

Fecal Carriage of Resistant *Escherichia coli* in Livestock in Algeria: Emergence of NDM and OXA-181.

Michael Boltz*

Department of Poultry Science, Mississippi State University, US

*Correspondence to: Michael Boltz, Department of Poultry Science, Mississippi State University, US. E-mail: boltzm@msstate.edu

Received: 03-Feb-2025, Manuscript No. AABID-25-169065; Editor assigned: 05-Feb-2025, Pre QC No. AABID-25-169065 (PQ); Reviewed: 11-Feb-2025, QC No. AABID-25-169065; Revised: 25-Feb-2025, Manuscript No. AABID-25-169065 (R); Published: 28-Feb-2025, DOI: 10.35841/aabid-9.1.189

Introduction

The global rise of antimicrobial resistance (AMR) poses a significant threat to public health, and zoonotic reservoirs particularly livestock play a critical role in the dissemination of resistant bacteria [1, 2].

In Algeria, recent research has revealed alarming levels of multidrug-resistant *Escherichia coli* (*E. coli*) in cattle and sheep, including strains carrying carbapenemase genes such as NDM-1 and OXA-181, which are typically associated with last-resort antibiotics in human medicine [3,4].

A study published in *BMC Microbiology* investigated the fecal carriage of resistant *E. coli* in livestock across 28 farms in Guelma, northeastern Algeria. This discovery underscores the urgent need for integrated surveillance and stewardship strategies under the One Health framework [5].

Researchers collected 285 fecal samples—145 from cattle and 140 from sheep—and screened them for third-generation cephalosporin (3GC)-resistant *E. coli*. Antibiotic susceptibility testing, phenotypic assays, and PCR-based molecular diagnostics were used to identify resistance genes and integrons [6,7].

The findings were striking: 27 cefotaxime-resistant *E. coli* isolates were recovered, with a prevalence of 17% in cattle and 1% in sheep. These isolates exhibited high levels of multidrug resistance (MDR), with 85% resistant to β -lactams, tetracyclines, fluoroquinolones, and trimethoprim-sulfamethoxazole [8].

Additionally, class 1 integrons were detected in 10 isolates, suggesting horizontal gene transfer and the potential for rapid dissemination of resistance traits. The detection of NDM-1 and OXA-181 in livestock is particularly concerning. These carbapenemase genes confer resistance to carbapenems—antibiotics considered the last line of defense against multidrug-resistant Gram-negative infections. Their presence in animals, despite carbapenems not being approved for veterinary use in Algeria, suggests environmental contamination or indirect transmission from human sources [9, 10].

Conclusion

This raises the possibility of reverse zoonosis, where resistant bacteria or resistance genes are transmitted from humans to animals, potentially through agricultural runoff, contaminated feed, or shared water sources. Once established in livestock, these bacteria can re-enter the human population via food, direct contact, or environmental exposure. The emergence of carbapenem-resistant *E. coli* in Algerian livestock mirrors trends observed in other regions. A study in Turkey reported similar findings, with *E. coli* isolates from

cattle carrying OXA-type carbapenemases and exhibiting resistance to multiple antibiotic classes. In sub-Saharan Africa, the prevalence of ESBL-producing *E. coli* in both humans and animals is rising, driven by antibiotic overuse and poor regulatory oversight.

References

1. Imran M, Mahmood S. An overview of human prion diseases. *Virology*. 2011;8:559.
2. Spiropoulos J, Lockey R, Sallis RE, et al. Isolation of prion with BSE properties from farmed goat. *Emerg Infect Dis*. 2011;17: 2253-61.
3. Kaski D, Mead S, Hyare H, et al. Variant CJD in an individual heterozygous for PRNP codon 129. *Lancet*. 2009;374:2128.
4. Konold T, Bone GE, Clifford D, et al. Experimental H-type and L-type bovine spongiform encephalopathy in cattle: Observation of two clinical syndromes and diagnostic challenges. *BMC Vet Res*. 2012;8:22.
5. Konold T, Sivan SK, Ryan J, et al. Analysis of clinical signs associated with bovine spongiform encephalopathy in casualty slaughter cattle. *Vet J*. 2006;171: 438-44.
6. Kovacs GG, Budka H. Prion diseases: From protein to cell pathology. *Am J Pathol*; 2008;172(3):555-65.
7. Lasmezas CI, Deslys JP, Robain O, et al. Transmission of the BSE agent to mice in the absence of detectable abnormal prion protein. *Science*. 1997;275:402-5.
8. Lewis PA, Tattum MH, Jones S, et al. Codon 129 polymorphism of the human prion protein influences the kinetics of amyloid formation. *J Gen Virol*. 2006;87:2443-9.
9. Mackay GA, Knight RSG, Ironside JW. The molecular epidemiology of variant CJD. *Int J Mol Epidemiol Genet*. 2011;2(3):217-27.
10. Masujin K, Mathews D, Wells GAH, et al. Prions in the peripheral nerves of bovine spongiform encephalopathy-affected cattle. *J Gen Virol*. 2007;88:1850-8.