A Review of Avian Pathogenic *Escherichia coli* (APEC) in Guinea Fowls (*Numida meleagris*) of Southern Africa: Antimicrobial Resistance and Virulence Genomics and Control Strategies.

Nyararai Yvonne O[1]., Ndau Blessing M[2]., Muzvondiwa John V[3]., Nyoni-Phili Sizanobuhle L[1].

[1]Department of Animal and Wildlife Sciences, Midlands State University, Gweru, Zimbabwe

[2]Department of Land and Water Management, Midlands State University, Gweru, Zimbabwe

[3]IFAD - Horticulture Enterprise Enhancement Project (HEEP). Climate Smart Agriculture and Environmental Safeguard Specialist; Masvingo Region

DOI: https://doi.org/10.51244/IJRSI.2026.13010152

Received: 27 January 2026; Accepted: 02 February 2026; Published: 10 February 2026

ABSTRACT

Avian Pathogenic *Escherichia coli* (APEC), the primary causative agent of colibacillosis, is a major and growing threat to global poultry production, leading to significant economic losses. In Southern Africa, the guinea fowl (*Numida meleagris*) is an economically, culturally, and nutritionally important indigenous poultry species for local communities. However, its production is constrained by infectious diseases such as colibacillosis posing a significant yet understudied challenge. This review synthesises the current, albeit limited, knowledge on APEC in Southern African guinea fowls. We detail the key virulence genes including adhesins (fimH), iron acquisition systems (iutA, fyuA), and immune evasion factors (iss) that facilitate pathogenesis. A critical concern is the convergence of these virulence genes with multidrug resistance (MDR) on mobile genetic elements. This fusion drives the emergence of dangerous, high-risk bacterial clones. Genomic studies confirm that APEC shares significant genetic overlap with human extra-intestinal pathogenic *E. coli* (ExPEC), such as sequence types ST95 and ST131, underscoring a substantial zoonotic threat. While conventional PCR has been instrumental in initial virulence profiling, we advocate for the widespread application of Whole-Genome Sequencing (WGS) to elucidate the unique population structure of guinea fowl APEC, precisely assess zoonotic risk, and guide interventions. Effective control requires an integrated, One Health approach encompassing stringent antibiotic stewardship, enhanced genomic surveillance, and the development of targeted strategies such as vaccines, phage therapy, and robust biosecurity measures tailored to guinea fowl farming systems. Filling these knowledge gaps is essential for safeguarding guinea fowl health, ensuring food security, and mitigating the public health crisis of antimicrobial resistance.

Keywords: Avian Pathogenic *E. coli* (APEC), Guinea Fowl, Virulence Genes, Antimicrobial Resistance, One Health.

INTRODUCTION

The guinea fowl (*Numida meleagris*) is a resilient poultry species indigenous to Africa, highly valued for its lean meat, eggs, and role in integrated pest control. In Southern Africa, guinea fowl farming is a vital component of rural livelihoods and a growing agricultural sector (Karikari et al., 2024). Despite their perceived hardiness, these birds are susceptible to infectious diseases, with colibacillosis standing out as a major constraint to productivity.

Colibacillosis, caused by Avian Pathogenic *Escherichia coli* (APEC), manifests as omphalitis, respiratory tract infections (air sacculitis), septicaemia, and peritonitis, leading to substantial mortality and morbidity (Barbieri et al, 2017).

APEC strains are a subset of extra-intestinal pathogenic *E. coli* (ExPEC) characterised by a diverse arsenal of virulence genes that facilitate colonisation, invasion, and survival within the avian host (Ilcebaylik and Turkyilmaz, 2020). Their pathogenicity is multifactorial, resulting from the cumulative effect of numerous virulence factors acting in concert (Kaper et al., 2004). While extensive research has mapped the virulence gene

landscape of APEC in commercial chickens globally (Ilcebaylik and Turkyilmaz, 2020), a pronounced paucity of comprehensive data exists for guinea fowls, particularly in Southern Africa.

Understanding the specific molecular epidemiology of APEC in this host is crucial for several reasons: it can reveal host-specific pathogenic adaptations; it informs the development of effective, tailored vaccines and diagnostics; and it helps assess the zoonotic potential of these strains, given that APEC shares genetic similarities with human ExPEC pathotypes such as uropathogenic *E. coli* (UPEC) (Mellata, 2013). This review consolidates the available literature on APEC in Southern African guinea fowls, systematically covering its classification, key virulence genes, detection methods, the critical intersection with antibiotic resistance, and future directions for research and control.

Classification And Transmission Routes Of APEC In Poultry

Classification of APEC

APEC strains are not a single, clonal entity but a diverse group of *E. coli* pathotypes classified based on their ability to cause extra-intestinal diseases in birds. They belong to the broader ExPEC group. Serotyping, based on O (somatic), H (flagellar), and K (capsular) antigens, has historically been used for classification, with serogroups O1, O2, and O78 being frequently associated with avian colibacillosis (Ilcebaylik and Turkyilmaz, 2020). However, serotyping has limitations in resolution and predictive power for virulence. Phylogenetically, APEC strains predominantly fall into groups B2 and D, which are also associated with human ExPEC, though they can be found in other phylogenetic groups (Landman et al, 2024). Modern classification increasingly relies on virulence gene-based profiling and multi-locus sequence typing (MLST), which provide a more robust and functionally relevant understanding of their pathogenic potential.

Transmission Routes in Poultry, with a Focus on Guinea Fowls

Avian pathogenic *Escherichia coli* (APEC) is a significant secondary pathogen that exploits breaches in host defense, with multiple transmission routes facilitating its spread in poultry, including guinea fowls (*Numida meleagris*). *Escherichia coli* is a diverse species inhabiting the avian intestinal tract, but only certain strains, collectively termed APEC, possess virulence genes that enable extra-intestinal disease. These virulence factors include adhesins (e.g., *fimC*, *papC*), iron acquisition systems (e.g., *iucD*, *fyuA*), protectins (e.g., *iss*), and toxins (e.g., *astA*), which distinguish them from commensal strains (Janßen et al., 2001; Borges et al., 2017; Feng et al., 2023). Pathogenicity is variable, with isolates classified into high, intermediate, low, or apathogenic categories based on lesion scoring and mortality (Souza et al., 2016).

The primary routes of APEC transmission in poultry are the respiratory route, vertical transmission, the faecal or oral route, and direct contact alongside environmental contamination. The respiratory route is the most common portal of entry, where environmental stressors such as high ammonia levels, dust, or concurrent viral infections damage the respiratory epithelium, allowing airborne APEC in dust and aerosols to colonize the trachea and lungs (Wang et al., 2022). This often leads to air sacculitis and systemic septicaemia. Guinea fowls, frequently reared in free-range or semi-intensive systems, are particularly exposed to such environmental stressors, facilitating this route. Vertical transmission occurs when infected breeder hens pass APEC to offspring through egg contamination, either trans-ovarially or via the shell membrane, causing omphalitis and high mortality in young birds (Khairullah et al., 2024). Regarding the faecal-oral route, the gastrointestinal tract acts as a reservoir, with birds ingesting APEC from contaminated feed, water, or litter; systemic infection can follow if the intestinal mucosa is compromised (Wang et al., 2020). Direct contact and environmental contamination are also significant, as dense stocking promotes bird-to-bird transmission, and APEC persists in litter, waterers, and feeders (Maron et al., 2013). Management practices in Southern African guinea fowl farming, which often mix free-ranging with confinement, influence environmental contamination dynamics (Gono et al., 2013).

In guinea fowls, clinical manifestations mirror those in other poultry, including respiratory colibacillosis with dyspnea and air sacculitis, septicemia with hepatosplenomegaly and pericarditis, and reproductive tract infections like salpingitis in laying birds (Dziva & Stevens, 2008). The prevalence of *E. coli* in guinea fowls is high, with studies reporting a 100% prevalence in gastrointestinal tracts in Ghana and significant infections in Nigeria, leading to production losses (Adzitey et al., 2019; Akanbi et al., 2022). A major concern is antimicrobial resistance (AMR), with multidrug resistance (MDR) observed in 56% of isolates in Nigerian poultry, including

guinea fowls showing 100% MDR in a limited sample, highlighting extensive antibiotic use and environmental contamination (Akanbi et al., 2022).

The transmission dynamics of *E. coli* in Southern Africa are complex, involving multiple routes that facilitate the spread of both intestinal and extraintestinal pathogenic strains among wildlife, livestock, food products, and humans. Wildlife, particularly wild birds, act as mobile reservoirs. In Egypt, wild birds near cattle farms carried *E. coli* strains genetically similar to those in cattle, indicating cross-species transmission via faecal contamination (Ibrahim et al., 2023). Similarly, in Brazil, free-ranging helmeted guinea fowl carried APEC with virulence genes associated with human ExPEC (Borzi et al., 2018). Food-producing animals and products are another major reservoir; in South Africa, whole-genome sequencing revealed a high prevalence of ExPEC pathotypes in poultry, pork, and processed meats, with AMR genes often on mobile genetic elements, facilitating horizontal transfer (Malesa et al., 2024). Environmental contamination through water and soil perpetuates transmission, with isolates from water sources sharing sequence types and virulence profiles with clinical human strains, suggesting dissemination via agricultural runoff or inadequate waste management (Malesa et al., 2024). The human-animal interface in farming communities, characterized by close contact and poor hygiene, enables zoonotic exchange, as seen in Egypt and South Africa where MDR *E. coli* from cattle and wild birds exhibited high resistance to commonly used veterinary antibiotics (Ibrahim et al., 2023). Finally, migratory birds can facilitate long-distance dispersal, acting as vectors across regions.

In summary, APEC transmission in guinea fowls and broader *E. coli* dissemination in Southern Africa are multifactorial, driven by specific poultry farming practices, wildlife reservoirs, contaminated food products, environmental pathways, and close human-animal interactions. These routes are compounded by the spread of AMR genes via mobile genetic elements, underscoring the urgent need for improved biosecurity, rational antibiotic use, and integrated One Health surveillance to mitigate infections and safeguard animal and public health.

Key Virulence Genes in Avian Pathogenic *E. coli* (APEC)

The pathogenicity of APEC is mediated by a sophisticated suite of virulence genes, typically encoded on plasmids and pathogenicity islands (PAIs). Their prevalence can vary significantly by host species and geographic location. These genes include Adhesion and colonisation genes (fimH, papC, tsh, iha), Iron Acquisition Systems (iutA, iuc, fyuA,sit) and Serum Resistance, Toxins, and Invasiveness Genes (iss, hlyE, ompT)

Adhesion and Colonization Genes

Initial attachment to host tissues is a critical first step. Key genes include fimH which encodes the tip adhesin of type 1 fimbriae, facilitating binding to mannose-containing receptors on epithelial cells (Kathayat et al, 2022). It is one of the most ubiquitous virulence genes found in APEC strains isolated globally. The papC is a key gene in the assembly of P fimbriae (pyelonephritis-associated pili), associated with binding to specific glycolipid receptors (Khatayat et al, 2021). While more classically associated with UPEC, its presence in APEC often correlates with higher septicaemic potential. The temperature-sensitive haemagglutinin gene (tsh) is an autotrans porter protein that acts as an adhesin/protease, frequently associated with APEC strains isolated from avian septicaemia and swollen head syndrome (Dozois et al, 2000). The iron-regulated gene homologue adhesion (iha) is a non-fimbrial adhesin that also contributes to siderophore function, enhancing both colonisation and iron acquisition.

Iron Acquisition Systems

Iron is a limiting nutrient within the host. APEC employs high-affinity iron acquisition systems crucial for survival and systemic spread through the iutA (Aerobactin receptor) which is a part of the Aerobactin siderophore system, and chelates environmental iron. The presence of iutA and its corresponding synthesis genes (iuc) is highly characteristic of virulent APEC strains and is often carried on the ColV plasmid (Foster-Nyorko et al, 2021). The fyuA (Yersiniabactin receptor) forms part of the Yersiniabactin system, frequently found on the high-pathogenicity island (HPI). It enables the bacterium to scavenge iron even in iron-restricted environments, crucial during septicaemia (Abdelhamid et al, 2024). Salmochelin iron transport (sit) gene system is often found

alongside Aerobactin and Yersiniabactin, further demonstrating the redundancy built into APEC's iron metabolism (Gao et al, 2024).

Serum Resistance, Toxins, and Invasiveness Genes

These factors mediate immune evasion, tissue damage, and systematic dissemination. Increased Serum Survival (iss) gene encodes a protein that confers resistance to complement-mediated killing in serum (Tivendale et al, 2004). It is considered one of the key diagnostic markers for virulent APEC and is strongly associated with the ColV plasmid. Its presence is vital for the transition from localized infection (airsacculitis) to septicaemia. Hemolysin E, or ClyA gene encodes a pore-forming toxin that causes cellular damage and lysis, potentially aiding bacterial spread and nutrient release (Oscarsson et al, 2002). While less potent than \alpha-hemolysin (hlyA), it contributes significantly to overall virulence. The Outer Membrane Protease T(ompT) encodes a protease that cleaves outer membrane proteins and potentially host defense peptides. This aids in immune evasion and tissue breakdown, facilitating invasion and spread within the host (Ilcebaylik and Turkyilmaz, 2020).

APEC Virulence Gene Profiles in Guinea Fowl: Southern African Context

Comparison with Conventional Poultry (Chickens, Ducks, Turkeys)

While extensive molecular studies exist for APEC in South African chickens (Dube, N., & Mbanga, J. 2018), data specific to guinea fowls are scarce. This scarcity presents a major knowledge gap. In conventional poultry worldwide and in regional studies in South Africa, APEC isolates typically exhibit high prevalence of the core virulence genes: fimH (nearly 100%), iutA, iss, and ompT (Ilcebaylik and Turkyilmaz, 2020). Strains causing severe systemic disease usually carry a combination of iron acquisition and serum resistance genes, often linked to the ColV plasmid (e.g., iss/iutA).

Guinea fowls are often raised under different management conditions (more extensive, less confined) than commercial chickens, their APEC isolates may exhibit specific differences which include higher environmental reservoir strain diversity, as free-ranging guinea fowl are exposed to a wider array of environmental *E. coli*, meaning APEC strains might be more heterogeneous in serotypes and phylogenetic groups compared to the highly clonal outbreak strains (O78/O2) found in intensively raised chickens (Foster-Nyorko et al, 2021). Furthermore, due to high environmental load and potentially more exposure to secondary respiratory infections, adhesion factors like type 1 fimbriae (fimH) are expected to be universally present (Van de Bogaard et al, 2000). Finally, host-specific adaptation may lead to the potential for novel virulence factors, where the discovery of unique or highly prevalent VFs in guinea fowl APEC that are optimized for the guinea fowl immune system or respiratory tract structure is anticipated, warranting in-depth WGS analysis.

Preliminary surveillance data, where available (localized veterinary reports in Southern Africa), suggest that guinea fowls often suffer from acute septicaemic colibacillosis in young keets, implying the involvement of highly pathogenic strains possessing the critical combination of iss and iutA, similar to patterns observed in turkey and chicken septicaemia (Barbieri et al, 2017).

Transmission Dynamics Specific to Guinea Fowl Flocks

The dual nature of guinea fowl farming (free-range and confined) in Southern Africa complicates transmission modelling. Free-ranging allows contact with wild birds and environmental sources, potentially increasing the introduction of novel APEC strains and resistance genes. Conversely, the frequent co-housing of guinea fowls with backyard chickens or ducks means that the exchange of APEC strains between host species is highly probable, making host-specific adaptations difficult to isolate without dedicated comparative genomics.

Modern Molecular Techniques for Profiling and Tracking

Traditional methods like PCR for detecting individual virulence genes and MLST for tracking clones are standard. However, the complexity of APEC virulence requires high-resolution techniques.

Whole-Genome Sequencing (WGS)

APEC isolates, with applications that encompass high-resolution virulence profiling by enabling the detection of the full complement of virulence genes, including those located on mobile genetic elements (MGEs) like

plasmids and pathogenicity islands, which is critical for assessing true pathogenic potential beyond the standard five-gene PCR panel (Barbieri et al, 2017). Furthermore, WGS facilitates detailed epizootic tracing and MLST analysis, allowing for the identification of specific sequence types (STs), such as the globally recognised highrisk, multi-host ExPEC lineages ST95, ST405, and ST131 (Mellata, 2013), whose tracking in Southern African guinea fowls is vital for epidemiological risk assessment. Finally, the technique enables comparative genomics, which permits the direct comparison of guinea fowl isolates with those from chickens and humans in the region to reveal shared genetic backbones and MGEs, thereby providing irrefutable evidence of inter-species transmission and the zoonotic bridge (Foster-Nyorko et al, 2020)

Phylogenetic Analysis

By placing guinea fowl isolates within the global *E. coli* phylogeny, researchers can better understand the evolution of virulence. If guinea fowl APEC strains consistently group within ExPEC phylogenetic groups B2 and D, it confirms their high intrinsic risk and similarities to human pathogens, necessitating robust surveillance efforts (Foster-Nyorko et al, 2021).

The Convergence of Virulence and Multidrug Resistance (MDR)

A critical public health threat in Southern Africa is the frequent convergence of high virulence potential and multidrug resistance (MDR) in APEC strains.

APEC as a Reservoir for AMR

In many poultry production settings, the use of antibiotics for prophylaxis or treatment drives selection pressure. APEC isolates in Southern Africa commonly exhibit resistance to multiple classes of antibiotics, including tetracyclines, sulfonamides, and critically important antimicrobials like third-generation cephalosporins (via bla_{CTX-M} genes) and colistin (via mcr-1 genes) (Dube, N., & Mbanga, J. 2018).

The genes encoding these resistance mechanisms are often physically linked to virulence genes (iss, iutA) on large conjugative plasmids, Col V plasmids. This genetic linkage ensures that selection pressure targeting resistance inadvertently maintains and propagates virulence factors within the bacterial population, creating highly virulent and untreatable superbugs.

Zoonotic Threat

APEC strains carrying both extensive VFs and MDR mechanisms act as a reservoir for extra-intestinal pathogenic *E. coli* (ExPEC) infections in humans. In Southern Africa, where guinea fowls often serve as a direct food source and share environmental space with human habitation, the risk of zoonotic transfer is elevated (Van Boeckel et al, 2015). The similarity of APEC VFs to those of UPEC and neonatal meningitis *E. coli* (NMEC) means that virulent guinea fowl strains represent a significant public health risk that warrants targeted surveillance and intervention strategies coordinated between animal and human health sectors (One Health approach).

Current Control Strategies and Their Limitations

Current strategies for controlling APEC in guinea fowls generally mirror those employed in chicken production, with varying degrees of success. These include Biosecurity which implements stringent biosecurity measures such as proper sanitation, restricted access, all-in/all-out systems, and effective waste management can reduce pathogen exposure. However, biosecurity can be challenging to implement fully and consistently, especially in extensive and semi-intensive guinea fowl farming systems prevalent in Southern Africa (Liu et al, 2021).

Antimicrobial Treatment

Antibiotics are often used for therapeutic purposes, and sometimes prophylactically or as growth promoters (though the latter is increasingly restricted). However, this practice is a primary driver of AMR, leading to treatment failures, increased costs, and the selection of resistant strains. The limited range of effective and approved antibiotics for guinea fowls further complicates this approach.

Vaccination

While vaccines exist for APEC in chickens, guinea fowl-specific vaccines are largely unavailable. Cross-species vaccines may offer some protection but might not be optimally effective due to host-specific immune responses or differences in dominant circulating APEC serotypes/clones. Autogenous vaccines (herd-specific) can be an option but are costly and require robust diagnostic capabilities (Alonso et al, 2017). Vaccination presents a proactive solution. These include autogenous Vaccines: Prepared from farm-specific isolates, offering targeted protection. Commercial and Novel Vaccines: Inactivated vaccines based on common sero-groups exist, but their efficacy is variable. Research into subunit vaccines targeting conserved virulence factors (FimH, IutA) is needed for broader, guinea fowl-specific protection (Tong et al, 2015).

Biosecurity and Management

Effective disease management relies on robust biosecurity as the first line of defense, including all-in-all-out systems, strict sanitation, and rodent control. According to Motola et al., (2023) Complementary management practices, such as reducing stress factors through optimal stocking densities, adequate ventilation, proper nutrition, and effective disease monitoring, further enhance host immunity and reduce susceptibility to infection. However, the limitations of these general strategies, particularly within the context of the guinea fowl industry, underscore the urgent need for targeted, evidence-based interventions that are specifically tailored to this host and its regional operating environment (Soara et al., 2020)

Alternative Therapies

In response to the push to reduce antibiotics, various alternatives are being explored, including probiotics and prebiotics to promote a healthy gut microbiome and competitively exclude APEC, phage therapy, which employs specific, self-replicating bacteriophages as biocontrol agents, and essential oils and phytogenics plant-derived compounds with antimicrobial and immune-stimulatory properties (Van Boeckel et al, 2015).

Future Research Directions and Conclusion

To effectively address the challenge of APEC in guinea fowls and mitigate its significant One Health implications, targeted and innovative research is imperative. Building upon the foundational understanding and addressing the critical knowledge gaps, the following research avenues are proposed:

Need for More Genomic Studies on Guinea Fowls

Currently, our understanding of APEC populations in guinea fowls, especially in Southern Africa, is fragmented. Large-scale, systematic Whole Genome Sequencing (WGS) studies on APEC isolates from guinea fowls across different Southern African countries are crucial. WGS offers unparalleled resolution compared to traditional PCR-based methods, enabling:

Defining Population Structure: WGS will precisely delineate the genetic relationships among APEC isolates, identifying distinct clades, lineages, and population shifts over time. This will reveal the dominant and emerging virulent-resistant clones circulating within guinea fowl populations, providing critical insights for surveillance and intervention strategies (Foster-Nyorko et al, 2021).

Characterizing Virulence and Resistance Gene Repertoires: WGS allows for the comprehensive identification of all virulence factor genes, their genomic context, and their association with specific genomic backgrounds. Simultaneously, it provides a complete picture of the antimicrobial resistance gene (ARG) resistome, including novel ARGs and their mobile genetic element associations (e.g., plasmids, transposons), which are critical for understanding horizontal gene transfer (Tcheou et al, 2025).

Assessing Zoonotic Potential: By comparing the WGS data of guinea fowl APEC isolates with human clinical *E. coli* isolates (both pathogenic and non-pathogenic) from the same geographical regions, researchers can precisely assess the zoonotic potential. This comparative genomics approach can identify shared virulentresistant clones, track their transmission routes, and pinpoint specific genetic determinants involved in crossspecies jumps, thus informing public health risk assessments (Standley et al, 2019).

Understanding Evolution and Transmission: WGS data can be used to construct phylogenetic trees, infer evolutionary relationships, and track the transmission dynamics of APEC strains within and between farms, across different hosts, and into the environment (Mageiros et al.,2021).

Development of Targeted Antimicrobial Treatments

The escalating AMR crisis necessitates a paradigm shift away from broad-spectrum antibiotics towards sustainable, targeted therapies. Research should focus on developing narrow-spectrum therapeutics that disarm bacteria without exerting broad selective pressure for AMR.

Phage Cocktails: Bacteriophages are viruses that specifically infect and lyse bacteria. Research into developing multi-phage cocktails, tailored to prevalent guinea fowl APEC strains, offers a promising narrow-spectrum approach. Phages are highly specific, self-replicating at the site of infection, and their lytic activity offers a rapid means of bacterial clearance (Mosimaun et al, 2021). Challenges include regulatory hurdles, delivery methods, and ensuring stability and efficacy against a dynamic bacterial population.

Anti-virulence Compounds: These compounds aim to neutralize bacterial virulence factors rather than killing the bacteria directly. Examples include siderophore inhibitors, which block bacterial iron acquisition systems (e.g., targeting iutA, fyuA) (Doi et al 2017), or inhibitors of quorum sensing, which disrupt bacterial communication and biofilm formation. By disarming the bacteria without eradicating them, these compounds may reduce selective pressure for resistance development, allowing the host immune system to clear the attenuated infection.

Immunomodulatory: Investigating compounds that enhance the guinea fowl's innate immune response could offer another avenue, making the host more resilient to APEC infection.

Understanding Environmental Factors Influencing APEC Virulence

The local environment plays a crucial role in the persistence, transmission, and pathogenicity of APEC. Studies are needed to elucidate how specific local environmental conditions in Southern Africa influence the expression and transmission of APEC virulence genes in guinea fowl populations.

Climate: Factors such as temperature, humidity, rainfall, and seasonal variations can impact bacterial survival in the environment, host susceptibility (e.g., heat stress), and disease incidence.

Farming Practices: Varying farming practices (e.g., intensive vs. extensive, hygiene standards, feed formulations, water sources, waste management) directly influence pathogen exposure and transmission risk.

Co-infections and Microbiome: The presence of other pathogens or the composition of the host's normal microbiota can alter the host's immune response and create ecological niches that either inhibit or promote APEC colonization and virulence (Maron et al, 2013).

Environmental Reservoirs: Research into identifying and characterising environmental reservoirs of APEC (e.g., water, soil, wild birds, insects) and their role in the epidemiology of guinea fowl colibacillosis is essential for developing effective biosecurity measures. This understanding could inform targeted interventions to break transmission cycles and reduce environmental contamination (Kathayat et al, 2021).

Host-Pathogen Interactions

A deeper understanding of the guinea fowl's specific immune response to APEC infection is crucial for developing host-centric control measures. The Immune Response Characterization involves investigating the cellular and humoral immune responses of guinea fowls to APEC infection (e.g., cytokine profiles, antibody production, specific immune cell activation) could reveal host-specific resistance mechanisms. This includes identifying particular genes or pathways associated with resistance or susceptibility (Standley et al ,2019).

Another Host-pathogen interaction is the Genetic Basis of Resistance, which involves research into the genetic architecture of resistance to APEC in guinea fowls could inform breeding programs for disease resilience. This

might involve identifying genomic regions (quantitative trait loci, QTLs) or specific genetic markers associated with enhanced resistance, enabling selective breeding for more robust guinea fowl lines.

Vaccine Development: Understanding host-pathogen interactions is fundamental for designing effective vaccines. This includes identifying conserved protective antigens that elicit a strong and durable immune response in guinea fowls, potentially leading to the development of host-specific subunit or inactivated vaccines.

CONCLUSION

In conclusion, APEC poses a significant and underappreciated threat to the sustainable growth of the guinea fowl industry in Southern Africa. The convergence of virulence and multidrug resistance in these strains represents a serious One Health concern. While current knowledge is fragmented, the path forward is clear: a concerted research effort leveraging modern genomics, integrated with holistic One Health control strategies, is essential to safeguard animal health, ensure food security, and mitigate the global public health threat of antimicrobial resistance.

AI Assistance Disclosure

This review was created with the assistance of generative AI tools (Deepseek ai). The AI was used for tasks such as editing and data synthesis. The authors reviewed, edited, and validated all output, and assumes full responsibility for the accuracy, integrity, and implications of the final content

REFERENCES

1. Abdallah, N., & Oluwaseun, O. A. (2025). Socio-economic and production dynamics of Guinea fowl farming in Northern Ghana: Insights into health management, challenges, and climate change impacts. Tropical Animal Health and Production, 57(181). https://doi.org/10.1007/s11250-025-04427-2
2. Abdallah, N., & Oyebamiji, O. A. (2024). Guinea fowl production in Africa: Economic importance and constraints. Egyptian Veterinary Science, 18 (1), 1–5.
3. Abdelhamid, M. K., Hess, C., Bilic, I., Reicher, M., Tarbiat, B., Razzazi-Fazeli, E., Schmalwieser, A. W., Hess, M., & Selberherr, E. (2024). A comprehensive study of colisepticaemia progression in layer chickens applying novel tools elucidates pathogenesis and transmission of *Escherichia coli* into eggs. Scientific Reports, 14, 8111. https://doi.org/10.1038/s41598-024-58794-1
4. Adzitey, F., Agbolosu, A. A., & Udoka, U. J. (2019). Antibacterial Effect of Aloe Vera Gel Extract on *Escherichia coli* and *Salmonella enterica* Isolated from the Gastrointestinal Tract of Guinea Fowls. World's Veterinary Journal, 9(3), 166–173.
5. Akanbi, O. B., Olorunshola, I. D., Osilojo, P., Ademola, E., Agada, G. O. A., Aiyedun, J. O., Odita, C. I., & Ola-Fadunsin, S. D. (2022). *Escherichia coli* Infections, and Antimicrobial Resistance In Poultry Flocks, in North Central Nigeria. MKH, 33(3), 188–207
6. Alonso, C. A., Zarazaga, M., Ben Sallem, R., Jouini, A., Ben Slama, K., & Torres, C. (2017). Antibiotic resistance in *Escherichia coli* in husbandry animals: The African perspective. Letters in Applied Microbiology, 64(4), 318–334. https://doi.org/10.1111/lam.12724
7. Barbieri, N. L., Vande Vorde, J. A., Baker, A. R., Horn, F., Li, G., Logue, C. M., & Ilcebaylik and Turkyilmaz, 2020, L. K. (2017). FNR regulates the expression of important virulence factors contributing to the pathogenicity of avian pathogenic *Escherichia coli*. Frontiers in Cellular and Infection Microbiology, 7, 265. https://doi.org/10.3389/fcimb.2017.00265
8. Borges, C. A., Beraldo, L. G., Maluta, R. P., Cardozo, M. V., Barboza, K. B., Guastalli, E. A. L., & Ávila, F. A. (2017). Multidrug-resistant pathogenic *Escherichia coli* isolated from wild birds in a veterinary hospital. Avian Pathology, 46(1), 76-83.
9. Borzi, M. M., Cardozo, M. V., de Oliveira, E. S., Pollo, A. S., Guastalli, E. A. L., dos Santos, L. F., & de Ávila, F. A. (2018). Characterization of avian pathogenic *Escherichia coli* isolated from free-range helmeted guinea fowl. Brazilian Journal of Microbiology, 49(Suppl 1), 107–112. https://doi.org/10.1016/j.bjm.2018.04.011
10. Doi, Y., Iovleva, A., & Bonomo, R. A. (2017). The ecology of extended-spectrum beta-lactamases (ESBLs) in the developed world. Journal of Travel Medicine,24S44–S51. https://doi.org/10.1093/jtm/taw102

11. Dozois, C. M., Dho-Moulin, M., Brée, A., Fairbrother, J. M., Desautels, C., & Curtiss, R. III. (2000). Relationship between the Tsh autotransporter and pathogenicity of avian *Escherichia coli* and localization and analysis of the Tsh genetic region. Infection and Immunity, 68(7), 4145–4154. https://doi.org/10.1128/IAI.68.7.4145-4154.2000
12. Dube, N., & Mbanga, J. (2018). Molecular characterization and antibiotic resistance patterns of avian fecal *Escherichia coli* from turkeys, geese, and ducks. Veterinary World, 11(6), 859.
13. Dziva, F., & Stevens, M. P. (2008). Colibacillosis in poultry: Unravelling the molecular basis of virulence of avian pathogenic *Escherichia coli* in their natural hosts. Avian Pathology, 37(4), 355–366.
14. Feng, A., Akter, S., and Leigh, S. A. (2023). Genomic diversity, pathogenicity and antimicrobial resistance of *Escherichia coli* isolated from poultry in the southern United States. BMC Microbiology, 23, 15.
15. Foster-Nyarko E., Alikhan N., Ravi N., Thomson N.M., Jarju S., Kwambana- Adams B.A., Secka A., O'Grady J., Antonio M., & Pallen M.J. (2021). Genomic diversity of *Escherichia coli* isolates from backyard chickens and guinea fowl in the Gambia. Microbial Genomics,7:00048. https//doi.org/10.1099/mgen.0.000484
16. Gao, Q., Wang, X., Xu, H., Xu, Y., Ling, J., Zhang, D., Gao, S., & Liu, X. (2012). Roles of iron acquisition systems in virulence of extraintestinal pathogenic *Escherichia coli*: Salmochelin and aerobactin contribute more to virulence than heme in a chicken infection model. BMC Microbiology, 12(1), 143. https://doi.org/10.1186/1471-2180-12-143
17. Gil, J. D., Reidsma, P., Giller, K., Todman, L., Whitmore, A., & van Ittersum, M. (2019). Sustainable development goal 2: Improved targets and indicators for agriculture and food security. Ambio, 48(7), 685– 698. https://doi.org/10.1007/s13280-018-1101-4
18. Gono, R. K., Svinurai, W., & Muzvondiwa, J. V. (2013). Constraints and opportunities to Guinea fowl production in Zimbabwe: A case study of the Midlands Province, Zimbabwe. International Journal of Science and Research, 2(3), 236–239.
19. Ibrahim, G. A., Salah-Eldein, A. M., Al-Zaban, M. I., El-Oksh, A. S. A., Ahmed, E. M., Farid, D. S., & Saad, E. M. (2023). Monitoring the genetic variation of some *Escherichia coli* strains in wild birds and cattle. Open Journal of Veterinary Research, 10(1), 1–10.
20. Ilcebaylik, A. and Turkyilmaz, S. (2020). Investigation of Important Virulence Genes and Antibiotic Resistance of *Escherichia coli* Isolated from Broiler Chickens. Israel Journal of Veterinary Medicine 75 (4), 204-214.
21. Janßen, T., Schwarz, C., Preikschat, P., et al. (2001). Virulence-associated genes in avian pathogenic *Escherichia coli* (APEC) isolated from internal organs of poultry having died from colibacillosis. International Journal of Medical Microbiology, 291, 371–378.
22. Jordan, T. J., & Kariyawasam, S. A. (2010). Sequence analysis and characterization of a transferable hybrid plasmid encoding multidrug resistance and enabling zoonotic potential for extra intestinal *Escherichia coli*. Infection and Immunity, 78(5), 1931–1942. https://doi.org/10.1128/IAI.01259-09
23. Kaper, J.B., Nataro, J.P., and Mobley, H.L.T. (2004). Pathogenic *Escherichia coli.* Nat Rev Microbiol 2:123–40.
24. Karikari, B.R.F, Adomako, A.R., Prince, A., Armstrong, D. and Alhassan, H.J. (2024). The state of Guinea fowl production and challenges faced by the industry in Ghana. International Journal of Current Research, 16, (03), 27535-27542.
25. Kathayat, D., Lokesh, D., Ranjit, S., & Rajashekara, G. (2021). Avian Pathogenic *Escherichia coli* (APEC): An overview of virulence and pathogenesis factors, zoonotic potential, and control strategies. Pathogens, 10(4), 467. https://doi.org/10.3390/pathogens10040467
26. Khairullah, A. R., Afnani, D. A., Riwu, K. H. P., Widodo, A., Yanestria, S. M., Moses, I. B., & Raissa, R. (2024). Avian pathogenic *Escherichia coli*: Epidemiology, virulence and pathogenesis, diagnosis, pathophysiology, transmission, vaccination, and control. Veterinary World, 17(12), 2747.
27. Kusina, N. T., Saina, H., Kusina, J. F., & Lebel, S. (2012). An insight into guinea fowl rearing practices and productivity by guinea fowl keepers in Zimbabwe. African Journal of Agricultural Research, 7(25), 3621–3625.
28. Landman, W. J. M., Buter, G.J., Dijkman, R., & van Eck, J. H. H. (2014). Molecular typing of avian pathogenic *Escherichia coli* colonies originating from outbreaks of *E. coli* peritonitis syndrome in chicken flocks. Avian Pathology, 43(4), 345-356. https://doi.org/10.1080/03079457.2014.935291

29. Lengthang, B., Tellah, M., Nideou, D., Assadi, M., Odjigue, N., & Logtene, Y. M. (2023). Guinea fowl farming and its egg production in the Western Tandjile Department, Chad. International Journal of Poultry Science, 22(1), 24–30.
30. Liu, Z., Wang, K., Zhang, Y., Xia, L., Zhao, L., Guo, C., Liu, X., Qin, L., & Hao, Z. (2021). High prevalence and diversity characteristics of blaNDM, mcr, and blaESBLs harboring multidrug-resistant *Escherichia coli* from chicken, pig, and cattle in China. Frontiers in Cellular and Infection Microbiology, 11, 755545. https://doi.org/10.3389/fcimb.2021.755545
31. Mageiros, L., Méric, G., Bayliss, S. C., Pensar, J., Pascoe, B., Mourkas, E., & Sheppard, S. K. (2021). Genome evolution and the emergence of pathogenicity in avian *Escherichia coli*. Nature communications, 12(1), 765.
32. Malesa, R., Pierneef, R., Magwedere, K., Mafuna, T., & Matle, I. (2024). Genomic characterisation of generic *Escherichia coli* from food-producing animals and products of animal origin in South Africa. Frontiers in Bacteriology, 3, 1432292. https://doi.org/10.3389/fbrio.2024.1432292
33. Maron, D. F., Smith, T. J. S., & Nachman, K. E. (2013). Restrictions on antimicrobial use in food animal production: An international regulatory and economic survey. Globalization and Health, 9(1), 48. https://doi.org/10.1186/1744-8603-9-48
34. Mellata M. (2013). Human and avian extraintestinal pathogenic *Escherichia coli*: infections, zoonotic risks, and antibiotic resistance trends. Foodborne Pathogenic Diseases 10 (1),916–932.
35. Mosimann, S., Desiree, K., & Ebner, P. (2021). Efficacy of phage therapy in poultry: A systematic review and meta-analysis. Poultry Science, 100(12), 101477. https://doi.org/10.1016/j.psj.2021.101477
36. Motola, G., Hafez, H. M., & Brüggemann-Schwarze, S. (2023). Assessment of three alternative methods for bacterial disinfection of hatching eggs in comparison with conventional approach in commercial broiler hatcheries. PloS one, 18(3), e0283699.
37. Oscarsson, J., Westermark, M., Löfdahl, S., Olsen, B., Palmgren, H., Mizunoe, Y., Wai, S. N., & Uhlin, B. E. (2002). Characterization of a pore-forming cytotoxin expressed by *Salmonella enterica* serovars *typhi* and *paratyphi A*.Infection and Immunity, 70(10), 5759–5769. https://doi.org/10.1128/IAI.70.10.5759-5769.2002
38. Osman, K. M., Kappell, A. D., Elhadidy, M., ElMougy, F., El-Ghany, W. A. A., Orabi, A., Mubarak, A. S., Dawoud, T. M., Hemeg, H. A., Moussa, I. M. I., & Hessain, A. M. (2018). Poultry hatcheries as potential reservoirs for antimicrobial-resistant *Escherichia coli*: A risk to public health and food safety. Scientific Reports, 8, 5859. https://doi.org/10.1038/s41598-018-23962-7
39. Schouler, C., Schaeffer, B., Bree, A., Mora, A., Dahbi, G., Biet, F., Oswald, E., & Moulin-Schouleur, M. (2012). Diagnostic strategy for identifying avian pathogenic *Escherichia coli* based on four patterns of virulence genes. Journal of Clinical Microbiology, 50(5), 1673–1678. https://doi.org/10.1128/JCM.0505711
40. Soara, A. E., Talaki, E., & Tona, K. (2020). Characteristics of indigenous guinea fowl (*Numida meleagris*) family poultry production in northern Togo. Tropical Animal Health and Production, 52(6), 3755-3767.
41. Standley, C. J., Carlin, E. P., Sorrell, E. M., Barry, A. M., Bile, E., Diakite, A. S., Keita, M. S., Koivogui, L., Mane, S., Martel, L. D., & Katz, R. (2019). Assessing health systems in Guinea for prevention and control of priority zoonotic diseases: A One Health approach. One Health, 7, 100101. https://doi.org/10.1016/j.onehlt.2019.100101
42. Suzuki, Y., Sato, T., Fukushima, Y., Nakajima, C., Suzuki, Y., Takahashi, S., & Yokota, S. I. (2020). Contribution of beta-lactamase and efflux pump overproduction to tazobactam-piperacillin resistance in clinical isolates of *Escherichia coli*. International Journal of Antimicrobial Agents, 55(3), 105919. https://doi.org/10.1016/j.ijantimicag.2020.105919
43. Tcheou, P., Bedekelabou, A. P., Adjei-Mensah, B., Kpomasse, C. C., Talaki, E., & Salou, M. (2025). Antibiotic use and residue detection in guinea fowl eggs in rural Togo: An assessment of practices and risks. Veterinary and Animal Science, 28, 100110.
44. Tivendale, K. A., Allen, J. L., Ginns, C. A., Crabb, B. S., & Browning, G. F. (2004). Association of iss and iucA, but not tsh, with plasmid-mediated virulence of avian pathogenic *Escherichia coli*. Infection and Immunity, 72(11), 6554–6560. https://doi.org/10.1128/IAI.72.11.6554-6560.2004
45. Tong, P., Sun, Y., Ji, X., Du, X., Guo, X., Liu, J., Zhu, L., Zhou, B., Zhou, W., Liu, G., & Li, T. (2015). Characterization of antimicrobial resistance and extended-spectrum beta-lactamase genes in *Escherichia coli* isolated from chickens. Foodborne Pathogens and Disease, 12(4), 345–352. https://doi.org/10.1089/fpd.2014.1874

46. Van Boeckel, T. P., Brower, C., Gilbert, M., Grenfell, B. T., Levin, S. A., Robinson, T. P., Teillant, A., & Laxminarayan, R. (2015). Global trends in antimicrobial use in food animals. Proceedings of the National Academy of Sciences, 112(18), 5649–5654. https://doi.org/10.1073/pnas.1503141112
47. Van den Bogaard, A. E., & Stobberingh, E. E. (2000). Epidemiology of resistance to antibiotics: Links between animals and humans. International Journal of Antimicrobial Agents, 14(4), 327–335. https://doi.org/10.1016/s0924-8579(00)00145-x
48. Wang, Y., Zhou, J., Li, X., Ma, L., Cao, X., Hu, W., Zhao, L., Jing, W., Lan, X., & Li, Y. (2020). Genetic diversity, antimicrobial resistance and extended-spectrum beta-lactamase type of *Escherichia coli* isolates from chicken, dog, pig and yak in Gansu and Qinghai Provinces, China. Journal of Global Antimicrobial Resistance, 22, 726–732. https://doi.org/10.1016/j.jgar.2020.07.005
49. Wang, Z.; Lu, Q.; Mao, X.; Li, L.; Dou, J.; He, Q.; Shao, H.; Luo, Q. Prevalence of Extended-Spectrum β-Lactamase-Resistant Genes in *Escherichia coli* Isolates from Central China during 2016–2019. Animals 2022, 12, 3191. https://doi.org/ 10.3390/ani1222319
50. Wu, C., Wang, Y., Shi, X., Wang, S., Ren, H., Shen, Z., Wang, Y., Lin, J., & Wang, S. (2018). Rapid rise of the ESBL and mcr-1 genes in *Escherichia coli* of chicken origin in China, 2008–2014. Emerging Microbes & Infections, 7(1), 30. https://doi.org/10.1038/s41426-018-0033-1