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Mechanisms of Differential Infection in Wildlife: Factors Influencing Susceptibility and Resistance

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Abstract Wildlife disease dynamics are influenced by a complex interplay of factors that determine differential infection and resistance among species. This study explores the genetic, environmental, behavioral, and microbiome-related mechanisms that influence susceptibility and resistance in wildlife. Through an analysis of genetic variation, environmental stressors, behavioral patterns, and microbiome diversity, this study identifies key factors contributing to differential infection rates. A detailed case study highlights how these factors converge in a specific wildlife species, offering insights into broader ecological and conservation implications. The findings underscore the importance of integrating susceptibility research into conservation strategies and suggest targeted approaches to enhance resistance in vulnerable species. This study aims to inform conservation policies and management practices, promoting a multidisciplinary approach to wildlife disease management and future research directions.
Keywords Wildlife disease dynamics; Differential infection; Susceptibility factors; Conservation strategies; Pathogen resistance

1 Introduction

Infectious diseases are increasingly recognized as significant drivers of population dynamics, conservation biology, and natural selection in wildlife populations. Pathogens can influence population size, distribution, growth rates, and migration patterns, often leading to the decline of small or endangered populations. The interaction between host genetics and disease susceptibility is a critical factor in understanding these dynamics. For instance, genetic variation in the prion protein gene (PRNP) has been linked to differential susceptibility to chronic wasting disease (CWD) in white-tailed deer, demonstrating how genetic differences can influence natural selection within wildlife populations (Robinson et al., 2012). Additionally, the movement of animals, whether through migration or other transient phases, plays a crucial role in the spread of diseases, further complicating the dynamics of wildlife diseases (Daversa et al., 2017).

Understanding the mechanisms behind differential infection in wildlife is essential for several reasons. Firstly, it helps in predicting and managing disease outbreaks, which is crucial for conservation efforts and maintaining biodiversity (DeCandia et al., 2018). For example, the differential susceptibility of cattle breeds to tropical theileriosis highlights the importance of host-pathogen interactions in determining disease outcomes (Larcombe et al., 2019). Secondly, it provides insights into the evolutionary pressures exerted by pathogens on host populations, which can lead to significant changes in genetic diversity and population structure over time (Jiao and Fefferman, 2021). Moreover, understanding how anthropogenic factors, such as urbanization and agriculture, influence wildlife-pathogen dynamics can inform strategies to mitigate the negative impacts of human activities on wildlife health (Becker et al., 2015).

This study aims to review and synthesize current knowledge on the factors influencing susceptibility and resistance to infectious diseases in wildlife. By examining genetic, behavioral, and ecological factors, this study provides a comprehensive understanding of the mechanisms driving differential infection; explores how genetic variation, host behavior, and environmental factors contribute to disease dynamics and the implications for wildlife conservation and management; additionally, highlights areas for future research to address gaps in our understanding and improve predictive models for wildlife disease management.

2 Genetic Factors Influencing Susceptibility and Resistance

2.1 Genetic variation among species and populations

Genetic variation in susceptibility to infections is a well-documented phenomenon across various species and populations. Studies have shown that natural selection by pathogens can increase genetic variation in host populations, particularly in those that have coevolved with their pathogens. For instance, research involving Drosophila species and their host-specific viruses demonstrated greater genetic variation in susceptibility to coevolved viruses compared to novel pathogens, suggesting that major-effect resistance polymorphisms play a significant role in this variation (Duxbury et al., 2018). Similarly, in dipteran insects like Drosophila melanogaster and vector mosquitoes, genetic and microbiota-dependent variations significantly influence antiviral immunity and virus susceptibility (Palmer et al., 2018). These findings underscore the importance of genetic diversity in shaping the susceptibility and resistance profiles of different species and populations.

2.2 Role of host genetics in immune response

Host genetics play a crucial role in modulating immune responses to infections. Genetic variants in immune response genes, such as cytokines, have been linked to differences in susceptibility to parasitic and microbial infections. For example, polymorphisms in cytokine genes like TNF, LTα, and IFNβ1 have been associated with varying susceptibility to nematodes and microbial pathogens in bank voles, highlighting the role of non-coding variants in immune regulation (Figure 1) (Kloch et al., 2021). Additionally, studies on human populations have identified numerous genes associated with severe viral infections, including those involved in TLR pathways and inflammasome activation, which are critical for immune response modulation (Elhabyan et al., 2020). These genetic determinants are essential for understanding the mechanisms underlying differential immune responses and susceptibility to infections.

The study of Kloch et al. (2021) shows the impact of different single nucleotide polymorphisms (SNPs) in LTα and IFNβ genes on the risk of infection and parasite burden in voles. The graphs suggest that specific genotypes at these SNP loci significantly influence the likelihood of infection and the intensity of parasitism. For instance, certain alleles appear to increase the risk of infection, as indicated by the higher percentage of infected individuals with those genotypes. Additionally, the intensity of infection varies among different genotypes, indicating that genetic variation plays a crucial role in host susceptibility to parasites.

2.3 Evolutionary adaptations to pathogens

Evolutionary adaptations to pathogens are evident in the genetic architecture of resistance traits. Host-pathogen coevolution often results in the selection of major-effect genes that confer resistance to infections. In Drosophila, for example, resistance to viruses like the sigma virus and DCV is largely controlled by a few major-effect loci, supporting the idea that pathogen-driven selection can simplify the genetic architecture of resistance traits (Cogni et al., 2016). Furthermore, gene expression studies in threespine stickleback populations have revealed population-specific immune responses to parasite infections, indicating that recent evolutionary divergence can lead to distinct genetic adaptations that enhance resistance (Lohman et al., 2017). These adaptations are crucial for the survival and fitness of host populations in the face of ongoing pathogen pressures.

In summary, genetic factors, including variation among species and populations, host genetics in immune response, and evolutionary adaptations, play pivotal roles in influencing susceptibility and resistance to infections in wildlife. Understanding these mechanisms provides valuable insights into the complex interplay between hosts and pathogens, which is essential for developing effective strategies for managing infectious diseases in natural populations.

3 Environmental Factors Affecting Infection Rates

3.1 Influence of habitat quality and availability

Habitat quality and availability play a crucial role in determining the infection rates among wildlife populations. Changes in habitat, often driven by anthropogenic activities, can lead to shifts in species diversity and population densities, which in turn influence the prevalence of pathogens. For instance, the modification of continuous habitats into isolated patches has been linked to changes in species assemblages and increased prevalence of

certain viruses in dominant host species (Schmid et al., 2018). Additionally, urbanization and agriculture can alter wildlife ecology by providing novel resources, such as food, which can either enhance or reduce disease transmission depending on the specific pathogen and host interactions (Becker et al., 2015). The availability of high-quality habitats can support better nutrition and immune function in wildlife, potentially reducing susceptibility to infections (Becker et al., 2019).

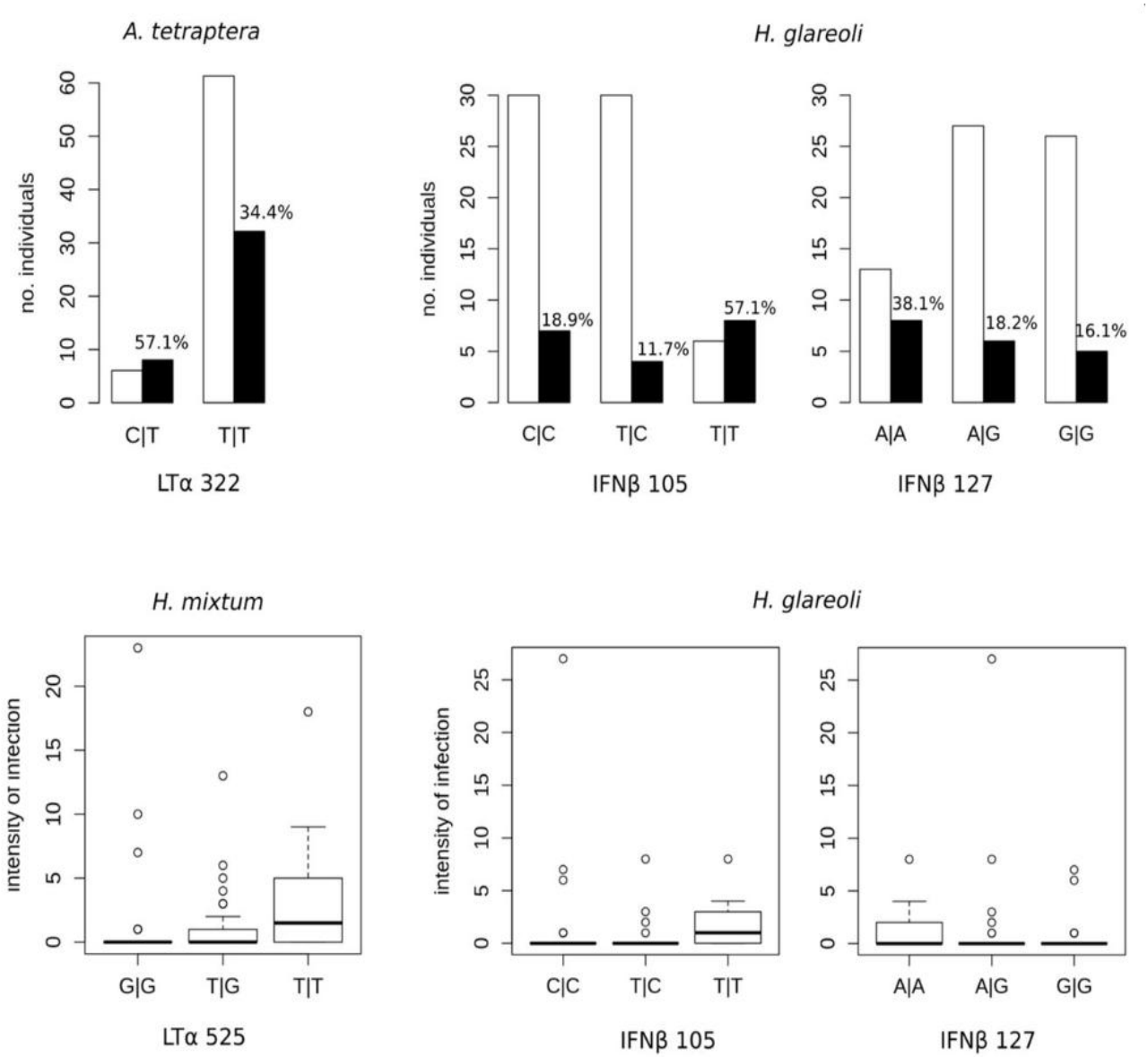

Figure 1 Effect of SNP genotype in a) *LTα* and b) *IFNβ* on the risk of infection and parasite abundance. % of infected voles is given in graphs where the allele affected the risk of infection (Adopted from Kloch et al., 2021)

3.2 Impact of climate change on disease spread

Climate change significantly impacts the spread of infectious diseases in wildlife by altering the interactions between hosts, pathogens, and vectors. Changes in temperature and weather patterns can affect the distribution, life cycles, and physiological status of these organisms, leading to shifts in disease dynamics (Gallana et al., 2013). For example, the thermal mismatch hypothesis suggests that hosts adapted to cooler climates experience increased disease risk during abnormally warm periods, while those from warmer climates face higher risks during cooler periods (Cohen et al., 2020). This effect is particularly pronounced in ectothermic hosts, whose immune responses are highly temperature-dependent. Climate change can also modulate disease through changes in ecological networks and interactions with other environmental stressors, leading to complex and non-linear responses in disease systems (Hemert et al., 2014).

3.3 Environmental stressors and immunosuppression

Environmental stressors, such as food limitation, pollution, and habitat loss, can lead to immunosuppression in wildlife, increasing their susceptibility to infections. Food stress, for instance, has been shown to elevate virus titres and prolong infectious periods in hosts, thereby enhancing transmission dynamics (Figure 2) (Owen et al., 2021). Similarly, pollution and other abiotic stressors can decrease host survivorship and increase pathogen intensity, further complicating disease management efforts (Vicente-Santos et al., 2023). The physiological stress response induced by these environmental factors can compromise immune function, making wildlife more vulnerable to diseases and potentially exacerbating the impact of emerging infectious diseases on species at risk of extinction (Hing et al., 2016). Understanding the interplay between stressors and disease dynamics is critical for predicting and mitigating zoonotic disease outbreaks (Norte et al., 2021).

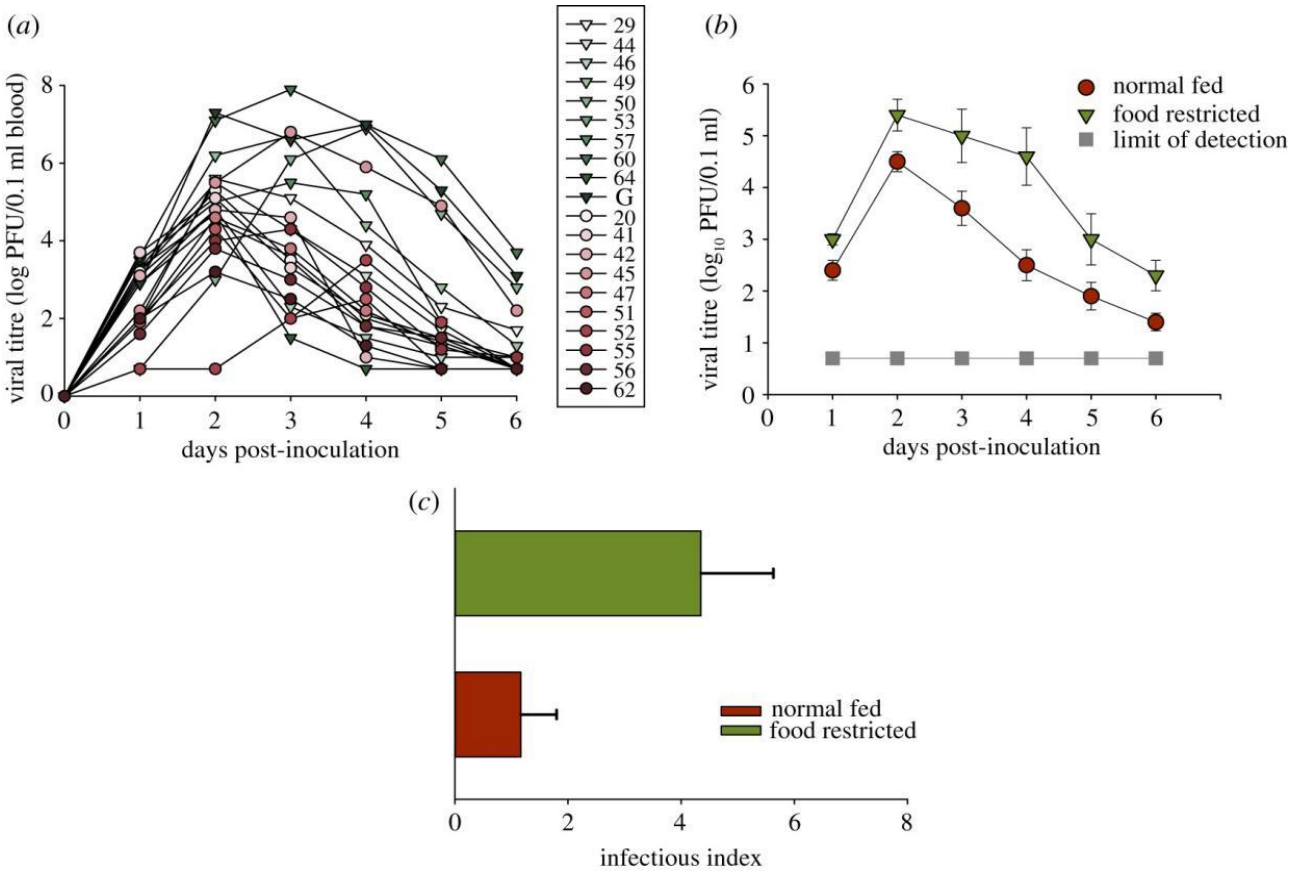

Figure 2 (a) WNV titres (log PFU/0.1 ml blood) for experimentally infected American robins fed normally (red circles; *n*=11) and robins food-deprived for 48 h prior to inoculation (green down triangles; *n*=10). (b) Average (±1 s.e.) virus titre per log PFU/0.1 ml blood for normal (red circles) and food-restricted (green down triangles) robins. Virus titres below 0.7 log pfu/0.1 ml are undetectable (grey squares) via Vero cell plaque assay. (c) Mean (±1 s.d.) infectious (i.e. capable of infecting a biting mosquito) index for the two groups (Adopted from Owen et al., 2021)

The study of Owen et al. (2021) illustrates the effect of food restriction on West Nile virus (WNV) infection in American robins. It shows that robins subjected to a 48-hour food deprivation prior to inoculation had higher WNV titres in their blood compared to those fed normally. This suggests that food restriction might increase the susceptibility or enhance the replication of the virus in these birds, making them more infectious. The data underscores the importance of nutritional status in modulating host-pathogen interactions, potentially affecting the spread of vector-borne diseases like WNV.

By integrating insights from various studies, it becomes evident that environmental factors, including habitat quality, climate change, and stressors, play a pivotal role in shaping the susceptibility and resistance of wildlife to infections. Addressing these factors through conservation and management strategies is essential for mitigating disease risks and preserving biodiversity.

4 Behavioral Factors Contributing to Differential Infection

4.1 Social behavior and disease transmission

Social behavior plays a critical role in the transmission of infectious diseases among wildlife populations. Individuals that are more socially active or occupy central positions within social networks are often at higher risk of infection due to increased contact rates with conspecifics. For instance, in a study on red-capped mangabeys, individuals that were central and well-connected within their social networks exhibited a higher risk of gastrointestinal parasite infection (Friant et al., 2016). Similarly, the social structure of host populations, including group size and social interactions, can significantly influence disease dynamics, as demonstrated in various wildlife species (Hawley et al., 2011). The non-random mixing of individuals within social networks can impact the demographic thresholds that determine disease amplification or attenuation, highlighting the importance of considering social behavior in disease management strategies (Silk et al., 2019).

4.2 Foraging and movement patterns

Foraging behavior and movement patterns are also crucial determinants of differential infection in wildlife. Animals that forage in specific habitats or exhibit particular movement patterns may experience varying levels of exposure to pathogens. For example, Bewick's swans that foraged in aquatic habitats were found to have a higher risk of avian influenza virus infection compared to those foraging in terrestrial habitats, due to the abiotic requirements of the virus (Hoye et al., 2012). Additionally, urbanization and agriculture can alter wildlife foraging behavior, leading to changes in disease dynamics. Provisioned food sources in human-dominated habitats can either amplify or reduce pathogen transmission depending on factors such as host aggregation and dietary exposure to parasites (Becker et al., 2015). These findings underscore the importance of understanding how foraging and movement behaviors influence pathogen exposure and infection risk in wildlife populations.

4.3 Behavioral avoidance and resistance mechanisms

Behavioral avoidance and resistance mechanisms are strategies employed by wildlife to mitigate infection risk. Some individuals may alter their behavior to avoid contact with infected conspecifics or environments that pose a high risk of pathogen exposure. For instance, certain male guppies modify their social behavior to avoid infection, with more susceptible males exhibiting reduced sociality to decrease their risk of parasite transmission (Stephenson, 2019). Additionally, behavioral changes in response to infection, such as sickness behaviors, can influence disease dynamics by reducing contact rates and transmission potential. Understanding these behavioral avoidance and resistance mechanisms is essential for developing effective disease management and conservation strategies, particularly for species threatened by emerging infectious diseases (Brannelly et al., 2020).

In summary, behavioral factors such as social behavior, foraging and movement patterns, and behavioral avoidance mechanisms play significant roles in influencing susceptibility and resistance to infections in wildlife. These behaviors can modulate both exposure to pathogens and the likelihood of infection, thereby shaping the dynamics of disease transmission within and among wildlife populations.

5 Microbiome Influence on Disease Susceptibility

5.1 Microbiome diversity and pathogen defense

Microbiome diversity plays a crucial role in the defense against pathogens. Studies have shown that higher microbiome richness is often correlated with increased resistance to infections. For instance, research on the frog *Rana sierrae* demonstrated that populations with higher skin microbiome richness were more likely to persist in the presence of the fungal pathogen B*atrachochytrium dendrobatidis* (Bd) (Jani et al., 2017). Similarly, the gut microbiome diversity in European common frogs (*Rana temporaria*) was linked to higher survival rates when exposed to the Ranavirus, suggesting that a diverse microbiome can enhance disease resistance (Harrison et al., 2019). However, this relationship is not always straightforward. In bumblebees (*Bombus terrestris*), higher gut microbiota diversity was associated with lower resistance to the intestinal parasite *Crithidia bombi*, indicating that the specific composition of the microbiome, rather than just its diversity, is critical for effective pathogen defense (Näpflin and Schmid-Hempel, 2018).

5.2 Impact of co-infections on health

Co-infections can significantly impact the health of wildlife by altering the microbiome and influencing disease outcomes. The presence of multiple pathogens can lead to complex interactions within the host's microbiome, which can either exacerbate or mitigate disease severity. For example, the study on amphibian skin microbiomes revealed that the severity of Bd infection influenced the composition of the microbiome, with certain bacterial phylotypes changing in abundance as the infection progressed (Jani and Briggs, 2018). This dynamic interaction suggests that co-infections can disrupt the stability of the microbiome, potentially leading to increased susceptibility to additional pathogens. Moreover, the gut microbiome of American white ibises (Eudocimus albus) showed that urbanization and diet changes, which can be considered as environmental co-factors, were associated with shifts in microbiome composition and increased prevalence of Salmonella enterica, highlighting the role of environmental factors in co-infection dynamics (Murray et al., 2019).

5.3 Symbiotic relationships and disease resistance

Symbiotic relationships between hosts and their microbiomes are fundamental to disease resistance. Symbiotic bacteria can provide colonization resistance against pathogens through various mechanisms, including competitive exclusion, production of antimicrobial compounds, and modulation of the host immune response (Tan et al., 2012; Libertucci and Young, 2018). For instance, the skin microbiome of amphibians has been shown to play a protective role against Bd infection, with certain bacterial communities being linked to host-pathogen coexistence rather than population extirpation. Additionally, the gut microbiome of cheetahs (*Acinonyx jubatus*) demonstrated that genetic relatedness and environmental factors shape the microbial community, which in turn influences the occurrence of potential pathogens and overall health (Wasimuddin et al., 2017). These symbiotic relationships underscore the importance of maintaining a healthy and diverse microbiome for effective disease resistance in wildlife.

In summary, the microbiome's influence on disease susceptibility in wildlife is multifaceted, involving the diversity and composition of microbial communities, the impact of co-infections, and the symbiotic relationships that enhance host defense mechanisms. Understanding these interactions is crucial for developing strategies to mitigate the effects of infectious diseases in wildlife populations.

6 Case Study: Differential Infection in Andean Condors

6.1 Detailed analysis of the case study

The Andean Condor (*Vultur gryphus*) serves as a critical case study for understanding differential infection in wildlife. These birds, essential for ecosystem health, are near threatened and face declining populations due to anthropogenic activities. Recent studies have identified pandemic lineages of multidrug-resistant extended-spectrum β-lactamase (ESBL)-producing Escherichia coli in Andean Condors. These pathogens, typically associated with hospital and healthcare environments, were found in condors admitted to wildlife rehabilitation centers in South America. The genomic analysis revealed resistance genes to clinically important cephalosporins and other substances, indicating a significant link to environmental pollution caused by human activities (Fuentes-Castillo et al., 2020).

6.2 Genetic, environmental, and behavioral factors

The susceptibility and resistance to infections in Andean Condors are influenced by a combination of genetic, environmental, and behavioral factors. Genetically, the presence of specific resistance genes such as *CTX-M-14*, *CTX-M-55*, and *CTX-M-65* in E. coli strains indicates a genetic predisposition to harboring these pathogens. Environmentally, the contamination of habitats with pollutants from human activities, including heavy metals, pesticides, and disinfectants, plays a crucial role in the spread of these resistant bacteria. Behaviorally, the scavenging nature of condors exposes them to a variety of pathogens present in carcasses and waste, increasing their risk of infection (Duxbury et al., 2018).

6.3 Lessons and broader implications

The case of Andean Condors highlights the broader implications of differential infection in wildlife. Firstly, it underscores the significant impact of anthropogenic activities on wildlife health, particularly through

environmental pollution. Secondly, it demonstrates the role of wildlife as reservoirs and vectors for the dissemination of antimicrobial-resistant bacteria, which can have far-reaching consequences for both animal and human health. This case study emphasizes the need for integrated public and ecosystem health policies, improved surveillance, and control strategies to mitigate the spread of antimicrobial resistance. The findings also suggest that conservation efforts must consider the complex interplay of genetic, environmental, and behavioral factors to effectively protect vulnerable wildlife populations (Palmeira et al., 2021; Laborda et al., 2022).

7 Implications for Conservation and Disease Management

7.1 Integrating susceptibility research into conservation

Understanding the mechanisms behind differential susceptibility and resistance to diseases in wildlife is crucial for effective conservation strategies. Research has shown that genetic variation plays a significant role in how different species and even individuals within a species respond to pathogens. For instance, studies on white-tailed deer have demonstrated that genetic differences in the prion protein gene (PRNP) significantly impact susceptibility to chronic wasting disease (CWD), with resistant genotypes showing lower infection rates and higher survival (Robinson et al., 2012). Similarly, research on amphibians has identified species-level characteristics, such as body size and reproductive behaviors, that influence susceptibility to the fungal pathogen *Batrachochytrium dendrobatidis* (Bd) (Bancroft et al., 2011). By integrating these findings into conservation plans, we can prioritize efforts on the most vulnerable species and develop targeted interventions to mitigate disease impacts.

7.2 Strategies for enhancing resistance in vulnerable species

Enhancing resistance in vulnerable wildlife populations can be achieved through several strategies. One approach is selective breeding programs that focus on increasing the prevalence of resistant genotypes within a population. For example, the identification of genetic markers associated with disease resistance in elephants has opened up possibilities for breeding programs aimed at enhancing resistance to diseases like tuberculosis and elephant endotheliotropic herpesvirus (EEHV) (Tollis et al., 2021). Another strategy involves habitat management to reduce disease transmission. Studies have shown that environmental factors, such as the availability of refugia and changes in community composition, can influence host persistence following disease outbreaks (Brannelly et al., 2020). By creating environments that support the survival of resistant individuals and reduce pathogen load, we can enhance the overall resilience of wildlife populations.

7.3 Policy and management recommendations

Wildlife monitoring is a critical component of conservation biology, ecology, and environmental management (Zhu, 2024). Effective disease management in wildlife requires a multifaceted approach that incorporates both scientific research and practical interventions. Policymakers should prioritize funding for research that explores the genetic and environmental factors influencing disease susceptibility and resistance. This includes supporting studies that use advanced molecular techniques to understand host-pathogen interactions and identify key genetic markers of resistance (Eskew et al., 2021; Morris et al., 2023). Additionally, management practices should be informed by social network models that consider the demographic and behavioral aspects of disease transmission (Silk et al., 2019). For instance, understanding the social structure of host populations can help in designing interventions that minimize contact between infected and susceptible individuals. Finally, policies should promote the conservation of genetic diversity within wildlife populations, as this diversity is crucial for the long-term adaptability and resilience of species to emerging infectious diseases (Duxbury et al., 2018).

By integrating susceptibility research into conservation efforts, enhancing resistance through targeted strategies, and implementing informed policy and management practices, we can better protect wildlife populations from the devastating impacts of infectious diseases.

8 Concluding Remarks

Differential infection in wildlife is influenced by a variety of mechanisms, including genetic variability, pathogen-host interactions, and environmental factors. Genetic differences among individuals and species can significantly impact susceptibility to diseases, as seen in the case of chronic wasting disease in white-tailed deer,

where specific genotypes confer resistance or susceptibility to infection. Similarly, the genetic basis of resistance to infections such as HIV in humans highlights the role of host genetic heterogeneity in disease progression and susceptibility. Additionally, the presence of co-infecting pathogens can alter the host's susceptibility to other infections, as demonstrated in wild voles where co-infections had a larger effect on disease dynamics than other factors. Environmental factors, including anthropogenic pressures, also play a crucial role in the spread of antimicrobial resistance in wildlife, as evidenced by the widespread dissemination of cephalosporinases in various wildlife species due to human activities.

Addressing the complexities of differential infection in wildlife necessitates a multidisciplinary approach. Integrating genetic, ecological, and environmental studies can provide a comprehensive understanding of disease dynamics. For instance, the study of antimicrobial resistance in wildlife has benefited from the One Health approach, which considers the interconnectedness of human, animal, and environmental health. Multidisciplinary research has also been pivotal in identifying the role of wildlife in the transmission of bacterial pathogens and antimicrobial resistance to the food chain, highlighting the need for collaborative efforts across various scientific disciplines. Furthermore, the use of advanced molecular techniques and genome-wide analyses has provided new insights into host-pathogen interactions and the genetic basis of disease resistance, underscoring the importance of integrating diverse scientific methodologies.

Future research should focus on filling the existing knowledge gaps and addressing the emerging challenges in wildlife disease management. There is a need for more studies employing molecular methods to understand the direction and importance of pathogen transmission between wildlife and other ecosystems. Additionally, research should aim to identify point sources of antibiotic resistance and evaluate the effectiveness of management practices in mitigating the spread of resistance. Conservation efforts should prioritize the development of surveillance and control strategies to monitor and manage the spread of infectious diseases and antimicrobial resistance in wildlife populations. Understanding the genetic basis of disease resistance and susceptibility can also inform breeding programs and conservation strategies aimed at enhancing the resilience of wildlife populations to emerging diseases. Overall, a concerted effort involving multidisciplinary research and collaborative conservation initiatives is essential to address the complex challenges posed by differential infection in wildlife.

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Conflict of Interest Disclosure

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