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Mini Review

Colistin Resistance in Algeria's Poultry Farms: A Mini Review

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ABSTRACT

Colistin resistance has become an important veterinary and One Health concern because mobile colistin resistance genes have emerged and spread in food-producing animals. The present study aimed to synthesize available evidence on colistin resistance in Algerian poultry, poultry-associated environments, and chicken meat from 2015 to July 2026. PubMed was used as the primary bibliographic database for publications from 1 January 2015 to 15 July 2026. The reference lists of eligible studies were checked, and relevant publisher and journal websites were examined. Eleven studies met the final eligibility criteria. The available evidence indicated that colistin resistance and mobile colistin resistance genes, particularly *mcr-1*, have been documented in diseased poultry, apparently healthy poultry, poultry-farm environments, and retail chicken meat in Algeria. The *mcr-5* gene has been reported in apparently healthy poultry, while some studies have documented phenotypic colistin resistance without confirming *mcr-1*. In avian pathogenic *Escherichia coli*, colistin resistance and *mcr-1* were reported alongside multidrug-resistance and virulence-associated markers. However, substantial heterogeneity in the sampled populations, study designs, geographical coverage, and susceptibility-testing methods limited the ability of the studies from Algeria to estimate the national prevalence of colistin resistance or *mcr* carriage, or to infer the direction of transmission among poultry, food, and environmental compartments.

1. Introduction

Colistin acts on susceptible Gram-negative bacteria by binding lipopolysaccharide and disrupting membrane integrity[1,2]. The therapeutic importance of colistin in the treatment of multidrug-resistant Gram-negative infections has made the emergence and spread of colistin resistance a persistent concern[3]. The identification of the plasmid-borne mobile colistin resistance gene, *mcr-1*, established a transferable mechanism for distributing colistin resistance between bacterial hosts[1,4].

In Algeria, colistin has been reported to be used in poultry production as a metaphylactic treatment for Gram-negative gastrointestinal infections and as a feed additive to promote animal growth[5]. Colistin resistance mediated by *mcr-1* has also been documented in *Escherichia coli* (*E. coli*) recovered from poultry in Algeria[6]. A survey of veterinary practitioners in Northeastern Algeria found widespread antimicrobial use on poultry farms and frequent self-medication practices in the sector[7]. These reports provided relevant local context for understanding colistin use, broader antimicrobial-use practices, and resistance in the Algerian poultry sector[5-7].

In poultry, orally administered colistin is poorly absorbed from the gastrointestinal tract and therefore results in substantial intestinal exposure[8]. Oral colistin treatment may create selective pressure in the intestinal tract, increasing the relative abundance of less susceptible *E. coli* populations[8]. Colistin-resistant *E. coli* have been reported in diseased and apparently healthy poultry in Algeria[9,10]. The *mcr-1*-positive *E. coli* have been recovered from poultry-farm environmental samples, including drinking water, wastewater, wall swabs, and soil[11]. Available Algerian studies differed substantially in populations, sample types, and production settings, including disease-associated avian pathogenic *Escherichia coli* (APEC) isolates[9], poultry fecal samples[6,12], retail chicken meat[5], apparently healthy poultry[10], and poultry-farm environmental samples[11]. Despite the growing evidence, important knowledge gaps remain regarding the national distribution, epidemiology, and transmission of colistin resistance and *mcr* genes in the Algerian poultry sector. The available Algerian studies are geographically fragmented and differ in poultry populations, bacterial species, sampling units, and colistin susceptibility-testing methods. These differences limit direct comparisons and leave the national distribution of colistin resistance and *mcr* genes in the Algerian poultry sector poorly characterized. The present study aimed to synthesize available evidence on phenotypic colistin resistance and *mcr* genes in Algerian poultry, poultry-

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associated environments, and chicken meat, and to assess current epidemiological and molecular evidence on colistin resistance in the Algerian poultry sector.

2. Search criteria

A focused narrative search was conducted for peer-reviewed reports on colistin resistance in the Algerian poultry sector. PubMed was searched for publications from 1 January 2015 to 15 July 2026 using colistin or polymyxin or *mcr* and poultry or chicken or broiler or turkey or avian or meat or farm and Algeria. The literature search, screening, and verification were conducted during August and July 2026. Full texts and publication details of identified records were retrieved and verified through official journal and publisher websites, including The Lancet, Elsevier, eLife, ASM Journals, ScienceDirect, and the World Health Organization website. Reference lists of eligible publications and articles citing those publications were also checked to identify additional reports not retrieved by the PubMed search.

Screening the reference lists and citing articles identified one additional eligible study that had not been retrieved through the PubMed search. Eligible publications reported phenotypic colistin susceptibility or resistance, or detection of an *mcr* gene in poultry, poultry products, or poultry-associated environments in Algeria. Reviews, reports without Algerian poultry data, conference abstracts without primary data, and duplicate records were excluded. No language restrictions were applied during the literature search or study-selection process. Records were excluded solely on the basis of the predefined eligibility criteria.

The PubMed search identified 21 records, and one additional report was identified through reference and citation checking. After title and abstract screening, nine records were excluded. The excluded records comprised reviews (n = 3), studies involving non-poultry animals (n = 3), an agricultural-environment study without poultry-specific sampling (n = 1), and studies involving pigeons or wild birds outside the defined scope (n = 2). Thirteen reports were sought for retrieval; two could not be retrieved for complete eligibility verification. Eleven reports were assessed in full and included in the final narrative synthesis. Figure 1 summarizes the study-selection process using a PRISMA-style flow diagram. To reduce the risk of selection and extraction errors, eligibility decisions and extracted data were re-checked against the original publications before the final synthesis. Eleven studies meeting the final eligibility criteria are summarized in Table 1, which presents the study populations, colistin, *mcr* assessment methods, principal findings, and relevant genetic or interpretive context. Given the substantial heterogeneity among the included studies, no pooled estimate of national prevalence was calculated. No formal risk-of-bias assessment tool was applied, as this study was designed as a focused mini review rather than a systematic review or meta-analysis.

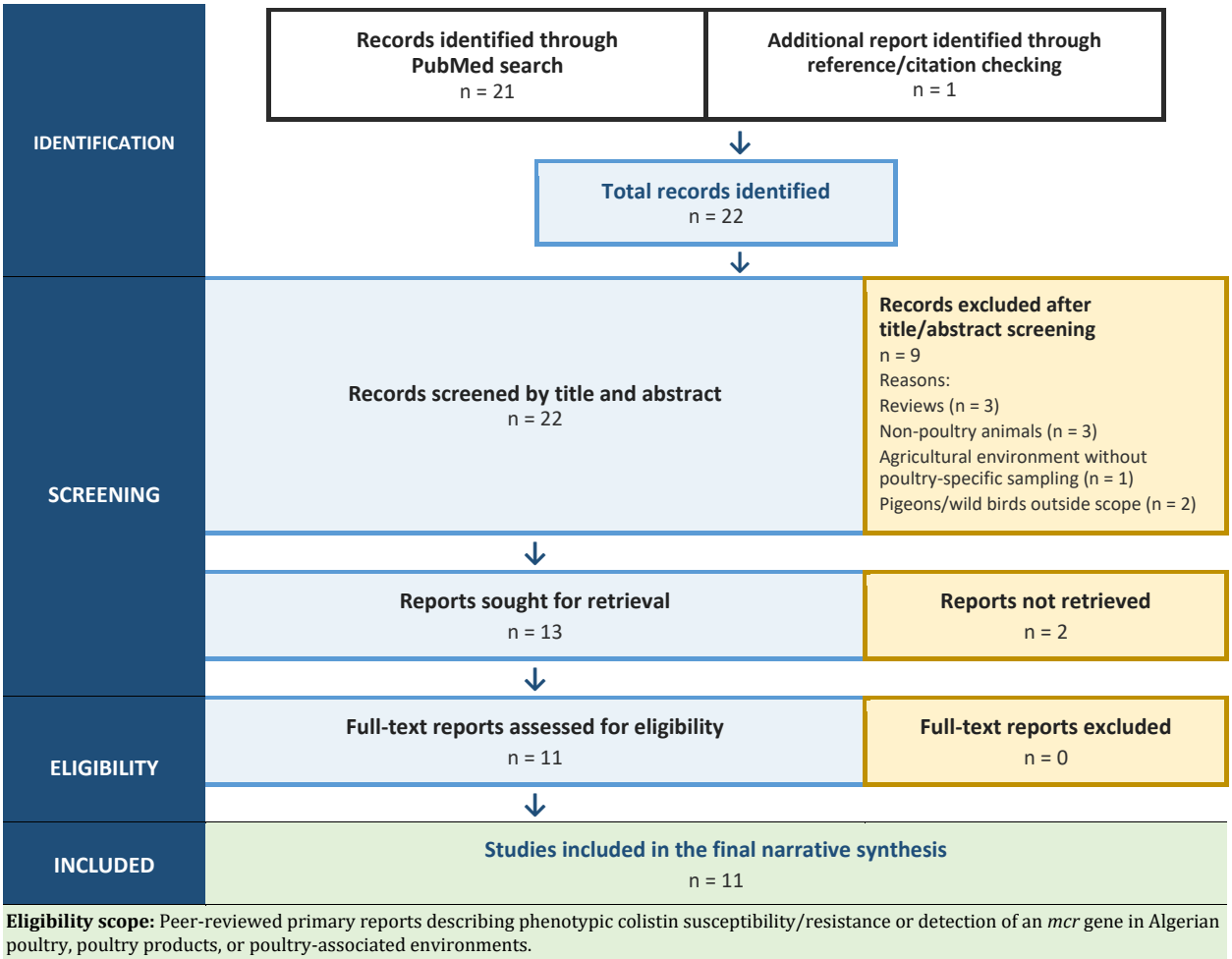


Figure 1. PRISMA flow diagram of study identification, screening, eligibility assessment, and inclusion on colistin resistance in the poultry sector in Algeria from 2015 to 15 July 2026

Table 1. Study-specific evidence and reported proportions of colistin resistance in the poultry sector in Algeria from 2015 to 2026

Study	Region/study area	Population/source and sample size	Colistin/*mcr* assessment	Main findings	Genetic/interpretive context
Chaalal et al.[5]	Western Algeria, three provinces	181 retail chicken-meat samples	Colistin susceptibility testing, *mcr-1* detection, plasmid characterization, conjugation	Colistin-resistant Enterobacterales were recovered from 22/181 samples (12.2%), *mcr-1*-positive *E. coli* were recovered from 11/181 samples (6.1%)	*mcr-1* was associated with IncFV (n=7) and IncFIIK (n=4) plasmid replicon types, and conjugative transfer was demonstrated
Chabou et al.[6]	Algiers, Blida, and Souk Ahras in Algeria	120 poultry fecal samples collected during a 2016 sampling campaign	Two qPCR systems, culture, conventional PCR, sequencing, MLST, and colistin susceptibility testing	*mcr-1* was detected in 25/120 samples (20.8%). Eight *mcr-1*-positive *E. coli* isolates were recovered and were phenotypically resistant to colistin	MLST assigned all eight *E. coli* isolates to sequence type 48 (ST48)
Halfaoui et al.[9]	Central Algeria	100 *E. coli* isolates from poultry suspected of colibacillosis	BMD for colistin MIC, PCR screening for *mcr-1* to *mcr-5*, virulence-gene PCR	Twenty-seven of 100 isolates were colistin-resistant, 26/27 colistin-resistant isolates carried *mcr-1* (96.3%)	The isolates were obtained from suspected clinical cases
Akkari et al.[10]	Eastern Algeria, Batna, Sétif, Biskra, and M'Sila	360 *E. coli* isolates from apparently healthy broiler chickens, broiler breeders, breeding hens, and laying hens	BMD, PCR, and Sanger sequencing for *mcr-1* to *mcr-5*, conjugation	Fourteen of 360 isolates (3.9%) were colistin-resistant, 11/14 carried *mcr-1*, and 8/14 carried both *mcr-1* and mcr-5. The remaining three colistin-resistant isolates were *mcr-1*-negative	*mcr-2*, *mcr-3*, and *mcr-4* were not detected. Conjugation demonstrated transferability of *mcr-1* from one isolate
Kirat et al.[11]	Guelma, Northeastern Algeria	Ten broiler farms, 186 pooled animal-environmental samples	*mcr-1* detection, WGS and molecular typing	All 54 recovered *E. coli* isolates carried *mcr-1* and were multidrug-resistant, positive isolates were recovered from feces, drinking water, wastewater, wall swabs, and soil	WGS identified 28 *E. coli* sequence types. The study identified sequence type 162 (ST162), sequence type 93 (ST93), and sequence type 3941 (ST3941) more than once, *mcr-1* was located on an IncI2(delta) plasmid
Chabou et al.[12]	El Tarf, Souk Ahras, Skikda, Sétif, Jijel, Algiers, Biskra, and Ouargla in Algeria	503 broiler fecal samples from Algeria and 330 samples from Marseille, France, for a total of 833 samples	Field application of the newly developed qPCR assay, culturing, conventional PCR and sequencing	*mcr-1* was detected in 5/503 Algerian samples, three *mcr-1*-positive *E. coli* isolates were subsequently recovered and confirmed	For the Algerian subset, the relevant sample size was 503 detected by qPCR
Olaitan et al.[15]	Algeria; specific region not reported	Chicken-derived *E. coli* isolates, source-population sample size was not reported	Phenotypic colistin resistance, PCR and sequencing for *mcr-1*	One colistin-resistant *E. coli* isolate carried *mcr-1*, the second colistin-resistant *E. coli* isolate was *mcr-1*-negative	Early evidence of colistin resistance and *mcr-1* occurrence in Algerian poultry
Djouzi et al.[16]	Algiers, Boumerdes, Bouira, Tizi Ouzou, Blida, Medea, Ain Defla, Batna, and M'Sila in Algeria	46 poultry farms, 113 APEC isolates recovered from broiler chickens, laying hens, and turkeys	Antimicrobial susceptibility testing, BMD for colistin MIC, *mcr-1* detection, Clermont phylogrouping, virulence profiling, ERIC-PCR	*mcr-1* was detected in 41/113 APEC isolates (36%). Among the 41 *mcr-1*-positive isolates, 33/41 (80%) were phenotypically resistant to colistin, and 8/41 (20%) remained phenotypically susceptible. Overall, 107/113 isolates (95%) were MDR	Among the *mcr-1*-positive isolates, 31/41 (76%) belonged to *E. coli* phylogroup B1. ERIC-PCR grouped the *mcr-1*-positive isolates into six clusters
Djeffal et al.[17]	Skikda, Northeastern Algeria	32 broiler farms and five slaughterhouses yielded 1,194 poultry environmental samples and 45 avian *Salmonella* isolates	Disk-diffusion susceptibility testing included colistin	The main article did not report a separate colistin-resistance proportion for the avian *Salmonella* isolates	The study provided historical poultry susceptibility data, but no directly extractable colistin-resistance proportion and no molecular *mcr* result
Aberkane et al.[18]	Batna, Biskra, Skikda, Tebessa, and Guelma, Eastern Algeria	204 broiler carcasses suspected of colibacillosis, 160 APEC isolates recovered	Disk-diffusion antimicrobial susceptibility testing	Twenty-three of 160 APEC isolates (14.37%) were reported as colistin-resistant	*mcr* genes were not investigated
Kamel et al.[19]	Souk Ahras, Eastern Algeria	200 cloacal swabs from apparently healthy broiler chickens, 241 commensal *Enterobacteriaceae* isolates recovered, including 194 *E. coli*	Disk diffusion for the general antimicrobial panel, BMD for colistin MIC	61/241 *Enterobacteriaceae* isolates (25.31%) were colistin-resistant, among *E. coli*, 38/194 isolates (19.58%) were colistin-resistant	No molecular screening for *mcr* genes was reported

APEC: Avian pathogenic *Escherichia coli*, BMD: Broth microdilution, ERIC-PCR: Enterobacterial repetitive intergenic consensus polymerase chain reaction, MDR: Multidrug-resistant, MIC: Minimum inhibitory concentration, MLST: Multilocus sequence typing, PCR: Polymerase chain reaction, qPCR: Quantitative polymerase chain reaction, ST: Sequence type, WGS: Whole-genome sequencing

3. Colistin exposure and selection pressure

By oral administration, colistin is poorly absorbed from the gastrointestinal tract and therefore remains largely within the intestinal lumen[8]. This pharmacokinetic pattern supports treatment of susceptible gastrointestinal infections caused by non-invasive *E. coli* in poultry[8]. In animal health, colistin has been used to prevent infections caused by *E. coli*[13]. Oral colistin exposure may simultaneously exert antimicrobial selection pressure on less susceptible intestinal *E. coli* populations[8]. Colistin resistance has been reported in bacteria from food-producing animals, including pathogenic *E. coli*, and the emergence of *mcr-1* has heightened concern regarding veterinary colistin use[13]. During oral colistin exposure, the dosing schedule and the presence of heteroresistant *E. coli* subpopulations may influence the bacterial response to treatment and the persistence of less susceptible subpopulations[8,14]. Among the included Algerian studies, *mcr-1* was reported in poultry fecal samples[6,12], retail chicken meat[5], disease-associated APEC[9], apparently healthy poultry[10], and poultry-farm environments[11]. Phenotypic colistin resistance was also documented in retail chicken meat[5], disease-associated APEC[9], and apparently healthy poultry[10].

4. Epidemiological evidence

Olaitan et al.[15] reported an *mcr-1*-positive, colistin-resistant *E. coli* isolate recovered from chicken feces in Algeria. Additionally, Olaitan et al.[15] reported another colistin-resistant *E. coli* isolate in which *mcr-1* was not detected[15]. These findings provided early evidence of colistin resistance in Algerian poultry and documented the occurrence of the plasmid-mediated colistin resistance gene *mcr-1*. As part of the field application of a newly developed real-time polymerase chain reaction (qPCR) assay, Chabou et al.[12] screened 503 broiler fecal samples collected from eight regions in Algeria. Five of the 503 Algerian samples tested positive for *mcr-1*, and three *E. coli* isolates were subsequently recovered and confirmed by conventional PCR and sequencing[12]. In a separate study published in 2019, Chabou et al.[6] presented findings from a 2016 sampling campaign that collected 120 poultry fecal samples in Algiers, Blida, and Souk Ahras, Algeria. The *mcr-1* gene was detected in 25/120 samples (20.8%). Eight *E. coli* isolates carrying *mcr-1* were subsequently recovered; all eight were phenotypically resistant to colistin, with minimum inhibitory concentrations (MICs) of 3–4 mg/L, and were assigned to sequence type ST48 by multilocus sequence typing (MLST)[6]. Chaalal et al.[5] examined 181 chicken-meat samples collected from three poultry farms across three provinces in Western Algeria. Colistin-resistant Enterobacterales were recovered from 22/181 samples (12.2%), comprising 17 *E. coli* and five *Klebsiella pneumoniae* isolates. The plasmid-mediated colistin resistance gene *mcr-1* was detected exclusively in 11 *E. coli* isolates, corresponding to 11/181 samples (6.1%). The *mcr-1* gene was associated with IncFV (n = 7) and IncFIIK (n = 4) plasmids, and the transferability of *mcr-1*-carrying plasmids was assessed by conjugation[5]. Halfaoui et al.[9] examined 100 *E. coli* isolates recovered from poultry suspected of colibacillosis in central Algeria. Broth microdilution identified 27/100 isolates as colistin-resistant. Among these 27 colistin-resistant isolates, 26 (96.3%) carried the plasmid-mediated colistin resistance gene *mcr-1*, while one resistant isolate was *mcr-1*-negative. In addition, 25/27 colistin-resistant isolates harbored at least three virulence-associated genes[9]. These clinical isolates did not reflect carriage prevalence in healthy poultry[9]. Djouzi et al.[16] recovered 113 APEC isolates from poultry sampled across 46 farms in several Algerian provinces. Overall, 107/113 isolates (95%) were multidrug-resistant. The plasmid-mediated colistin resistance gene *mcr-1* was detected in 41/113 isolates (36%), all of which were multidrug-resistant. Colistin MIC testing indicated that 33/41 (80%) *mcr-1*-positive isolates were phenotypically resistant to colistin, while 8/41 (20%) remained phenotypically susceptible. Among the *mcr-1*-positive isolates, 31/41 (76%) belonged to phylogroup B1[16]. Akkari et al.[10] examined 360 *E. coli* isolates recovered from apparently healthy broiler chickens, broiler breeders, breeding hens, and laying hens in Algeria. Fourteen isolates (3.9%) were phenotypically resistant to colistin by broth microdilution, with MICs ranging from 4 to 32 μg/mL. Among these 14 colistin-resistant isolates, 11 carried *mcr-1*, and eight carried both *mcr-1* and *mcr-5*. The remaining three colistin-resistant isolates were *mcr-1*-negative. The *mcr-2*, *mcr-3*, and *mcr-4* genes were not detected by Akkari et al.[10]. Conjugation assays demonstrated transferability of *mcr-1* from one isolate[10]. These findings indicated intestinal carriage of colistin-resistant, *mcr*-positive *E. coli* in apparently healthy poultry in Algeria[10]. Kirat et al.[11] investigated ten broiler farms in Guelma, Algeria, using 186 pooled animal and environmental samples, and recovered 44 *E. coli* isolates; all carried *mcr-1* and were multidrug-resistant. The *mcr-1*-positive isolates were recovered from feces, drinking water, wastewater, wall swabs, and soil. Whole-genome sequencing identified 28 sequence types, including recurrent ST162, ST93, and ST3941, and demonstrated that *mcr-1* was carried on an IncI2(delta) plasmid[11]. Djeffal et al.[17] assessed antimicrobial susceptibility in 45 avian *Salmonella* isolates collected from poultry farms and slaughterhouses in Skikda, Northeastern Algeria. Colistin was included in the disk-diffusion susceptibility panel; however, the study did not report a separate colistin-resistance proportion and did not investigate *mcr* genes[17]. Aberkane et al.[18] recovered 160 APEC isolates from 204 broiler carcasses suspected of colibacillosis in eastern Algeria. Using disk-diffusion susceptibility testing, 23/160 isolates (14.37%) were reported as resistant to colistin[18]. Kamel et al.[19] examined 200 cloacal swabs collected from apparently healthy broiler chickens on poultry farms in Souk Ahras, Eastern Algeria, and recovered 241 commensal *Enterobacteriaceae* isolates. Colistin susceptibility was determined by broth microdilution, and 61/241 isolates (25.31%) were colistin-resistant; among the 194 *E. coli* isolates, colistin resistance was 19.58%[19]. The conceptual pathways for the selection and dissemination of colistin-

resistant Enterobacterales in the poultry production chain are summarized in Figure 2.

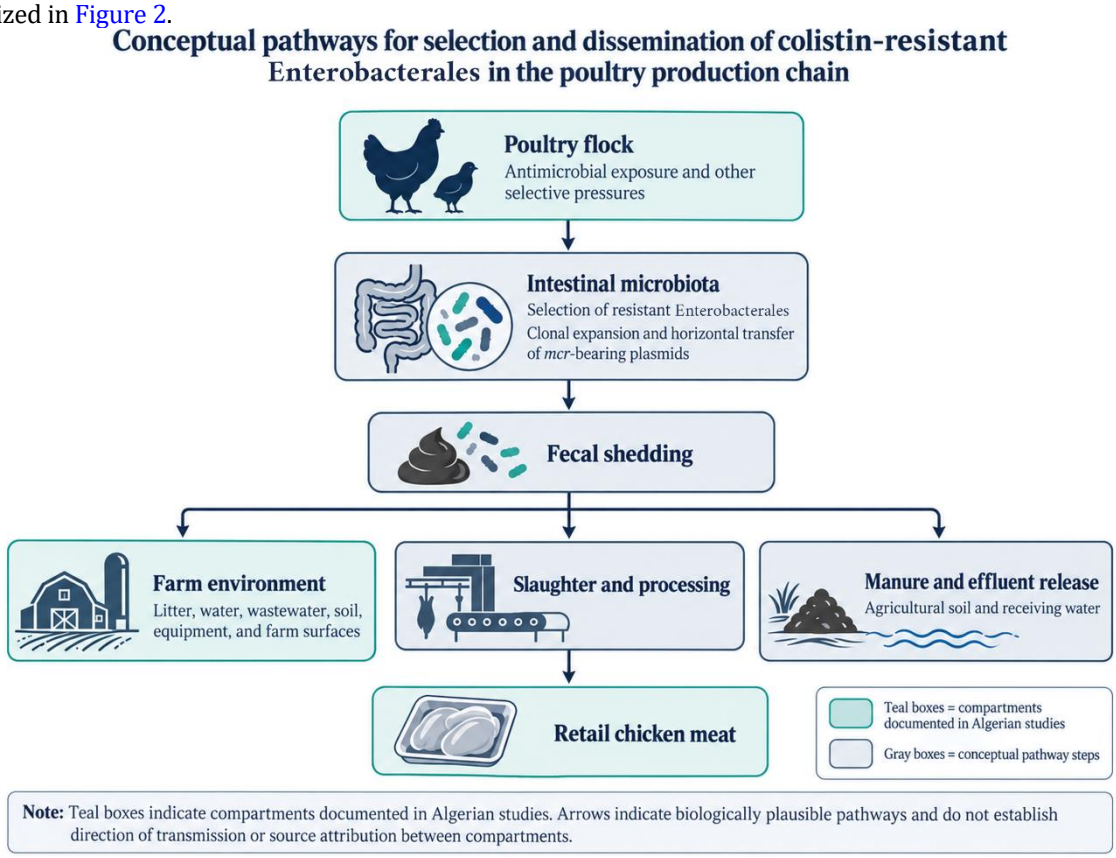


Figure 2. Conceptual pathways for selection and dissemination of colistin-resistant Enterobacterales in the poultry production chain in Algeria. The author generated the conceptual figure using OpenAI ChatGPT and subsequently edited it in Adobe Photoshop for graphical and typographical refinement.

5. Molecular interpretation of colistin resistance

5.1. Mobile colistin resistance

Mobile colistin resistance enzymes confer resistance by adding phosphoethanolamine to lipid A, reducing colistin interaction with the bacterial outer membrane[1,3]. *mcr-1* was the most frequently reported mobile colistin-resistance gene among the included Algerian poultry studies, whereas Akkari et al.[10] reported *mcr-5*. The dissemination of *mcr* genes is closely associated with mobile genetic elements, particularly plasmids. In the. Algerian poultry studies reviewed, Chaalal et al.[5] demonstrated conjugative transfer of *mcr-1*-carrying plasmids from retail chicken-meat isolates, while Akkari et al.[10] demonstrated transferability of *mcr-1* from one isolate recovered from apparently healthy poultry. Kirat et al.[11] reported that all 54 *E. coli* isolates recovered from poultry farms in Guelma, Northeastern Algeria, carried *mcr-1*. The *mcr-1* gene was located on an IncI2(delta) plasmid[11]. Whole-genome sequencing provides detailed characterization of *mcr*-carrying isolates by identifying sequence types, antimicrobial resistance genes, plasmid replicons, and genetic relatedness[4]. In the Algerian poultry context, Kirat et al.[11] used whole-genome sequencing to identify 28 *E. coli* sequence types among the recovered isolates, providing additional information on the genetic diversity of the *mcr-1*-positive population.

5.2. Chromosomal resistance and susceptibility testing

Colistin resistance may result from chromosomal changes affecting lipid A regulation, including alterations in the PmrAB and PhoPQ regulatory systems and disruption of *mgrB* in some Enterobacterales[3]. Phenotypic colistin resistance did not necessarily indicate the presence of an *mcr* gene; therefore, phenotypic susceptibility results and molecular detection of *mcr* genes were interpreted separately[3]. Broth microdilution was the reference method for determining colistin MICs[3]. Diffusion-based methods were not recommended for colistin susceptibility testing because polymyxins diffuse poorly in agar and may produce unreliable categorical results[3].

6. Veterinary and One Health context

In Algerian APEC collections, *mcr-1* occurred with multidrug-resistance and virulence-associated genes[9,16]. Chaalal et al.[5] detected *mcr-1*-positive *E. coli* in retail chicken meat in western Algeria, demonstrating the presence of the *mcr-1* colistin resistance gene in a poultry-derived food product. However, the study did not assess transmission to consumers. Kirat et al.[11] recovered *mcr-1*-positive *E. coli* from poultry-farm environmental sources in Guelma, Algeria, demonstrating the presence of *mcr-1*-positive *E. coli* in the sampled farm environment, but the study did not establish the direction of transmission between poultry and environmental sources. Beyond the poultry sector, *mcr-1* has also been reported in a human clinical *E. coli* isolate in

Algeria and in an *Enterobacter kobei* isolate recovered from a cat in Batna, indicating that the *mcr-1* colistin resistance gene was not confined to poultry-associated settings[20,21]. Studies conducted outside Algeria have reported *mcr-1*-carrying bacteria and plasmids in animal, food, human, and environmental sources[1,4,13].

7. Control priorities

Control priorities should focus on reducing antimicrobial use and the disease burden that creates treatment demand. International guidance recommended not using antimicrobials to promote growth and advised avoiding routine preventive use of medically important antimicrobials in healthy food-producing animals[22]. Antimicrobial treatment in poultry should be guided by veterinary assessment, a defined clinical indication, and, when appropriate, bacterial culture and antimicrobial susceptibility testing[22]. Disease-prevention measures should include improved hygiene, biosecurity, appropriate poultry husbandry practices, and vaccination[22]. In broiler chickens, probiotics, prebiotics, organic acids, and other feed interventions may support intestinal health, although their effects vary according to the product, dose, flock conditions, and measured outcome[23]. During slaughter and processing, evisceration, equipment surfaces, carcass chilling, and subsequent handling are key points where bacterial contamination or cross-contamination of poultry meat may occur. Appropriate hygiene and temperature control during these stages therefore help limit microbial contamination[24]. Integrated monitoring of antimicrobial resistance and antimicrobial use within a One Health framework can support the assessment of resistance trends in humans and food-producing animals[22].

8. Conclusion

The present study reported phenotypic colistin resistance and *mcr* genes in Algerian poultry and poultry-associated sources, including diseased and apparently healthy chickens, poultry feces, farm environment, water, soil, and retail chicken meat. The *mcr-1* was the most frequently detected gene, while *mcr-5* was identified in apparently healthy poultry. However, differences among the included studies in geographic coverage, poultry populations, bacterial species, sampling units, and susceptibility-testing methods limited estimation of a national prevalence of colistin resistance or *mcr* carriage in Algeria. Future studies are recommended to use representative sampling across major poultry-production regions and categories, reference colistin susceptibility testing, and molecular characterization of colistin-resistant *E. coli* and other Enterobacterales, including *mcr* genes and their associated plasmids.

Declarations

Availability of data and materials

No new datasets were generated or analyzed. All evidence discussed in this study is available in the cited publications.

Authors' contributions

The author conceived the study, defined its scope, searched and interpreted the literature, drafted and critically revised the manuscript, approved the final edition, and accepts accountability for the study.

Competing interests

The author declares no competing interests.

Ethical considerations

The author confirms adherence to accepted standards of scholarly publishing, including avoiding plagiarism, fabrication, falsification, and duplicate submission. The manuscript was checked for originality and had not been submitted elsewhere before submission to this journal. The author used ChatGPT (OpenAI) only for AI-assisted language editing to improve wording and clarity. The author reviewed and verified the scientific content and final presentation of the AI-edited text and takes full responsibility for it. It was not used to generate data, conduct the literature search, select studies, or interpret the evidence. The author reviewed and verified the final text and accepts full responsibility for the accuracy, originality, and integrity of the manuscript.

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