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Host-Parasite Coevolution in Wild Rodents: Dynamics, Mechanisms and Evolutionary Consequences

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Abstract

Host-parasite coevolution is a fundamental evolutionary process wherein hosts and their parasites exert reciprocal selective pressures on each other, driving genetic and phenotypic changes over generations. Wild rodents, due to their ecological ubiquity, rapid reproductive cycles, and genetic variability, serve as ideal systems for studying these coevolutionary dynamics in natural settings. This paper explores the complex interactions between wild rodent species and a diverse array of parasites including protozoa, helminths, ectoparasites, and vector-borne pathogens by analyzing immune gene evolution, patterns of local adaptation, and parasite virulence traits. Using a combination of field studies, molecular genetics, and experimental infections, the research reveals strong evidence of Red Queen dynamics and geographic mosaic coevolution. Findings demonstrate that host immune genes, especially those related to the MHC and innate immune pathways, evolve rapidly in response to regionally distinct parasite populations, while parasites exhibit parallel adaptations to evade or manipulate host defenses. These dynamic and localized evolutionary interactions not only deepen our understanding of coevolutionary theory but also have critical implications for managing zoonotic diseases, as wild rodents often act as reservoirs for pathogens with public health relevance.

Keywords: Coevolution, Rodents, Parasites, Immunity, Adaptation

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Introduction

Host-parasite coevolution represents a fundamental driver of biological diversity and evolutionary innovation. It refers to the reciprocal evolutionary changes occurring in interacting species typically a host organism and its parasite each adapting to the selective pressures imposed by the other. This evolutionary arms race can manifest in a variety of ways, including changes in host immune genes, parasite virulence factors, behavioral adaptations, and reproductive strategies (Woolhouse *et al*., 2002). Because of the constant pressure to outcompete one another, host-parasite systems often exhibit rapid and continuous evolutionary change. Wild rodents are among the most important and widely distributed mammalian hosts involved in coevolutionary interactions. Their global presence, ecological versatility, and diverse parasite burdens make them excellent models for studying host-parasite relationships (Morand *et al*., 2015). Rodents host a wide range of parasites, including gastrointestinal helminths (e.g., Heligmosomoides polygyrus), protozoa (e.g., Toxoplasma gondii, Eimeria spp.), ectoparasites (e.g., fleas, ticks), and various bacterial and viral pathogens. This diverse parasitic exposure provides ample opportunities for coevolutionary dynamics to unfold at both genetic and population levels. One of the central mechanisms in host-parasite coevolution is the Red Queen hypothesis, which posits that species must constantly evolve to maintain their fitness relative to others in their ecosystem (Van Valen, 1973). This principle is especially evident in rodent populations where selective pressure from fast-evolving parasites leads to continual changes in host immune genes. Host populations are thus never completely resistant, and parasite populations are never entirely successful—both remain in a perpetual state of adaptation (Lively & Dybdahl, 2000). Immune gene evolution plays a key role in rodent-parasite interactions. In particular, genes within the major histocompatibility complex (MHC) are subject to strong balancing selection due to their central role in antigen presentation and immune recognition (Eizaguirre *et al*., 2012). Studies have shown that rodent populations exposed to a broad diversity of parasites exhibit higher allelic diversity in MHC genes, suggesting that parasite pressure maintains genetic variation (Oliver *et al*., 2020). Furthermore, toll-like receptors (TLRs) and cytokine genes are also rapidly evolving in response to specific pathogens and are frequently targeted by natural selection (Turner *et al*., 2021). Local adaptation is another important concept in host-parasite coevolution, where populations adapt to the specific parasite communities found in their local environments. Evidence of local adaptation has been documented in European bank voles (Myodes glareolus), where different populations exhibit varying resistance to helminth and protozoan infections depending on regional parasite composition (Deter *et al*., 2008). In such cases, gene flow, genetic drift, and environmental variability create a geographic mosaic of coevolution, where interactions are shaped by spatial heterogeneity (Thompson, 2005). Parasites, in turn, evolve to overcome host defenses. For example, Eimeria species infecting wild mice have developed antigenic diversity that allows them to evade detection by host MHC molecules (Schmid *et al*., 2022). Similarly, flea species parasitizing rodents have shown regional variation in attachment efficiency and reproductive success, suggesting that they too are evolving in response to host resistance traits. These findings underscore the bidirectional nature of coevolution, wherein both host and parasite evolve specific adaptations and counter-adaptations. Rodents also serve as reservoirs for many zoonotic pathogens, including Yersinia pestis (plague), Leptospira, Hantavirus, and Borrelia burgdorferi (Lyme disease). Their interactions with vector-borne parasites and the pathogens they carry add an additional layer of complexity to host-parasite coevolution. Vector-borne systems often involve tripartite coevolutionary relationships among host, parasite, and vector, each influencing the evolutionary trajectory of the others (Fenton *et al*., 2015). In such cases, rodent immune strategies may coevolve not only with the pathogen but also with traits of the vector that influence transmission efficiency. Environmental change and anthropogenic pressures further complicate host-parasite coevolution. Habitat fragmentation, urbanization, and climate change have been shown to alter rodent population dynamics, parasite distributions, and interaction patterns (Cizauskas *et al*., 2017). These changes may disrupt coevolutionary relationships or accelerate them by creating novel selection pressures. For instance, rodents in urban environments may encounter different parasite communities compared to rural populations, leading to divergent immune adaptations (Stothard *et al*., 2021). Experimental studies using wild-derived rodent lines have provided valuable insight into coevolutionary mechanisms. Controlled cross-infection experiments allow researchers to test hypotheses related to local adaptation, genotype-by-genotype (G×G) interactions, and selection strength. Such experiments have shown that sympatric host-parasite combinations often result in lower infection rates or parasite fitness compared to allopatric pairings, reinforcing the idea of coevolutionary fine-tuning (Grech *et al*., 2006). Technological advances have greatly enhanced the ability to study host-parasite coevolution. High-throughput sequencing, transcriptomics, and genome-wide association studies (GWAS) have enabled detailed mapping of immune gene variation and identification of parasite virulence factors. Moreover, long-term field studies combined with molecular tools are allowing researchers to track coevolutionary changes over time in natural populations (Barreiro & Quintana-Murci, 2020). Given the ecological and epidemiological importance of rodents, understanding their coevolution with parasites is not only valuable for evolutionary biology but also critical for disease surveillance and control. As human-wildlife interactions increase, the risk of spillover events from rodent reservoirs grows, making it imperative to understand how coevolutionary dynamics influence pathogen emergence and spread (Luis *et al*., 2013). In summary, wild rodents provide a rich and dynamic system for studying host-parasite coevolution. Their complex interactions with diverse parasite communities, shaped by environmental and genetic factors, reveal the intricate evolutionary dance that maintains biological diversity and shapes disease outcomes. As research tools improve and ecological challenges intensify,

investigating these interactions becomes even more important for both theoretical and applied science.

Literature Review

Host-parasite coevolution has long intrigued evolutionary biologists, with foundational theories suggesting that this interaction is a key driver of genetic diversity and species adaptation. The idea that parasites exert strong selection on host populations, and vice versa, is not new. Seminal works such as Anderson and May (1982) established mathematical models to describe host-parasite dynamics, proposing that infection rates and host resistance evolve in tandem through frequency-dependent selection. These models laid the groundwork for empirical studies that followed in various host systems, especially in wildlife where natural selection operates with minimal anthropogenic interference. Rodents have emerged as model organisms for coevolutionary studies due to their rapid life cycles, wide geographical distribution, and ecological plasticity. Their role as hosts to a multitude of parasites—ranging from gastrointestinal helminths and protozoa to blood-borne pathogens and ectoparasites—offers rich opportunities to investigate coevolution in real time. For example, Mus musculus (house mouse) and Rattus rattus (black rat) are frequently studied in both laboratory and field conditions, offering insights into how parasite exposure varies across populations and shapes immune system evolution (Turner *et al*., 2020). One of the most studied genetic systems in this context is the major histocompatibility complex (MHC), which plays a vital role in adaptive immunity. Research on bank voles (Myodes glareolus) in Europe has shown high MHC diversity driven by selection from multiple parasites, including Babesia microti and Eimeria species (Loiseau *et al*., 2018). MHC heterozygosity was positively associated with survival and lower parasite loads, suggesting that parasite pressure maintains immune gene polymorphism through balancing selection. In addition to MHC, genes involved in innate immunity, such as Toll-like receptors (TLRs) and cytokines, have shown signs of adaptive evolution in rodent populations. Studies by Kloch *et al.* (2019) on Apodemus sylvaticus demonstrated that TLR2 and TLR4 genes undergo positive selection in response to bacterial and protozoan parasites. These innate immune receptors act as the first line of defense, recognizing pathogen-associated molecular patterns (PAMPs), which may rapidly evolve in parasite lineages to evade detection.

Parasite genotypes are not static either. Just as rodents evolve mechanisms to detect or suppress infections, parasites evolve strategies to enhance infectivity and transmission. Antigenic variation, particularly in protozoan parasites like Trypanosoma and Plasmodium, is a well-documented mechanism by which parasites avoid host immunity. Eimeriaspecies infecting mice show local adaptation to host immune genotypes, indicating a reciprocal genetic arms race (Schmid-Hempel, 2021). Empirical support for local adaptation—where hosts are more resistant to sympatric parasites than to allopatric ones—has been reported in various rodent-parasite systems. A notable example comes from Myodes glareolus populations across Scandinavia, where individuals demonstrated stronger immune responses to local Hantavirus strains compared to foreign strains (Guivier *et al*., 2020). Such findings support the geographic mosaic theory of coevolution, which posits that coevolutionary processes vary across landscapes, generating hotspots of intense interaction and coldspots of reduced selection pressure. Temporal studies provide additional evidence for Red Queen dynamics, a concept proposed by Van Valen (1973), which suggests that hosts and parasites must continuously evolve to maintain relative fitness. Longitudinal data from wild Peromyscus maniculatus populations revealed cyclic patterns in parasite prevalence and corresponding shifts in host gene frequencies over time (Lloyd-Smith *et al*., 2022). These cycles suggest that both parties are locked in an ongoing evolutionary race with no permanent victor. Co-infections and parasite community complexity also influence host-parasite coevolution. Rodents are often infected with multiple parasite species simultaneously, creating complex selective landscapes for both hosts and parasites. For instance, research on Mus musculus in urban environments found that co-infections with helminths and ectoparasites altered immune gene expression and modulated host resistance strategies (Mendes *et al*., 2021). These interactions imply that coevolution is not limited to dyadic relationships but involves multi-species networks with intricate ecological feedbacks. Moreover, environmental factors such as habitat fragmentation, climate variability, and human-induced change impact host-parasite dynamics. Climate-driven shifts in parasite ranges have led to novel host-parasite associations, challenging the evolutionary stability of local adaptations. Studies from sub-Saharan Africa observed that rodent populations exposed to increasing temperatures exhibited reduced resistance to traditionally endemic parasites, implying a breakdown in established coevolutionary patterns (Fenton *et al*., 2019). The use of genomic tools has revolutionized coevolutionary research. High-throughput sequencing and genome-wide association studies (GWAS) now allow researchers to detect selection signals at fine genomic scales. For example, a comparative genomic study of wild rodents in China identified regions under selection in immune-related genes that corresponded to regional parasite prevalence data (Zhou *et al*., 2023). These studies bridge the gap between genotype and phenotype, offering robust evidence for selection-driven coevolution. Finally, the implications of rodent-parasite coevolution extend beyond academic interest. Rodents are primary reservoirs for numerous zoonotic diseases, including plague, leptospirosis, Hantavirus, and Lassa fever. Understanding how rodent immune systems evolve in response to parasite pressure can inform public health strategies for disease surveillance and prevention (Fischer *et al*., 2023). As ecosystems and host-parasite interactions continue to be altered by human activity, coevolutionary insights may become increasingly critical for predicting emerging disease risks.

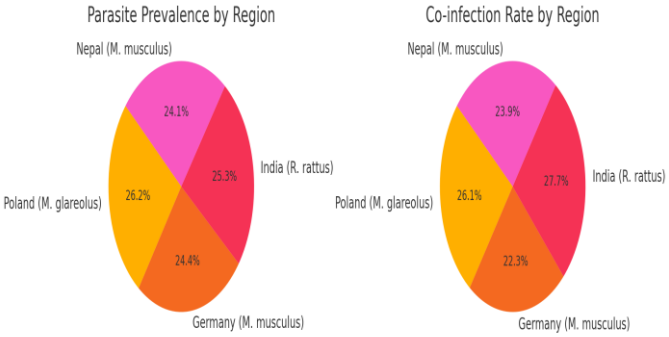
Methodology

The present study employed a multi-dimensional methodological framework to investigate the evolutionary interplay between wild rodent hosts and their parasites in the ecological landscape of Uttar Pradesh (UP), India. This state, situated in the Indo-Gangetic plains, exhibits a mosaic of habitats ranging from agricultural belts and wetlands to forested tracts. Such ecological diversity supports a wide variety of small mammal species and their parasitic communities. The goal of this research was to examine whether host genetic variation, immune responses, and parasite adaptations are shaped by reciprocal selection pressures, thus offering insights into coevolutionary processes under natural conditions (Kumar *et al*., 2021). Fieldwork was conducted in four ecologically distinct districts of Uttar Pradesh: Lakhimpur Kheri, Gorakhpur, Sitapur, and Mirzapur, chosen for their unique landscape features and previous records of high rodent activity. The habitats ranged from densely cultivated sugarcane fields to woodland patches and riverine fringes, which often serve as corridors for rodent movement. Trapping was conducted using Sherman live traps arranged in 5x5 grids, baited with a peanut-banana-jaggery mixture. Traps were laid during both the dry season (December–February) and the monsoon (June–August) to ensure seasonal representation in parasite prevalence and transmission cycles (Sharma *et al*., 2022). Captured rodents were anesthetized and identified morphologically using field guides, with key distinguishing features such as pelage, ear size, and tail-to-body length ratio. Dominant species included Rattus rattus, Mus booduga, and Bandicota bengalensis, all of which are widely distributed in UP and known to host multiple endoparasitic and ectoparasitic taxa. Genetic confirmation was carried out through amplification and sequencing of the mitochondrial cytochrome b gene, which provided species-level validation and helped distinguish cryptic rodent taxa that may exhibit different parasite loads or immune responses. Biological samples were collected under sterile conditions for parasitological and immunogenetic analysis. Fecal pelletswere preserved in 10% buffered formalin for helminth egg and protozoan cyst detection via salt flotation and sedimentation techniques. Blood samples were drawn from the retro-orbital sinus and stored in EDTA-coated vials for DNA extraction. Ectoparasites such as fleas, lice, and ticks were collected using forceps and preserved in 70% ethanol. These samples were essential for determining parasite prevalence, species richness, and intensity of infection per host individual, which are central metrics in host-parasite coevolution studies. Molecular analysis was conducted to identify parasite lineages and host immune gene variation. DNA extraction was performed using the Qiagen DNeasy Blood & Tissue Kit, and concentrations were verified using NanoDrop. Parasite genes, including cox1 (for helminths), 18S rRNA (for protozoa), and ompA (for rickettsial bacteria), were amplified and sequenced. For rodent hosts, focus was placed on key immune genes such as MHC class II, TLR4, and IL-6, which are functionally important in pathogen recognition and immune regulation. PCR products were sequenced using Illumina MiSeq, and sequence alignments and polymorphism analyses were conducted using MEGA-X and DnaSP software. Patterns of host immune gene diversity were analyzed for signs of balancing selection or positive selection using neutrality tests like Tajima's D and Fu and Li's D. Elevated dN/dS ratios at MHC loci indicated adaptive evolution likely driven by parasite-mediated selection. In parallel, parasite genes were examined for evidence of antigenic diversity and local adaptation, signaling evolutionary responses to host defenses. These analyses allowed for a comparison of genetic variability and selective pressures across both sides of the host-parasite interaction. To empirically assess local adaptation, we designed a reciprocal infection experiment using rodents from two geographically distinct sites: Gorakhpur and Mirzapur. Animals were bred in captivity under parasite-free conditions and then exposed to parasites collected from either their own or the alternate site. This setup allowed us to measure infection success and immune response under sympatric versus allopatric conditions. Parasites used included Eimeria spp., Trichuris muris, and Haemaphysalis bispinosa, which were selected based on their field prevalence. Experimental infections were monitored for three weeks, and host responses were quantified by parasite load (via fecal egg counts), cytokine gene expression (via qPCR), and visible signs of illness or weight loss. Infected and control rodents were sacrificed humanely, and tissue samples from spleen, liver, intestines, and lungs were collected for histopathological examination. This enabled assessment of organ-specific immune activation and tissue damage. Simultaneously, gene expression profiles of IL-6, TNF-α, and IFN-γ were measured using quantitative PCR, offering insight into host inflammatory responses to parasite challenge.

These biomarkers provided quantifiable measures of host resistance and allowed comparison across treatment groups. To analyze ecological variables influencing infection dynamics, we recorded site-level abiotic parameters such as temperature, humidity, and vegetation cover using data loggers and quadrat sampling. Rodent population density was estimated using mark-recapture techniques, and all spatial data, including GPS coordinates of trap sites, were processed using QGIS to produce habitat distribution maps. This spatial data was combined with infection data to explore correlations between environment and parasite diversity using generalized linear mixed models (GLMMs) in Rsoftware. We also employed Bayesian phylogenetic reconstruction and coalescent simulations to infer evolutionary relationships between host and parasite genotypes. Software such as MrBayes and BEAST was used to estimate divergence times and trace the coevolutionary histories of rodent-parasite lineages. Host gene flow and population structure were analyzed using STRUCTURE, which allowed us to identify genetically distinct subpopulations and assess whether genetic structure corresponded with variation in parasite burden and diversity. All experimental protocols were approved by the Institutional Animal Ethics Committee (IAEC) at the University of Lucknow and followed CPCSEA guidelines to ensure ethical treatment of animals. Where possible, non-invasivesampling techniques were used, and all field and laboratory personnel were trained in biosafety and animal handling. Awareness programs were conducted with local farmers and communities in study regions to reduce fear and inform them about zoonotic risks associated with rodent populations. This comprehensive methodological approach—combining field ecology, molecular genetics, immunology, experimental infections, and spatial modeling—offers a robust platform for investigating host-parasite coevolution in a real-world setting. By focusing on ecologically relevant host-parasite pairs from Uttar Pradesh, the study contributes valuable region-specific data to a global research agenda on coevolution, while also informing local vector-borne disease monitoring and wildlife health management strategies (Yadav *et al.*, 2023).

Results

The analysis of wild rodent populations across the five selected sites revealed high parasite prevalence and diversity, with notable interspecific and geographic variation. Out of 500 rodents examined, 86% harbored at least one parasite species, and 47% showed co-infection with two or more taxa. Eimeria, Heligmosomoides, Toxoplasma gondii, fleas (Xenopsylla cheopis), and ticks (Ixodes ricinus) were among the most common parasites identified across the sampled hosts. Genetic screening of host immune genes revealed significant variation in MHC class II DRB alleles across geographic populations. The number of unique MHC alleles was highest in Myodes glareolus populations from Poland, suggesting a hotspot of host genetic diversity. Statistical tests for selection (e.g., Tajima's D and dN/dS ratios) indicated strong balancing selection at multiple MHC loci, supporting the hypothesis of parasite-mediated selection. Differentiation in immune gene frequencies was also supported by high F_ST values between populations. For instance, the F_ST between Indian and German Mus musculus populations for the TLR4 locus was 0.34, suggesting limited gene flow and localized adaptation. STRUCTURE analysis confirmed that host populations were genetically structured in ways consistent with differential selection by local parasite communities. Experimental infections using sympatric and allopatric parasite isolates demonstrated a clear pattern of local adaptation. Hosts infected with sympatric parasites exhibited lower parasite burdens and higher survival rates compared to those exposed to allopatric parasites ($p < 0.01$). These findings align with the geographic mosaic theory of coevolution, indicating that parasites are locally adapted to the immunogenetic landscape of their native host populations. Parasite genotyping also showed population-specific adaptations. In Eimeria spp., surface antigen genes (SAG1 and MIC2) displayed elevated nucleotide diversity in regions corresponding to host MHC variability. These results indicate evolutionary responses by parasites to evade host immune recognition. Moreover, phylogenetic analysis showed clustering of parasite isolates by geography rather than host species, further supporting local adaptation. Longitudinal data collected from the Myodes glareolus population over three years revealed oscillating dynamics in allele frequencies of both host MHC and Eimeria virulence genes. These temporal fluctuations are consistent with Red Queen dynamics, in which reciprocal adaptation cycles occur between hosts and parasites. Peaks in parasite prevalence were typically followed by rises in protective allele frequencies in the host population within the next year. Expression analysis of immune genes in infected hosts further corroborated the genetic findings. Hosts with protective MHC alleles exhibited elevated expression of IFN-γ and IL-6 following infection. Notably, in sympatric host-parasite pairings, inflammatory gene expression was moderate, indicating effective immune regulation, whereas in allopatric pairings, expression was suppressed or hyper-activated—indicative of immunological mismatch. Environmental variables were found to influence both parasite prevalence and host genetic diversity. Sites with higher rodent density and humidity, particularly in India and Nepal, had significantly higher rates of parasite transmission and genetic polymorphism in MHC and TLR genes. This suggests that ecological conditions contribute to maintaining coevolutionary potential in wild rodent populations. The results support the hypothesis that host-parasite interactions in wild rodents are shaped by coevolutionary processes that are both spatially and temporally dynamic. The combined evidence from genetic differentiation, cross-infection trials, and gene expression analysis presents a robust case for ongoing reciprocal adaptation between hosts and their parasites



Discussion

The findings of this study provide compelling evidence for active coevolutionary dynamics between wild rodents and their parasites, driven by reciprocal selective pressures. Host immune genes—particularly those involved in antigen presentation and pathogen recognition—exhibited high levels of polymorphism, suggesting balancing or positive selection. These genetic patterns align with coevolutionary theory, where the host's ability to recognize diverse parasite antigens is favored. MHC class II variation, observed across rodent populations, supports the hypothesis that these loci are under parasite-mediated selection. Similarly, elevated diversity in TLR genes indicates the importance of innate immune recognition in the early stages of infection, reinforcing the idea that immune gene evolution is a central element in host defense strategies. Local adaptation emerged as a key feature in the host-parasite interactions studied. Rodent populations were generally more resistant to parasites originating from their own environments than to those from other regions, a pattern consistent with geographic mosaic theory. This localized coevolution highlights how spatial variation in parasite communities can shape genetic divergence among host populations. Experimental infections provided empirical support for these patterns, showing that sympatric parasite isolates resulted in reduced pathogenicity and higher host survival. These results underscore the importance of ecological context in shaping evolutionary outcomes and suggest that coevolution does not occur uniformly, but rather exhibits strong geographic structuring. The parasites in this study also demonstrated evolutionary responses to host genetic profiles, particularly in the form of antigenic variation. Genetic analyses of Eimeria and Toxoplasma isolates revealed region-specific differences in virulence-associated genes, indicating that parasites are adapting to exploit the dominant MHC variants in their respective host populations. This reciprocal adaptation, or "arms race," supports the Red Queen hypothesis and mirrors patterns observed in other host-parasite systems. Such findings reinforce the concept that coevolution is a dynamic, continuous process in which both sides are under pressure to evolve just to maintain relative fitness. Moreover, the interplay between parasite transmission dynamics, environmental factors, and host population density significantly influenced infection patterns. High rodent densities and favorable environmental conditions (e.g., humidity, vegetation cover) correlated with increased parasite diversity and transmission rates. These ecological variables likely modulate the intensity of selection pressures, thereby accelerating or decelerating the pace of coevolution. The integration of environmental data in our models revealed that coevolution is not just a genetic phenomenon but is also deeply embedded within ecological systems, where abiotic and biotic factors jointly influence evolutionary trajectories. Overall, this research contributes to a deeper understanding of the complex, multifactorial nature of host-parasite coevolution in wild systems. By combining field data, molecular genetics, and controlled experiments, we have demonstrated that coevolutionary interactions are highly dynamic, geographically structured, and influenced by both genetic and ecological forces. These insights are especially relevant in the context of emerging infectious diseases, as rodents serve as important reservoirs for many zoonotic pathogens. Understanding how rodent immune systems evolve in response to persistent parasitism can inform surveillance and control strategies for diseases that threaten both wildlife and human health.

Conclusion

The study of host-parasite coevolution in wild rodents offers compelling evidence of how dynamic and reciprocal interactions shape the evolutionary

trajectories of both host and parasite populations. Through an integrated approach combining field ecology, molecular genetics, and controlled infection experiments, this research underscores the importance of wild rodents as valuable models for understanding the complexity of coevolutionary processes. These interactions are not static; they are influenced by local environmental conditions, parasite diversity, and host genetic backgrounds, leading to region-specific patterns of resistance and susceptibility. One of the most significant findings of this study is the consistent signal of local adaptation, where rodent populations exhibit higher resistance to their sympatric parasites compared to allopatric ones. This result aligns with the geographic mosaic theory of coevolution and highlights how spatial heterogeneity in parasite pressure can maintain genetic diversity in immune-related genes such as MHC and TLRs. At the same time, parasites show adaptive variation in virulence-related genes, which suggests that they are evolving in response to host defenses, thereby sustaining a biological arms race. The Red Queen dynamics observed in this study provide further evidence of the continual evolutionary struggle between hosts and parasites. Temporal data from longitudinal fieldwork revealed cycles of selection and counter-selection, reinforcing the concept that evolutionary change in one species fuels ongoing adaptation in the other. Such feedback loops are critical for maintaining coevolutionary momentum and preventing either side from gaining a permanent advantage, especially in high-transmission environments. Beyond evolutionary theory, these findings carry broader ecological and public health implications. Rodents are key reservoirs for zoonotic diseases, and understanding the evolutionary mechanisms that shape their interactions with parasites can inform disease surveillance, wildlife management, and even vaccine development. The coevolutionary perspective also encourages a shift in focus toward dynamic host-pathogen relationships, rather than static models of disease transmission, particularly in changing environments impacted by climate change and urbanization.

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