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DIETARY EXPOSURE TO ANTIMICROBIAL RESISTANCE GENES (ARGs) FROM LIVESTOCK PRODUCTS AND ITS IMPLICATIONS FOR GUT HEALTH IN CHILDREN – A REVIEW

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ABSTRACT

Background: *ARGs found in animal-derived foods represent a significant route of antimicrobial resistance transmission beyond clinical antibiotic use. Livestock products such as meat, milk, eggs, and fish from animals exposed to antibiotics frequently contain diverse ARGs. Because the gut microbiota of infants and young children is still forming, dietary exposure to ARGs may have amplified biological effects during early development.*

Objective: *This review aims to synthesize current evidence the presence of ARGs in livestock-based foods, evaluates how dietary intake may contribute to ARG exposure in children, and describes the mechanisms through which these genes may influence gut microbiota development and child health.*

Methods: *A comprehensive literature search were conducted in PubMed, MEDLINE, Web of Science, and Google Scholar. English-language studies involving human or animal subjects were included if they examined ARGs in animal-source foods or their implications for gut microbiota and pediatric health. Findings from cohort studies, randomized controlled trials, and mechanistic research were integrated narratively.*

Results: *Studies report ARGs such as blaCTX-M, mecA, tetM, sul1, and gyrA in poultry, beef, milk, eggs, and fish. Following ingestion, ARGs may interact with the pediatric gut through horizontal gene transfer—via conjugation, transformation, or transduction—promoting dysbiosis, increasing susceptibility to infection, reducing antibiotic effectiveness, and influencing immune function during early life.*

Conclusion: *Dietary exposure to ARGs from animal-source foods may contribute to early establishment of the gut resistome in children and alter microbiota-related functions. Strengthened control of ARGs along the food production chain is essential to reduce exposure and protect child health.*

Keywords*: Antimicrobial Resistance Genes (ARGs); gut microbiota; diet; children*

INTRODUCTION

Antimicrobial Resistance (AMR) is a critical global health challenge of the 21st century, with projections of 10 million annual deaths by 2050.[1] The origin of the problem of AMR is associated with the emergence of Antimicrobial Resistance Genes (ARGs) – which are genetic components able to confer survival advantage to bacteria in the presence of antibiotics.[2] ARGs originate from environmental bacteria and can be transferred to pathogens through mobile genetic elements.[3] Although clinical overuse of antibiotics is a widely acknowledged contributor AMR, recent research highlights the growing concern over non-clinical exposure pathways to AMR, particularly through the food chain.[4] The presence of resistant bacteria in the food supply chain poses a potential risk to public health.[5]

A key yet overlooked contributor to ARGs is the extensive use of antibiotics in livestock production, where animals routinely receive antibiotics for the treatment and prevention of diseases as well as for enhancing growth.[4] These practices, along with the feeding of animal byproducts to livestock, may result in the selection and enrichment of resistant bacteria within a host's microbiota and its environment (manure), as well as in the animal's food products, including meat, milk, and eggs.[6–9] By consuming these products, children and other susceptible populations may be exposed to dietary ARGs that are capable of integrating into the gut microbiome and facilitating horizontal gene transfer to microbiota associated with the intestine—both commensals and pathogenic microbes.[10]

In this case, children represent an age group that is especially at risk. Because their gut microbiota has not fully developed, it is prone to

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outside influence.[11,12] The damage caused by exposure to ARGs might not only heighten the danger of maintaining resistant bacteria, but also undermine immune functions, metabolic health, and the efficacy of subsequent antibiotic treatments.[11,13]

This review aims to synthesize current knowledge on the presence of ARGs in livestock-derived foods, assess potential pathways of dietary exposure in children, and explore the impacts of such exposure on gut microbiome composition and health outcomes.

METHODS

A comprehensive literature search was conducted in PubMed, MEDLINE, Web of Science, and Google Scholar to identify studies discussing dietary exposure to antimicrobial resistance genes (ARGs) from animal-derived foods and their implications for gut microbiota development in children. No restrictions on publication year were applied, ensuring inclusion of both foundational studies and recent advances.

The search strategy incorporated relevant keywords and their synonyms—such as "antimicrobial resistance genes", "ARGs", "livestock products", "animal-sourced food", "dietary exposure", "gut microbiota", "infant", "children", and "randomized controlled trial"—combined using Boolean operators (AND, OR) to refine the query. Both human and animal studies were considered eligible when they examined the presence or transmission of ARGs through food consumption and its potential influence on the gut microbiota or host health outcomes. Only articles published in peer-reviewed journals and available in English were included. Priority was given to experimental evidence, including randomized controlled trials (RCTs), controlled feeding studies, and mechanistic investigations that elucidate transmission pathways or biological consequences of ARG exposure.

Although adopting a narrative review framework, this work integrates data from multiple study types to provide a broad, evidence-based synthesis rather than a systematic meta-analysis. RCTs and cohort studies were used selectively to reinforce mechanistic arguments and clarify causal relationships, not to perform pooled quantitative analysis. By combining contextual interpretation with high-quality empirical evidence, this review presents a balanced overview of current knowledge regarding ARG prevalence in livestock-derived foods, dietary exposure pathways in children, and their implications for microbiome development and antibiotic resistance dynamics.

RESULT AND DISCUSSION

The use of antibiotics in livestock production has become a key contributor to the emergence and dissemination of ARGs in the food chain.[14] These ARGs, initially selected within the gut microbiota of food-producing animals, can persist in meat, milk, and eggs that are commonly consumed by humans, including young children.[7,15,16] As a result, dietary exposure to ARGs has emerged as a significant, yet underexplored, route of AMR transmission, with potential long-term consequences for gut health—especially in early childhood when the microbiome is most vulnerable.[10,14]

Sources of ARGs in Livestock Products

Antibiotics are routinely administered to livestock for therapeutic, prophylactic, and growth-promoting purposes, practices that create selective pressure for antibiotic-resistant bacteria.[17,18] These practices generate strong selective pressures that favor the proliferation of antibiotic-resistant bacteria in the gastrointestinal tracts, skin, and mucosal surfaces of livestock.[18] The result is a microbiome enriched with ARGs, which can persist in animal waste, the environment, and eventually in the food chain.[19] Once acquired, these genes can be maintained in bacterial populations even in the absence of continuous antibiotic exposure, increasing the likelihood of long-term environmental and foodborne contamination.[18]

These ARGs and their bacterial hosts can contaminate animal-derived food products.[14,19] Multiple studies have documented the presence of ARGs in raw and processed meats, as well as in milk, dairy, and eggs.[20,21] For example, genes such as *mecA*, *tetM*, *sul1*, and *blaCTX-M* have been detected in *Staphylococcus aureus* and *E. coli* strains isolated from retail meat and ready-to-eat products (Tabel 1).[8,16,22] Metagenomic analyses of raw milk have also confirmed ARG enrichment during improper storage, further underscoring the importance of post-farm handling practices.[21,23]

The presence of ARGs in livestock products is not necessarily limited to viable bacterial cells.[14] Heat-treated or processed foods can still carry extracellular DNA fragments that retain the capacity to transfer ARGs to gut bacteria under favorable conditions.[24] Recent studies have shown that DNA can survive common cooking temperatures and remain functionally active, suggesting that ingestion of even heat-processed animal products could contribute to ARG exposure.[25,26] This challenges traditional food safety paradigms that primarily focus on microbial viability and neglect the genetic dimension of resistance.[26]

Dietary Exposure to ARGs in Children

Dietary exposure is increasingly recognized as an important, yet underexplored, pathway through

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which ARGs are introduced into the human gut microbiome, particularly in children.[10] Although environmental and clinical antibiotic exposures have historically dominated discussions on resistance, current research has revealed the substantial impact of animal-derived food products in promoting the dietary intake of ARGs.[31] This issue is exacerbated in the pediatric population, where dietary practices, metabolic susceptibility, and the developing condition of the gut microbiota collectively increase the potential effects of such exposures.[32–34]

Children often ingest milk, eggs, poultry, beef, and processed meat products, all of which have demonstrated the presence of measurable quantities of ARGs and antibiotic-resistant bacteria.[35,36] Specifically, cattle subjected to regular antibiotic exposure—either for growth enhancement or preventive purposes—exhibit gut microbiota characterized by elevated ARG loading. These genes may be transmitted to zoonotic and commensal bacteria that contaminate meat, milk, and egg products during slaughter, processing, or storage.[20,31] Numerous studies have validated the existence of clinically significant ARGs, including blaCTX-M, tetM, sul1, and mecA, in raw chicken, beef, retail pork, and unpasteurized milk intended for human consumption.[7–9,15,16,28]

Children ingest a greater quantity of food per kilogram of body weight than adults and frequently depend significantly on animal-derived proteins and dairy for their nutritional requirements.[10,37] Infants and toddlers, especially during weaning, may ingest substantial quantities of formula or milk and pureed meats.[10,38] If these products derive from livestock administered antibiotics, they may serve as conduits for dietary ARG transfer.[35,36] Processed or cooked foods are not completely devoid of risk; although heat treatment can diminish bacterial viability, extracellular DNA harboring antibiotic resistance genes may persist stable and biologically active in gastrointestinal environments, particularly amid inflammation or dysbiosis.[24,25]

In addition to animal products, vegetables and fruits irrigated with contaminated water or fertilized with untreated manure may potentially transmit antimicrobial resistance genes into the diet.[39,40] This topic centers on animal-based sources; nevertheless, it is important to recognize that integrated food systems can generate intricate exposure pathways, allowing resistant bacteria to disseminate from farm to fork via common water, soil, and handling conditions.[41]

Once ingested, ARGs from food may interact with the growing gut microbiota of the infant.[42] During the initial years of life, the gastrointestinal environment is exceptionally amenable to colonization, and the microbiome is in the process of development.[42,43] ARGs can be assimilated by indigenous gut microbiota via HGT processes, so creating a reservoir of resistance independent of therapeutic antibiotic use.[44,45] This may not only lead to long-term dysregulation of microbiota but also elevate the risk of colonization by multidrug-resistant pathogens that are challenging to treat if an infection arises.[46,47]

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Tabel 1. Recent Studies of ARGs Source in Foods

Study Title	Food type	Detected ARGs	Methodology	Main Findings
Antimicrobial Susceptibility, and Molecular Characterization of *Staphylococcus aureus* Isolated from Different Raw Milk Samples in China.[16]	Raw Milk	blaZ, aac6′-aph2″, tet(M), mecA, ant(6)-Ia, fexA, sec	qPCR, metagenomics	Raw milk samples contained multiple ARGs, with higher loads in samples stored improperly.
Presence of Antibiotic Resistance Genes in Bacteria Isolated from Raw Cow Milk obtained from Bowen University Dairy Farm.[7]	Raw milk	TEM, SHV-1, CTX-M	qPCR, metagenomics	A high rate of antibiotic resistance was observed in bacteria isolated from raw cow milk, with 81.8% resistance to cefuroxime and 98.1% resistance to cefixime and amoxicillin/clavulanate.
Prevalence of *Escherichia coli* in Marked Poultry Carcasses in Egypt.[27]	chicken meat samples, including breast, thigh, lung, liver, and gizzard cuts-up	*stx1* and *stx2*	qPCR, metagenomics	The study found a high prevalence (66.3%) of multidrug-resistant *E. coli* in retail chicken meat in Egypt.
Molecular detection of antimicrobial resistance genes in *E. coli* isolated from slaughtered commercial chickens in Iran.[8]	Chiken meat	*tet(A), tet(B), dfrA1, qnrA, aac(3)-IV, sul1, bla SHV , bla CMY , ere(A), catA1 and cmlA.*	qPCR, metagenomics	This study detected the distribution of antibiotic-resistant genes in *E. coli* isolates from commercial chickens in Iran using PCR, finding a high prevalence of multi-resistance and validating PCR as an effective detection method.
Multi-Drug Resistance to *Salmonella spp*. When Isolated from Raw Meat Products.[9]	Beef, pork, poultry meat	blaSHV, blaPSE-1, blaTEM.	qPCR, metagenomics	Salmonella spp. is a major cause of foodborne diseases with an increasing problem of multi-drug resistance (MDR) strains.
Diversity of Antibiotic Resistance Genes in *Enterococcus* Strains Isolated from Ready-to-Eat Meat Products.[28]	Ready-to-eat meat products	ant(6')-Ia, tetM, ermB	qPCR, metagenomics	Enterococcus strains were found in 74.1% of ready-to-eat meat product samples, indicating a widespread presence. A high percentage of isolates showed resistance to various antibiotics, including streptomycin and erythromycin.
Prevalence and distribution of antibiotic resistance in marine fish farming areas in Hainan, China.[29]	Fish	*sul1* and *tetB*	qPCR, metagenomics	Sul and tet family genes were widely distributed in marine fish farming areas in Hainan, China. The total abundance of ARGs increased significantly from the rearing to the harvesting period.
ARGs Detection in *Listeria Monocytogenes* Strains Isolated from the Atlantic Salmon (Salmon salar) Food Industry: A Retrospective Study.[30]	Salmon fresh and smoke fillets	tetC, tetD, tetK, tetL, tetS,aadA, strA, aacC2, aphA1, aphA2, cmlA1, catI, catII, cfr, optrA, poxtA	qPCR, metagenomics	This retrospective study investigated the trend of antibiotic resistance genes in Listeria monocytogenes isolates from Atlantic salmon products and environmental samples over 15 years, finding

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Tabel 1. Recent Studies of ARGs Source in Foods *(Continue…)*

Study Title	Food type	Detected ARGs	Methodology	Main Findings
				significant resistance to various antibiotics and a consistent increase in resistance due to improper antimicrobial use.
Detection of Antibiotic Resistance Genes in *Escherichia coli* Isolated from Chicken Eggs.[15]	Eggs	tetA, tetB, gyrA	qPCR, metagenomics	High resistance rates were observed in E. coli isolates against various antibiotics, with 57.78% resistance to ciprofloxacin being the highest. PCR analysis showed that 82.22% of isolates carried the tetracycline resistance gene (tetA), 71.11% carried (tetB), and all isolates carried the quinolone resistance gene (gyrA).
Presence of antimicrobial resistance in coliform bacteria from hatching broiler eggs with emphasis on ESBL/AmpC-producing bacteria.[6]	Eggs	blaSHV–12, blaTEM–52, blaACT–39	qPCR, metagenomics	The study investigates the presence of antimicrobial-resistant coliform bacteria in broiler hatching eggs, finding that approximately 30% of isolates are resistant to certain antibiotics and confirming the presence of ESBL-producing bacteria, highlighting broiler hatching eggs as a potential source of these resistant bacteria for broiler chicks.

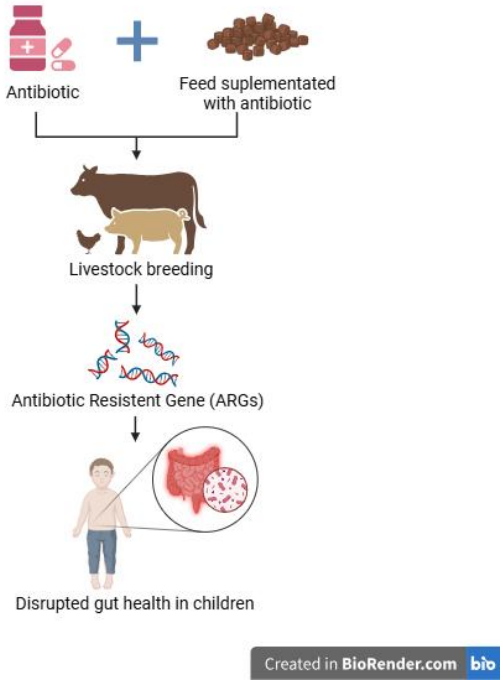

Figure 1. From Farm Antibiotics to Children's Gut Health

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Dietary exposure to ARGs in children has significant implications for treatment efficacy and immunological development.[10] If resistance traits get integrated into the microbiome, future antibiotic treatments may be less efficacious, heightening the risk of therapeutic failure.[42,48,49] This may result in prolonged illness durations, complications, or dependence on last-resort antibiotics.[49,50] Furthermore, dysbiotic changes caused by ARG-carrying bacteria may modify gut-immune interactions, which are particularly vital in early life for developing immunological tolerance and preventing inflammatory disorders.[43,50]

Implications for Gut Microbiota in Children

Antibiotic use within livestock and poultry farming practices, especially for growth stimulation and disease prophylaxis, has amplified the proliferation of ARGs in the food chain.[14] Meats, dairy products, and eggs are some of the animal-derived foods that often contain antibiotic-resistant bacteria along with mobile ARGs.[26,31] Child population is considered one of the most vulnerable groups and in this case, they are especially at risk due to relatively greater consumption of milk and protein-rich products per unit of body weight and their developing physiological systems.[32,42,43]

Once absorbed, antibiotic resistance genes ARGs may interact with the child's gut microbiota through various mechanisms on contaminated livestock products.[42] The gastrointestinal tract, primarily during the formative years of life, offers a liquid environment that is propitious for HGT to occur.[44] This includes processes such as conjugation (genetic material is directly transferred from one bacterial cell to another), transduction (mediated through viruses that infect bacteria known as phages), and transformation (where the bacteria take in foreign DNA found within its surroundings).[45] These ways embed ARGS into ordinary bacteria like obligate anaerobic commensal *Escherichia coli*, *Bacteroides*, and *Enterococcus* species.[51] Many young infants and toddlers have not fully developed their gut microbiome and thus possess and have low microbial diversity, which may promote colonization of in resistant strains.[34]

Establishing ARGs in the early-life microbiome poses threat to short or long-term health.[43,47] The infant gut microbiome has certain stages of colonization, so any newly introduced ARGs will always disrupt metabolic succession and stimulate gut microbial succession.[46] Introduction of ARGs at this time may hinder normal succession, inhibit fragments of versions of microbes that serve host functions, and accelerate opportunistic or antibiotic resistants version of microbes to dominate.[52] Changes of this nature can severely interfere with short chain fatty acid synthesis, disrupt gut barrier strength, activate immune signaling sas and cause alterations on overall effect of the immune system and metabolism.[10,52] (Table 2)

Increased risk of infections is one of the most immediate clinical consequences of this type of colonization.[47] The resistant strains that survive in the gastrointestinal tract can serve as reservoirs for potential systemic infections, such as urinary tract infections, bloodstream infections, or other gastrointestinal diseases, especially when the host is immunocompromised.[53] These infections may be difficult to manage because there is resistance to standard treatment, requiring costlier and more harmful substitutes.[47,48,54] In addition, the presence of ARGs among the commensal flora may render some antibiotic treatment less effective because resistance genes can either deactivate antiobiotics or expel them from the bacterial cell before any therapeutic action can take place.[11,32,53] This is particularly concerning in pediatric populations, where antibiotics are often prescribed empirically, and where the dosing schedules are directly proportional to age and weight.[48,50,54,55]

Beyond infection control, ARGs in gut may also contribute to immune dysregulation.[53,56] The early microbiota plays a pivotal role in educating the immune system, helping it distinguish between harmless commensals and pathogenic threats.[57] Disruption of this process—through exposure to ARG-carrying pathogens or commensals—may skew immune development toward pro-inflammatory or allergic phenotypes.[58] Several studies have proposed associations between early-life dysbiosis and the onset of conditions such as asthma, eczema, type 1 diabetes, and inflammatory bowel disease, although the role of ARGs in these processes remains an area of active investigation.[57–59]

Infants possess metabolic and developmental characteristics that differ from older children, rendering them more vulnerable to the effects of dietary exposure to antimicrobial resistance genes (ARGs).[10,19] During early life, rapid growth and the maturation of organ systems place substantial metabolic demands on the host. .[60–62] At this stage, the gut microbiota remains limited in diversity and highly responsive to external inputs, such that even modest alterations in microbial composition may influence nutrient processing and metabolic function.[10,48,63] The infant gut relies on microbial fermentation to produce short-chain fatty acids (SCFAs), which support epithelial integrity, mucosal immunity, and energy regulation.[64] Exposure to ARG-carrying bacteria or extracellular DNA has the potential to disturb these fermentative pathways, modify SCFA profiles, and reduce

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mucosal protection, thereby contributing to inefficient energy utilization and increased susceptibility to inflammatory processes.[10,42,48] In contrast, older children exhibit greater microbial diversity and a more stable gut ecosystem, which affords partial resilience against dietary disturbances.[64,65] Nonetheless, continued intake of ARGs from livestock-derived foods may contribute to low-grade dysbiosis and subtle shifts in metabolic pathways.[10,50] Evidence from pediatric microbiome studies indicates that prolonged ARG exposure can affect bile acid metabolism, insulin sensitivity, and lipid regulation, linking early-life resistome enrichment with heightened metabolic risk later in childhood.[66,67]

Mechanistically, ARGs located on mobile genetic elements can alter the functional capacity of commensal microbes by influencing pathways involved in carbohydrate fermentation and amino acid metabolism.[68–70] These genomic shifts may reduce microbial efficiency, diminish diversity, and limit the production of metabolites essential for maintaining homeostasis. In infants, such disturbances may have longer-term implications due to the ongoing development of metabolic and immune systems.[10,70] Taken together, these observations underscore that the metabolic consequences of dietary ARG exposure vary across developmental stages.[62,71] Infancy represents a period of heightened susceptibility due to rapid physiological development and an evolving gut microbiome.[70] Reducing ARG exposure during this window is therefore essential for supporting normal metabolic and immunological maturation.[10,38,63,70]

Tabel 2. How Antibiotics Affected Gut microbiome in Children

Authors	Study title	Design Study	Populations	Results
Samarra A et al. 2024.[49]	Unravelling the evolutionary dynamics of antibiotic resistance genes in the infant gut microbiota during the first four months of life	Cohort Study	72 infants during their first months of life (7 days, 1, 2, and 4 months)	An increase in antibiotic resistance was observed over the first four months of life, with delivery mode playing a fundamental role in shaping resistance profiles. Feeding methods were also found to influence the development of the resistome, with significant shifts in resistance gene composition over time and differences based on feeding type.
Trosvik P et al. 2024.[48]	Antibiotic resistance gene dynamics in the commensal infant gut microbiome over the first year of life	Longitudinal Cohort	12 infants over 1 year	Revealed high carriage of ARGs and pathobionts in the infant gut microbiome, with implications for horizontal gene transfer and long-term health outcomes.
Li x et al. 2024.[50]	Co-localization of antibiotic resistance genes is widespread in the infant gut microbiome and associates with an immature gut microbial composition	Cohort study	662 Danish children	Found that co-localization of ARGs with other resistance and virulence genes is common in the early gut microbiome and is associated with gut bacteria indicative of low maturity, suggesting potential risks for health.
Schwartz, Drew J et al. 2023.[72]	Effect of amoxicillin on the gut microbiome of children with severe acute malnutrition in Madarounfa, Niger: a retrospective metagenomic analysis of a placebo-controlled trial	Randomized controlled trial (RCT)	301 children aged 6 to 59 months.	Amoxicillin increased gut antibiotic resistance gene abundance to 6044 reads per kilobase million (95% CI 4704–7384) at week 1, up from 4800 (3391–6208) at baseline, which returned to baseline 3 weeks later
Doan T et al.2024.[73]	Gut Microbiome Diversity and Antimicrobial Resistance After a Single Dose of Oral Azithromycin in Children: A Randomized Placebo-Controlled Trial.	randomized, placebo-controlled trial	450 children	A single dose of oral azithromycin in children aged 8 days to 59 months transiently decreased gut microbiome diversity and increased macrolide resistance determinants at 2 weeks, but these effects were not detectable at 6 months, indicating temporary disruptions.
Lebeaux, R et al. 2022.[55]	Impact of antibiotics on off-target infant gut microbiota and resistance genes in cohort studies.	Cohort study	Over 200 infants	Infants who attended day care and were exposed to antibiotics within the first year had a higher abundance of *Escherichia coli* and antibiotic resistance genes

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Emerging information about dietary ARG exposure underscores the imperative to integrate antibiotic resistance issues into food safety and public health policy. Implementing more stringent rules on antibiotic usage in animal agriculture, particularly the prohibition of non-therapeutic drugs, is crucial for diminishing the prevalence of antibiotic-resistant genes in livestock products. Children dietary guidelines and clinical practices should consider the possible dangers linked to ARGs in frequently ingested foods, particularly during early developmental phases when the gut microbiota is still establishing. The food business must adopt antibiotic-free production processes and ensure hygienic processing to reduce ARG contamination. Addressing dietary ARG exposure underscores the significance of a One Health approach that amalgamates human, animal, and environmental health interventions. Ultimately, these findings underscore the necessity for concerted, multisectoral efforts to avert ARG spread throughout the food chain and safeguard children's health from the subsequent consequences of antibiotic resistance.

RESEARCH GAPS IDENTIFIED

Acknowledged risks of ARGs propagating through the food chain are becoming more recognized. However, their associated risks remain inadequately researched. Causal relationships are rarely longitudinal, as dietary ARG intake's influence on gut microbiome health in children is unexplained. There are gaps in monitoring and reporting ARGs in food products, as no standardized protocols exist for livestock-derived food products, which enables fragmented surveillance. Existing studies tend to utilize in vitro and animal testing. There is a distinct lack of human in vivo studies evaluating the persistence and transfer of ARGs within the gastrointestinal tract after ingestion. Moreover, resistome research that analyses dietary patterns among children is rather underdeveloped, impeding the understanding of daily food consumption and ARG exposure. Dietary trials aiming to lower ARGs in diet or through dietary regulation are also lacking in literature, signifying a gap in applied research.

CONCLUSION

Consuming livestock products that contain antimicrobial resistance genes (ARGs) provides a clear pathway for these genes to reach the gut microbiota of children. As discussed in this review, antibiotics used in livestock production can enrich ARGs within animal microbiota, and these genes may persist in meat, milk, and eggs even after processing. ARGs carried by bacteria or extracellular DNA can survive the digestive tract and interact with the gut microbiome through well-recognized mechanisms of horizontal gene transfer, including conjugation, transformation, and transduction.

Children—especially infants—are more vulnerable to these exposures because their gut microbiota is still developing and their immune and metabolic systems are not yet mature. These conditions create an environment in which ARGs can be more easily integrated into the gut ecosystem, potentially influencing future responses to infection and antibiotic treatment. The long-term implications include a higher likelihood of resistant infections, reduced treatment effectiveness, and disturbances in immune or metabolic development.

Taken together, the evidence highlights the importance of reducing dietary exposure to ARGs and strengthening oversight within the food production chain. Addressing this issue requires cooperation across public health, agriculture, and clinical sectors. Future research should clarify levels of exposure, track how ARGs move within the human body, and evaluate how dietary or policy changes might reduce these risks.

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