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A Review of Pediatric *Campylobacter* Infections: Epidemiology, Pathogenesis, and Antimicrobial Resistance Patterns .

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Abstract

Diarrheal diseases continue to be a leading cause of illness and death among children under five years of age, particularly in low- and middle-income countries (LMICs), where inadequate water, sanitation, and hygiene (WASH), food contamination, and limited access to healthcare perpetuate a high burden of disease. Among bacterial pathogens, *Campylobacter jejuni* and *Campylobacter coli* are the major causes of pediatric gastroenteritis, most commonly transmitted through contaminated poultry, water sources, and contact with animals. This review summarizes recent evidence on pediatric *Campylobacter* infections, focusing on epidemiology, pathogenesis, and antimicrobial resistance (AMR), with an emphasis on LMICs and the specific context of Yemen. Infection rates are highest among children aged 6–24 months, coinciding with weaning practices, increased environmental exposure, and immature immune defense. Key virulence factors, including flagellar motility (FlaA), adhesion proteins (CadF, FlpA), invasion antigens (Cia), and cytolethal distending toxin (CDT), enable intestinal colonization, epithelial invasion, inflammation, and impaired nutrient absorption, contributing to growth faltering and environmental enteric dysfunction. Rising resistance to first-line therapies, particularly macrolides and fluoroquinolones, driven by mutations in *gyrA* and 23S rRNA, *erm(B)*-mediated methylation, and CmeABC efflux mechanisms, presents significant treatment challenges in pediatric populations. The interconnected roles of human, animal, and environmental reservoirs underscore the need for a One Health approach that integrates WASH improvements, food safety measures, and antimicrobial stewardship. Enhancing diagnostic capacity, AMR surveillance, and vaccine development while addressing vulnerabilities is critical for reducing *Campylobacter*-associated morbidity and improving child health outcomes in LMICs.

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Introduction

Diarrheal diseases remain among the leading causes of morbidity and mortality in children under five years of age worldwide, with the greatest burden concentrated in low- and middle-income countries (LMICs), where access to clean water, sanitation, and healthcare is limited [1–3]. The persistence of diarrheal illness in these settings reflects the intersection of infection, malnutrition, and poverty, which together hinder global progress despite advances such as oral rehydration therapy and vaccination [4]. Within this burden, bacterial, viral, and parasitic pathogens act synergistically to cause disease, with *Campylobacter jejuni* and *Campylobacter coli* recognized as the leading bacterial causes of enteric infections in young children [5, 6].

Recent global estimates have identified *Campylobacter* as one of the top contributors to pediatric diarrhea, along with *Shigella* and enterotoxigenic *Escherichia coli*

(ETEC) [1]. In LMICs, high transmission rates are driven by unsafe food handling, contaminated water, poor sanitation, and close contact with animals [7]. The combination of environmental and nutritional vulnerabilities increases both the risk of infection and the likelihood of growth faltering and long-term health effects [8, 9].

Epidemiological studies have demonstrated substantial heterogeneity in circulating *Campylobacter* lineages across regions, with evidence of zoonotic transmission from poultry and livestock as the dominant reservoirs [10, 11]. Children in LMICs are exposed early and repeatedly, often developing asymptomatic carriage or mixed infections that complicate diagnosis and clinical management [12, 13]. The highest incidence occurs between 6 and 24 months of age, a critical developmental period when dietary transitions, incomplete immunity, and environmental exposure converge to increase vulnerability [11, 14].

Beyond acute diarrhea, *Campylobacter* infection contributes to chronic nutritional consequences through intestinal inflammation, altered microbiota, and impaired nutrient absorption, forming part of the broader syndrome of environmental enteric dysfunction (EED) [8]. Repeated infections are associated with stunting, poor weight gain, and reduced cognitive development in early life, illustrating the wider developmental impact of enteric pathogens in resource-limited settings [9, 11].

Of growing concern is the emergence of antimicrobial resistance (AMR) in *Campylobacter* species, particularly to fluoroquinolones and macrolides, which are the mainstays of treatment for severe or persistent cases [10, 15]. Resistant strains circulate in both human and animal populations, reflecting antibiotic misuse and unregulated veterinary practices that are common in LMICs [16]. This trend threatens effective therapy and highlights the urgency of integrating surveillance and stewardship across all sectors.

In Yemen and similar LMICs, overlapping crises—conflict, water scarcity, poor sanitation, and food insecurity—create ideal conditions for *Campylobacter* transmission and the emergence [17, 18]. Limited diagnostic infrastructure and surveillance further obscure the true burden of the disease. Addressing these gaps requires a One Health approach that links human, animal, and environmental health to guide interventions and research.

This review synthesizes recent evidence (2019–2025) on *Campylobacter* infections in pediatric populations, focusing on epidemiology, pathogenesis, and antimicrobial resistance in LMIC, with special reference to Yemen. The aim of this study was to provide an updated, integrative framework that supports improved prevention, diagnosis, and management of *Campylobacter*-associated diarrhea in young children.

1.Epidemiology and Transmission of Pediatric *Campylobacter* Infections

Campylobacter is among the leading bacterial causes of gastroenteritis worldwide, with the greatest burden observed in low- and middle-income countries (LMICs). In these settings, inadequate water, sanitation, and hygiene (WASH) infrastructure, widespread malnutrition, and limited access to healthcare substantially increase children's vulnerability to infections [1, 2]. *Campylobacter jejuni* and *Campylobacter coli* are frequently detected in pediatric diarrheal samples and commonly occur alongside other enteric pathogens, including *Shigella*, enterotoxigenic *Escherichia coli* (ETEC), and rotaviruses. [12, 13]. Co-infections complicate attribution and can exacerbate dehydration and inflammatory responses, especially in malnourished children.

The prevalence of *Campylobacter* infection peaks between 6 and 24 months of age, coinciding with weaning, dietary transitions, and exposure to contaminated food and water [11, 14]. Longitudinal studies in LMIC cohorts have linked early life exposure to growth faltering, stunting, and environmental enteric dysfunction (EED), reflecting the chronic effects of recurrent enteric infections [8].

Zoonotic transmission plays a central role in *Campylobacter* epidemiology in humans. Poultry remains the dominant reservoir, with contamination occurring at multiple points along the food chain, from farms and slaughterhouses to household preparation [10, 19]. In addition to poultry, other domestic animals, including cattle and small ruminants, are important reservoirs [20, 21]. In LMICs, informal food markets, inadequate refrigeration, and cross-contamination during food handling amplify the risk of exposure [22].

Environmental and waterborne transmission further contributes to the infection. Studies in Ethiopia, Bangladesh, and Kenya have shown strong associations between proximity to poultry, contaminated water, and infant *Campylobacter* carriage [14, 23]. Poor sanitation infrastructure facilitates fecal-oral spread and sustains the endemic circulation of the virus within communities. Seasonal variation, typically with a higher incidence during warmer, wetter months, reflects the influence of temperature and rainfall on bacterial survival and transmission [24].

Regionally, data from the Middle East and North Africa (MENA) reveal substantial but underreported *Campylobacter* activities. In Lebanon and neighboring countries, pediatric infections have been linked to poultry consumption, unpasteurized milk consumption, and direct animal contact [25]. Yemen shares similar ecological and socio-economic risk factors, including disrupted WASH infrastructure, food insecurity, and high dependence on informal poultry supply chains [17, 18]. Although formal surveillance is limited, evidence suggests a significant but unquantified *Campylobacter* burden among Yemeni children, with a potential overlap between diarrheal outbreaks and broader zoonotic transmission cycles.

Table[1]: Prevalence and Distribution of Campylobacter Species among all ages and Pediatric Patients Across Different Countries.

Country	Sample size	Age	Prevalence (Positive)	Species/ (%)	Detection Procedure	Year	Reference
Burkina Faso	324	< 5	25%	*C. jejuni* 51% *C. coli* 10%	PCR	2024	[26]
Ethiopia	235	2–60 months	–	*Campylobacter*	Cultural (Campylobacter agar)	2022	[27]
Ethiopia	99	–	10.1%	*C. jejuni* 50% *C. coli* 10%	Cultural, PCR (MCCDA)	2021	[28]
Ethiopia	214	< 12	6.5%	*C. jejuni* 85.7% *C. coli* 14.3%	Cultural (Kirby Bauer, MCCDA)	2024	[29]
Ethiopia	428	< 5	7.0%	*C. jejuni* 73.3% *C. coli* 26.7%	Cultural (MCCDA)	2024	[30]
Ethiopia	303	< 5	–	*C. jejuni* 19% *C. coli* 1%	Cultural, PCR	2024	[31]
India	55	12–60 months	10.9%	*C. jejuni*	Cultural, PCR	2024	[32]
Kenya	540	6–24 months	4.8%	*C. coli* 26.9% *C. jejuni* 80.8%	Cultural, PCR	2023	[23]
Rwanda	385	< 5	7.0%	*C. jejuni* 92.6% *C. coli* 7.4%	Cultural, PCR (MCCDA)	2025	[33]
South Africa	564	0–24 months	13.2%	*C. jejuni* 90.3% *C. coli* 9.7%	ELISA, PCR	2022	[34]
Spain	1319	–	57.8%	*C. jejuni* 87.7% *C. coli* 12.3%	Cultural (MCCDA)	2023	[35]
Taiwan	64	25 months–	1.3%	*Campylobacter spp*	Cultural, PCR	2023	[36]
Turkey	500	–	–	*C. jejuni* 67.8% *C. coli* 29% *C. concisus* 3.2%	Filtration, PCR (Butzler agar, MCCDA)	2023	[37]
Yemen	200	One month to five years	–	*C. jejuni* 25.9% *C. coli* 22.2%	–	2013	[38]

The epidemiological landscape across LMICs reflects a convergence of foodborne, zoonotic, and environmental pathways driven by poverty and weak sanitation systems. Effective control requires integrated One Health interventions addressing the poultry production chain, water safety, and antimicrobial stewardship to mitigate *Campylobacter* transmission and its health consequences in children in Yemen and similar settings.

2. Pathogenesis and Mechanisms of Disease

Key Pathogens and Virulence Factors

C. jejani and *C. co* possess multiple virulence factors that enable colonization, invasion, and immune evasion within the intestinal tract (Figure 1).

Motility and chemotaxis, mediated by the polar flagellum and structural protein FlaA, are essential for movement through intestinal mucus and attachment to epithelial cells [39, 40]. Adherence is facilitated by the fibronectin-binding proteins CadF and FlpA, which anchor the bacterium to host cells and activate intracellular signaling pathways that support invasion [41]. Once attached, *Campylobacter* employs invasion antigens (Cia proteins), particularly CiaB, to disrupt cytoskeletal organization and penetrate epithelial barriers, enabling its intracellular survival [42].

Cytolethal distending toxin (CDT), composed of CdtA, CdtB, and CdtC, is a key virulence determinant. CdtB exhibits DNase activity that induces DNA strand breaks, cell cycle arrest, and apoptosis, leading to epithelial cell damage and impaired intestinal function [43, 44]. CDT also triggers inflammatory responses via the cGAS–STING pathway, amplifying mucosal inflammation [45]. Collectively, these virulence factors promote epithelial injury, barrier dysfunction, and nutrient malabsorption, which are central to the pathophysiology of *Campylobacter*-associated diarrhea.

Host–Pathogen Interaction in Children

Infants and toddlers possess immature intestinal and immune defenses, characterized by reduced mucosal IgA and developing microbiota, which render them highly

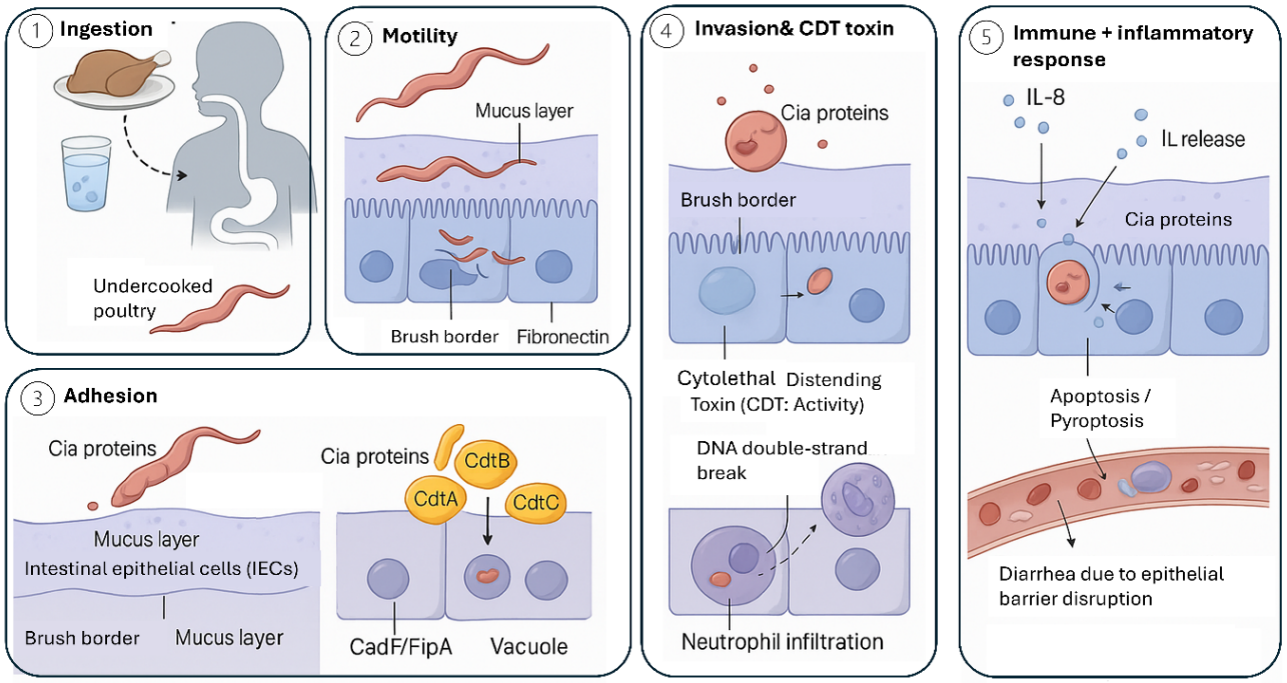

Figure 1. Mechanisms of Campylobacter Pathogenesis and Host Intestinal Response.

susceptible to infection [11]. The 6–24-month age window coincides with peak exposure and limited immune maturity, facilitating colonization and the development of symptomatic disease. Repeated infections during this period are linked to environmental enteric dysfunction (EED), chronic inflammation, and impaired nutrient absorption, which contribute to stunting and poor growth outcomes [8, 14].

Histopathological evidence from pediatric studies demonstrates villous atrophy, epithelial erosion, and neutrophilic infiltration, all driven by cytokine responses, particularly IL-8 and TNF-α, mediated by NF-κB activation [11, 46]. CDT-induced DNA damage further intensifies inflammation, causing mucosal permeability and diarrhea. Co-infection with other enteric pathogens, common in LMICs, can potentiate these inflammatory cascades and exacerbate malnutrition [13].

Context in LMICs and Yemen

In low- and middle-income country (LMIC) settings, particularly Yemen, nutritional deficiencies, inadequate sanitation, and repeated exposure to contaminated food and water exacerbate the pathogenic effects of enteric infections. Immature immune systems, frequent reinfections, and increasing antimicrobial resistance interact to create a persistent cycle of diarrheal disease, chronic intestinal inflammation, and growth failure in young children. Elucidating these host–pathogen interactions is essential for developing context-specific interventions to improve pediatric health outcomes in Yemen and similar settings.

The widespread collapse of water and sanitation infrastructure, large-scale population displacement, disruption of routine immunization programs, and deterioration of primary healthcare services have contributed to the recurrent outbreaks of communicable diseases nationwide [47–51]. Concurrent with this, foodborne illnessescaused by pathogens such as Campylobacter, Salmonella, Shigella, and pathogenic Escherichia colihave risen substantially due to unsafe food handling, inadequate cold-chain systems, and increased reliance on informal food markets amid economic instability [52–54]. Pervasive childhood malnutrition further compromises immune defence, increasing susceptibility to infection and disease severity. In parallel, limited diagnostic capacity, shortages of trained laboratory personnel, and restricted access to advanced testing technologies hinder timely detection, surveillance, and outbreak responses. Together, these intersecting challenges substantially amplify the burden of Campylobacter and other enteric pathogensamong the most vulnerable settings globally for infectious and foodborne diseases.

3. Antimicrobial Resistance (AMR) in *Campylobacter*

Current Therapeutic Context

Treatment of pediatric *Campylobacter* infections is generally supportive, with antibiotics reserved for severe or prolonged infection. Macrolides, particularly azithromycin and erythromycin, remain the preferred first-line agents for children owing to their proven effectiveness and favorable safety profiles. In contrast, fluoroquinolone resistance among *Campylobacter* species has increased globally, limiting their clinical utility in many settings [55, 56]. Tetracyclines are rarely used in young children be-

Table[2]: Molecular Mechanisms of Resistance

Antibiotic Class	Main Mechanism	Key Genes/Mutations	Representative References
Fluoroquinolones	Point mutation in DNA gyrase	*gyrA* (T86I)	[59]
Macrolides	Ribosomal modification & methylation	23S *r*RNA (A2058G/A2059G), *erm(B)*	[60]
Tetracyclines	Ribosomal protection proteins	*tet(O)*, *tet(L)*	[61]
Multidrug resistance	Efflux-pump system	*CmeABC* operon	[55, 63]

cause of toxicity concerns; however, resistance to them is also common in animal and environmental isolates [57].

Antimicrobial misuse in human medicine and livestock production contributes substantially to the emergence of resistance, especially in LMICs, where regulations are weak [16]. Consequently, multidrug-resistant (MDR) *Campylobacter* strains have become prevalent, complicating therapy and increasing the risk of treatment failure, prolonged illness duration, and transmission among young children [54, 58].

Macrolide resistance is primarily associated with ribosomal modification and methylation, especially *23S rRNA* (A2058G/A2059G) and *erm(B)*, leading to reduced drug binding and high-level resistance [59]. Fluoroquinolone resistance arises mainly from mutations in *gyrA* (T86I) or methylation mediated by *erm(B)*, both of which block macrolide binding to the ribosome [60]. The CmeABC efflux pump contributes to multidrug resistance by actively exporting diverse antibiotic classes, thereby enhancing survival under antimicrobial pressure [55]. Resistance to tetracyclines, mediated by *tet(O)* and *tet(L)* genes, is widespread in humans, poultry, and environmental isolates [61].

Mobile genetic elements, such as plasmids, genomic islands, and efflux-regulatory mutations, facilitate the horizontal transfer of these determinants, promoting co-resistance across multiple antibiotic classes and amplifying public health risks [62] (Table 2).

Regional and Context

Across Africa and the Middle East, *Campylobacter* isolates from humans and animals frequently display resistance to fluoroquinolones and macrolides, with variable resistance to tetracycline [62, 64]. In Tunisia, plasmid-mediated quinolone resistance (*qnr* genes) has been reported, underscoring the regional spread of novel resistance mechanisms [62].

In Yemen, formal AMR surveillance is lacking; however, contextual evidence suggests that local strains likely harbor the same resistance determinants found regionally: *gyrA* and *23S rRNA* mutations, *CmeABC* efflux, and *tet(O)* genes [16]. The combination of unregulated antibiotic use, poor infection control, and limited diagnostics heightens the risk of MDR *Campylobacter* emergence among children under five years of age, particularly in pediatric populations where diarrhea and malnutrition coexist.

Public Health Implications

Rising *Campylobacter* resistance undermines effective case management, prolongs illness, and increases hospitalization rates in children. Expanding local AMR surveillance, improving diagnostic capacity, and implementing stewardship programs in both the human and veterinary sectors are essential to curb the spread of resistance. Integrating these measures within a One Health framework is critical in fragile contexts such as Yemen, where zoonotic and environmental sources of *Campylobacter* overlap with human disease and limited healthcare infrastructure.

There is also growing interest in natural alternatives, such as plant extracts used as phytogenic feed additives in poultry, to reduce antibiotic use and limit the emergence of antimicrobial-resistant Campylobacter. Many botanical compounds possess antibacterial and immunomodulatory properties that can lower intestinal colonization in poultry, offering a sustainable and low-cost strategy, particularly in settings like Yemen, where antibiotic misuse in agriculture remains common [65, 66].

4. Diagnosis and Laboratory Challenges

Accurate diagnosis of *Campylobacter* infection in children is essential for effective management and antimicrobial resistance (AMR) surveillance. However, it remains challenging in low- and middle-income countries (LMICs) such as Yemen. Diagnostic methods are divided into traditional culture-based techniques and molecular or culture-independent approaches, each with distinct advantages and limitations.

4.1. Limitations of Culture-Based Diagnosis

Conventional stool culture remains the reference method for confirming *Campylobacter* infection and performing antimicrobial susceptibility testing (AST). However, the fastidious growth requirements of the organism, includ-

ing a microaerophilic atmosphere, selective media, and incubation at 42 ℃, limit its recovery in many LMIC laboratories [67]. Factors such as delayed specimen transport, prior antibiotic exposure, and suboptimal incubation conditions often result in false negatives [68]. Culture is also labor-intensive and may miss non-*jejuni/coli* species, leading to an underestimation of the true disease burden [69].

These limitations are particularly critical in pediatric contexts, where diarrheal illness progresses rapidly, and timely treatment decisions are needed. Furthermore, without viable isolates, phenotypic AST cannot be performed, constraining local AMR surveillance and evidence-based therapies [16].

4.2. Advantages and Drawbacks of Molecular and Culture-Independent Diagnostics

Molecular diagnostics, especially multiplex PCR and syndromic gastrointestinal panels, allow rapid and sensitive detection of *Campylobacter* DNA directly from stool samples [70, 71]. These methods can detect infections even when the organisms are non-viable or present in low numbers, offering faster turnaround and improved clinical decision-making. However, they cannot distinguish between viable and dead organisms and may yield false-positive results in cases of asymptomatic carriage [72]. Moreover, molecular panels do not provide phenotypic resistance data, which are essential for guiding empirical therapy in high-AMR settings [69].

To overcome these gaps, integrated workflows that combine molecular screening with selective culture are recommended. Molecular assays enable early detection and case management, whereas culture confirmation allows phenotypic AST, genomic analysis, and long-term surveillance of resistance trends [53, 73]. This dual strategy is particularly valuable in resource-limited environments, as it balances diagnostic sensitivity with the ability to monitor evolving AMR patterns.

4.3. Diagnostic Capacity Gaps in LMICs and Yemen

In Yemen, diagnostic challenges are compounded by conflict-related infrastructure disruption, limited laboratory supplies and a shortage of trained personnel. Most laboratories lack access to molecular panels and reliable microaerophilic culture systems [74, 75]. Consequently, *Campylobacter* infections are frequently underdiagnosed, and AMR patterns remain largely undocumented [16]. Strengthening diagnostic capacity through affordable multiplex PCR panels, regional reference laboratories, and workforce training is critical for effective disease surveillance.

Investing in hybrid diagnostic systems that incorporate both culture and molecular tools, paired with quality assurance and data sharing across human and veterinary sectors, will improve detection accuracy and resistance monitoring. Establishing such integrated approaches under a One Health framework is a key step toward improving pediatric diarrheal disease management and AMR control in Yemen and similar LMIC settings.

5. Prevention and Control Strategies

Effective prevention of pediatric *Campylobacter* infections requires an integrated One Health approach that combines improvements in water, sanitation, and hygiene (WASH), food safety, and antimicrobial stewardship. In LMICs, such as Yemen, where unsafe water, informal food systems, and unregulated antibiotic use overlap, comprehensive interventions across sectors are essential to reduce disease transmission and resistance spread.

5.1. Water, Sanitation, and Hygiene (WASH) Interventions

Safe drinking water, adequate sanitation, and hygiene education are the foundations for preventing enteric infections [76, 77]. Studies consistently show that access to clean water and improved sanitation reduces diarrheal incidence and supports better growth outcomes in young children [78, 79].

Hand hygiene, especially handwashing with soap after animal contact and before food preparation, significantly lowers *Campylobacter* transmission [80, 81]. In Yemen, where the infrastructure is fragile, community-based WASH programs linked to maternal and child health services can strengthen prevention, particularly when coupled with hygiene education and breastfeeding promotion during the first 6 months of life [82].

However, low public awareness of infectious diseases and limited health education further hinder the adoption of essential WASH practices, increasing the risk of enteric and foodborne infections among vulnerable populations [83].

Integrating WASH interventions with nutritional support (*babyWASH* initiatives) addresses both infection prevention and malnutrition, reducing stunting and improving cognitive development. In conflict or crisis settings, deploying mobile or low-cost WASH systems that ensure continuous access to safe water and sanitation is vital [84, 85].

5.2. Food Safety and One Health Measures

As poultry is the primary reservoir for *Campylobacter*, control strategies must target the entire farm-to-table continuum. Critical actions include farm biosecurity, proper slaughterhouse sanitation, cold chain maintenance, and consumer education on safe food handling [19, 21].

Antibiotic misuse in animal husbandry is a major driver of antimicrobial resistance (AMR). Strengthening veterinary oversight, prohibiting non-therapeutic antibiotic use, and promoting poultry vaccination and hygiene can reduce the emergence of resistant strains [24, 62] Coordinated

surveillance of *Campylobacter* in animals, food, and human samples allows early detection of resistant strains and helps evaluate control measures [86].

Implementing One Health surveillance frameworks that link the veterinary, environmental, and public health sectorscan help identify hotspots of transmission and resistance flow. This approach is especially critical in Yemen, where informal poultry markets and limited inspection capacity increase the risk of exposure [16, 18].

5.3. Antimicrobial Stewardship and Public Health Policy

Rational antibiotic use is key to limiting the development of AMR. Stewardship programs should promote evidence-based pediatric treatment protocols, restrict over-the-counter antibiotic sales, and expand public awareness of the dangers of misuse [18].

Integrating stewardship with WASH and food safety initiatives ensures a sustainable reduction in infection risk while preserving antibiotic effectiveness.

In Yemen, establishing national guidelines for diarrheal management, supported by local resistance data, and building AMR surveillance laboratories should be prioritized. Cross-sector collaboration among the ministries of health, agriculture, and the environment is essential to implement and monitor these programs under a unified One Health governance framework [85].

Recent clinical reports from Yemen have highlighted the consequences of delayed diagnosis and limited laboratory capacity in managing severe infections in pediatric populations [87].

6. Future Perspectives and Research Priorities

The growing recognition of *Campylobacter* as a major contributor to pediatric diarrhea and antimicrobial resistance (AMR) underscores the urgent need for coordinated research, diagnostic innovation, and One Health policy action, particularly in LMICs such as Yemen. The following areas represent the most critical priorities for the next decade.

1. Improved Diagnostics and Surveillance

Rapid, affordable, and reliable diagnostics are essential for accurate case management and AMR monitoring. Integrating multiplex PCR–based panels for quick pathogen detection with culture and whole-genome sequencing (WGS) for resistance profiling will enable more precise therapy and surveillance [69, 70].

Developing national diagnostic networks and sentinel laboratories in Yemen can provide region-specific data to guide empirical treatment and strengthen the public health response [16]. Surveillance systems should integrate human, animal, and environmental data to reflect the One Health reality.

2. Vaccine Development

A pediatric *Campylobacter* vaccine remains a self-limited unmet need. Experimental capsule-conjugate and protein-based vaccines targeting virulence factors such as CadF, FlaA, and CDT have shown promise in early studies [56, 88].

Future research should focus on vaccine efficacy in infants (6–24 months), strategies for maternal immunization, and the potential integration of *Campylobacter* vaccination into existing child immunization programs in LMICs.

3. Host–Microbiome Interactions and Nutritional Outcomes

Further research is required to elucidate how repeated *Campylobacter* exposure alters the gut microbiota and contributes to environmental enteric dysfunction (EED) and growth stunting [11, 14].

Longitudinal birth cohort studies could identify microbiome signatures or biomarkers predictive of adverse outcomes, supporting interventions such as targeted probiotics or nutritional supplementation to improve growth and immunity in high-risk populations.

4. Strengthening Antimicrobial Stewardship

Expanding stewardship across the human health, veterinary, and agricultural sectors is crucial for slowing the spread of resistance. This includes enforcing regulations on antibiotic sales, improving prescribing practices, and encouraging alternative control measures, such as vaccination and biosecurity [16, 62].

National AMR frameworks should link stewardship activities with ongoing WASH and food safety programs to create a sustainable, cross-sectoral impact.

5. One Health Integration and Climate-Responsive Policy

Future efforts should institutionalize One Health collaboration by linking clinical data with animal and environmental surveillance. Integrating climate data—temperature, rainfall, and flooding trends—will improve the prediction of seasonal outbreaks and inform adaptive interventions [89, 90].

For Yemen, building integrated systems through partnerships between health, agriculture, and environmental authorities will ensure resilience amid ongoing humanitarian challenges.

7. Conclusion

Campylobacter infections are a major cause of pediatric diarrheal disease and an emerging antimicrobial resistance (AMR) concern, particularly in low- and middle-income countries (LMICs). Recurrent exposure during early childhood contributes not only to acute gastrointestinal illness but also to long-term consequences, including malnutrition, chronic intestinal inflammation, and impaired linear growth.

Effective prevention and control require an integrated One Health strategy that reinforces WASH infrastructure, improves food safety throughout the poultry production and supply chain, promotes responsible antimicrobial use, and enhances diagnostic and surveillance capacities. In parallel, coordinated research on vaccine development, host–microbiome interactions, and AMR mechanisms is essential for informing targeted and sustainable interventions.

By integrating human, animal, and environmental health efforts, comparable LMICs can significantly reduce the burden of *Campylobacter*-associated diseases and improve health outcomes for vulnerable pediatric populations.

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