generalist ectoparasites of bats, and they may facilitate the distribution of *Trypanosoma* lineages across host species (Balvín et al., 2014). In contrast, the *Trypanosoma* lineages observed in *Mi. schreibersii* may rely on a different, highly specific vector, as *Cimex* species are rarely found on *Miniopterus* (Balvín et al., 2014). These considerations highlight the importance of understanding vector ecology in addition to host ecology when interpreting patterns of host-parasite associations. While this hypothesis aligns with our findings, it remains speculative and warrants further investigation. Incorporating direct screening of potential vectors across regions would provide a more robust understanding of transmission dynamics and the role of vector specificity in shaping *Trypanosoma* distribution.

Finally, the phylogenetic analysis showed a significant congruence between the phylogeny of bats and the phylogeny of their trypanosome parasites with PACo but not with ParaFit. The contrasting results of the global PACo and ParaFit tests reflect methodological differences, with PACo potentially detecting subtler patterns of phylogenetic structure (Balbuena et al., 2013). However, the signal is only due to the presence of the link between *Mi. schreibersii* and one of its parasite lineages and our results rather suggest the absence of co-evolution between the different lineages of *T. dionisii* and bat species and both approaches converge on the conclusion that most host-parasite links are not constrained by shared evolutionary history. The scarcity of significant ParaFitLink1 associations and the high PACo residuals support a scenario in which host switching, rather than co-divergence, is the primary driver of trypanosome distribution across bat species. Detecting co-evolution between hosts and parasites requires extensive sampling of host taxa and suitable molecular tools to infer their phylogenies, and to date, evidence for matching phylogenies is limited compared to the number of known host-parasite assemblages (Alcala et al., 2017; Bruyndonckx et al., 2009; de Vienne et al., 2013; Megía-Palma et al., 2018). Although co-evolution between hosts and parasites has been considered a major factor in the diversification of host-parasite associations, several studies showed that host-switching is far more common in shaping these relationships than previously thought (Agosta et al., 2010; Araujo et al., 2015). Hamilton et al. (2007) already showed that co-speciation played little role in trypanosome evolution. Therefore, it seems that host switching, rather than co-speciation, explains the observed associations between bats and their trypanosome parasites in our study.

As with any ecological field study, our conclusions are shaped by the limitations of available methods and sampling design. While Sanger sequencing was sufficient to identify the dominant *Trypanosoma* lineages in our dataset, it may underestimate rare variants or co-infections due to its lower sensitivity. Future studies could incorporate high-throughput sequencing approaches, such as metabarcoding, to provide a more comprehensive view of *Trypanosoma* diversity and co-infection complexity. Our study focused on field-derived sequences, incorporating additional trypanosome sequences from GenBank could help refine parasite phylogenetic resolution. However, such integration may be limited by variability in sequence quality, sampling methods, gene regions, and the lack of host metadata in many public records. We therefore chose to rely on a consistent set of sequences to avoid introducing methodological biases.

CRedit authorship contribution statement

Antoine Perrin: Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis. **Laura Clément:**

Writing – review & editing, Resources, Data curation, Conceptualization. **Tamara Szentiványi:** Writing – review & editing, Resources. **Philippe Théou:** Writing – review & editing, Resources. **Adrià López Baucells:** Writing – review & editing, Resources. **Laura Bonny:** Writing – review & editing, Resources. **Dino Scaravelli:** Writing – review & editing, Resources. **Olivier Glaizot:** Writing – review & editing, Supervision, Resources, Methodology, Investigation, Data curation, Conceptualization. **Philippe Christe:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization.

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Appendix A. Supplementary material

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