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Campylobacter jejuni as an emerging abortifacient pathogen in small ruminants: Pathophysiology, placental tropism, economic, zoonotic and One Health implications, prevention and control – A review

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Abstract

Campylobacter jejuni is increasingly being recognized as an emerging abortifacient pathogen in small ruminants, contributing to reproduction losses, reduced flock/herd productivity and significant economic strain in resource-limited production systems. The present review synthesized current information on the pathophysiology of *C. jejuni*-induced abortions, emerging hyper-virulent abortifacient *C. jejuni* strains infecting small ruminants, the transmission dynamics, differential diagnosis and comparison of abortifacient pathogens in small ruminants, clinical impacts, economic, food security, zoonotic and public health significance of *C. jejuni* infections, and the prevention and control strategies for *C. jejuni*-associated abortions in sheep and goats. It highlights the One Health relevance of *C. jejuni*-induced abortions in small ruminants and underscores the importance of accurate differential diagnosis, strengthened surveillance and early detection to distinguish *C. jejuni* from other abortifacient pathogens. Actionable control measures include improved farm biosecurity, sanitation of water and feed sources, strategic isolation of aborting dams, screening of breeding males and proper disposal of aborted foetuses and after-birth materials. Integration of veterinary services with public health awareness will minimize zoonotic risks associated with contaminated environments and animal products. Strengthening herd health management, environmental hygiene and coordinated One Health interventions offers a practical pathway to mitigating reproduction losses and improving resilience in small ruminant production systems.

Keywords: Small ruminants (sheep and goats); *Campylobacter jejuni;* Abortion; Reproduction losses; Placental infection.

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Introduction

Small ruminants, particularly sheep and goats, play a foundational role in rural agricultural systems across sub-Saharan Africa and other resource-constrained regions. They represent a critical reservoir of household wealth, nutritional security and socio-economic stability. In semi-arid and marginal areas where crop farming is unreliable and cattle production is relatively costly, sheep and goats offer a sustainable, fast-return livestock option. They supply meat, milk and income, while also contributing manure for crop production (Baltenweck *et al*., 2020). In many rural African households, small ruminant-derived protein constitutes one of the few available sources of high-quality dietary amino acids, particularly for children and pregnant women during festive periods (Muema *et al*., 2023). Consequently, reproductive efficiency is central to the sustainability of these systems.

Infectious abortion is one of the most devastating constraints to small ruminant production. Reproduction losses reduce flock replacement rate, delay genetic progress, diminish milk availability and weaken household income streams (Choudhury *et al*., 2019). Although classical abortifacient pathogens such as *Brucella melitensis*, *Chlamydia abortus* and *Toxoplasma gondii* have long been recognised (Ekere *et al*., 2018), increasing evidence indicates that *Campylobacter jejuni* has emerged as a significant and sometimes dominant cause of ovine and caprine abortion (Sahin *et al*., 2008; 2012; 2017). Historically regarded primarily as an enteric pathogen and leading cause of zoonotic gastroenteritis, *C. jejuni* has reportedly demonstrated a distinct capacity for systemic dissemination and placental invasion in pregnant small ruminants (Veronese and Dodi, 2024).

The emergence of hyper-virulent clones associated with abortion outbreaks has heightened concern within veterinary practice (Sahin *et al.,* 2008; 2017). These strains exhibit enhanced expression of invasion-associated genes, toxin production and immune evasion mechanisms that facilitate colonisation of the gravid uterus (Sahin *et al*., 2008; 2012; 2017). Their pathogenic behaviour in late gestation reflects complex host-pathogen interactions involving hormonal modulation of immunity, increased uteroplacental blood flow and bacterial adaptation to placental microenvironments (Janssen *et al.,* 2008; Elmi *et al.,* 2021). This reproductive tropism distinguishes abortigenic strains from commensal intestinal isolates.

The consequences of *C. jejuni*-induced abortion extend beyond individual flock/herd losses. In under-resourced rural systems, abortion storms can destabilise household economies and reduce animal protein availability in communities already burdened by malnutrition. Moreover, the zoonotic nature of *C. jejuni* creates an interface between livestock health and public health, particularly where informal slaughter and shared water sources are common. This review critically examined *Campylobacter jejuni* as an emerging abortifacient pathogen in small ruminants, with emphasis on virulence determinants, molecular pathophysiology of abortion, economic and food security implications, zoonotic significance and evidence-based control strategies appropriate for rural production systems.

Bacterial biology and virulence determinants linked to abortion

The pathogenicity of *Campylobacter jejuni* in small ruminant abortion is reportedly driven by a coordinated network of virulence determinants that facilitate intestinal colonisation, systemic dissemination and placental destruction (Omole *et al.,* 2024). Although *C. jejuni* is frequently a commensal of the gastrointestinal tract, abortion-associated strains demonstrate enhanced virulence gene expression profiles that distinguish them from non-pathogenic isolates (Tikhomirova *et al*., 2024).

Motility mediated by bipolar flagella encoded by *flaA* and *flaB* is fundamental to early colonisation. Beyond propulsion, the flagellar apparatus functions as a secretion system for *C. jejuni* invasion antigens such as *ciaB* (Gabbert *et al*., 2023). This dual functionality enables the organism to penetrate intestinal epithelial cells. In pregnant ewes and does, efficient epithelial translocation increases the likelihood of transient bacteraemia, which is a prerequisite for placental localisation. Adhesion is mediated primarily by fibronectin-binding proteins, notably *CadF* and *FlpA* (Larson *et al*., 2013). These proteins anchor the bacterium to epithelial surfaces, facilitating cytoskeletal rearrangement and internalisation (Larson *et al*., 2013). Abortion-associated isolates reportedly frequently exhibit upregulated *cadF* expression, enhancing placental tissue tropism. Once internalised, *ciaB*-dependent invasion disrupts epithelial tight junctions and promotes systemic spread. The cytolethal distending toxin (CDT) encoded by *cdtA*, *cdtB* and *cdtC* plays a central role in reproductive pathology. The CDT-complex induces DNA double-strand breaks and G2/M cell cycle arrest in trophoblasts, leading to apoptosis and placental necrosis (Tremblay *et al.,* 2021). Placental tissue is particularly susceptible due to rapid cellular turnover and high vascular activity during late gestation. Consequently, CDT-mediated damage directly compromises foetal oxygen and nutrient exchange. Lipooligosaccharide structures contribute to immune evasion and serum resistance. Molecular mimicry reduces complement-mediated killing, enabling survival during bacteraemia. Capsule-associated genes further enhance environmental persistence, promoting transmission during abortion events (Sahin *et al*., 2017). The principal virulence determinants implicated in ovine and caprine abortion are summarised in Table 1. Their coordinated expression underlies the transition from intestinal coloniser to systemic abortifacient pathogen.

Table 1. Key virulence factors of *Campylobacter jejuni* and their roles in abortion pathogenesis.

Virulence determinant	Gene(s)	Mechanistic role	Contribution to abortion	References
Flagella.	*flaA, flab.*	Motility and secretion of invasion proteins.	Enables epithelial penetration and systemic spread.	Guerry *et al*., 2006.
Adhesins.	*cadF, flpA.*	Fibronectin binding and colonisation.	Facilitates placental tissue attachment.	Konkel *et al*., 2004; 2005.
Invasion antigens.	*ciaB.*	Host cell internalisation.	Promotes bacteraemia and placental invasion.	Konkel *et al*., 2004; 2005.
Cytolethal distending toxin.	*cdtABC.*	DNA damage and apoptosis.	Induces trophoblast necrosis and placentitis.	Louwen *et al*., 2005.
Lipooligosaccharide.	*LOS* genes.	Immune evasion and serum resistance.	Sustains survival in bloodstream.	Parker *et al*., 2005.
Capsule.	*CPS* genes.	Environmental persistence.	Enhances outbreak transmission.	Guerry *et al*., 2006.

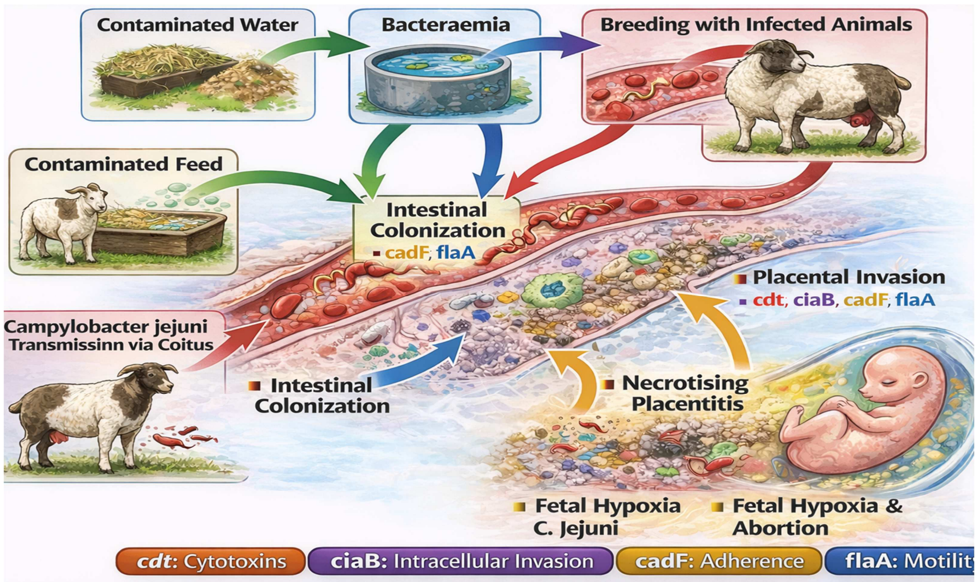

Figure 1. Sequential pathophysiology of *Campylobacter* jejuni-induced abortion in small ruminants. Primary infection sources include ingestion of contaminated feed or water and mating with infected males. Bacteria colonise the intestine, translocate into the bloodstream, invade the placenta, induce necrotising placentitis, and compromise foetal oxygenation, with virulence factors (*cdt, ciaB, cadF, flaA*) mediating key pathogenic processes. Progressive placental damage results in foetal hypoxia, death and consequent expulsion (abortion).

Pathophysiology of *Campylobacter*-induced abortion

The pathophysiology of abortion caused by *C. jejuni* in small ruminants and the progression of the infection from environmental and breeding sources to placental invasion and foetal compromise is illustrated in Figure 1.

Infection begins with ingestion of contaminated feed or water. Flagella motility enables the organism to navigate intestinal mucus and adhere to epithelial cells via *CadF*-mediated fibronectin binding (Haiko and Westerlund-Wikström, 2013). Although gastrointestinal signs may be absent, sub-clinical mucosal invasion occurs. Following epithelial penetration, *ciaB*-mediated internalisation facilitates translocation across the intestinal barrier. Survival in the bloodstream is supported by LOS-mediated complement resistance (Omole *et al*., 2024). In late gestation, increased uterine perfusion and local immunomodulation favour bacterial localisation in placental tissues. The gravid uterus represents a unique immunological niche characterised by partial immune tolerance to protect the foetus; this tolerance may inadvertently facilitate bacterial colonisation (Hussain *et al*., 2022).

Upon reaching the placenta, the bacteria adhere to trophoblasts and proliferate within chorionic tissues. The CDT production induces apoptosis and DNA fragmentation in placental cells, resulting in necrotising placentitis (Sharp *et al*., 2010). Histopathologically, lesions are reportedly characterised by neutrophilic infiltration, vasculitis, thrombosis and fibrin deposition (Sharp *et al*., 2010). Vascular compromise restricts foetal oxygenation, leading to hypoxia and intrauterine death. Foetal infection may occur secondary to placental breakdown, leading to septicaemia in utero. Therefore, abortion typically occurs in the final trimester when placental demand for oxygen and nutrients peaks (Sharp *et al*., 2010). The release of heavily contaminated placental tissues amplifies environmental bacterial load, promoting rapid flock-level transmission. The mechanistic cascade from colonisation to abortion underscores the importance of virulence gene expression in disease severity. Hyper-virulent strains capable of robust CDT production and enhanced invasion are therefore more likely to trigger abortion storms, whereas low-virulence strains may remain confined to the intestine (Sharp *et al*., 2010). The mechanistic interactions between *C. jejuni* virulence factors and the maternal-foetal interface, including trophoblast invasion, apoptosis, and placental necrosis leading to foetal compromise, are illustrated in Figure 2.

Host immunological response and pregnancy-associated susceptibility

Pregnancy represents a unique immunological state characterised by finely regulated immune tolerance that protects the semi-allogeneic foetus. In small ruminants, this physiological modulation may inadvertently create susceptibility to systemic pathogens such as *Campylobacter jejuni*. Successful gestation involves a shift toward anti-inflammatory and regulatory immune responses at the maternal-foetal interface, particularly during mid to late gestation (Satue *et al*., 2025). While this modulation prevents foetal rejection, it may reduce the efficiency of bacterial clearance (Satue *et al*., 2025).

Intestinal colonisation by *C. jejuni* typically induces innate immune activation involving Toll-like receptor signalling and pro-inflammatory cytokine production. However, during pregnancy, systemic inflammatory responses are tightly controlled. Reduced Th1-type responses may impair intracellular bacterial elimination, facilitating transient bacteraemia (Otto *et al*., 2012). Once bacteria localise within the placenta, local immune activation can paradoxically contribute to tissue damage (Hoo *et al.,* 2020).

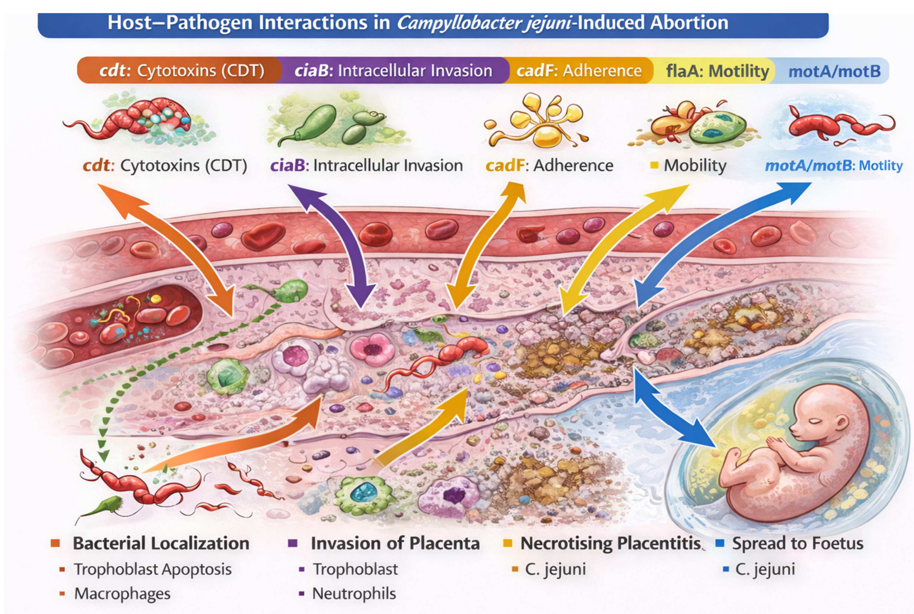

Figure 2. Host–pathogen interactions in *Campylobacter jejuni*–induced abortion in small ruminants. The diagram illustrates placental colonisation following systemic spread, highlighting adhesion (CadF, FlaA), invasion (CiaB) and cytotoxicity (CDT) as key virulence mechanisms causing trophoblast apoptosis, inflammatory necrotising placentitis and vascular compromise.

Placental infection reportedly stimulates recruitment of neutrophils and macrophages, leading to production of tumour necrosis factor-α, interleukin-1β and other pro-inflammatory mediators (Hu *et al.,* 2006). Although intended to control infection, excessive cytokine release promotes vascular inflammation, thrombosis and disruption of the maternal-foetal interface. Cytolethal distending toxin-mediated apoptosis further amplifies placental injury, and the combined effect of bacterial cytotoxicity and host inflammatory response results in compromised oxygen and nutrient exchange (Jinadasa *et al*., 2022).

Late gestation appears particularly vulnerable due to increased uteroplacental blood flow and metabolic demand. The heightened vascularity may enhance bacterial deposition, while elevated progesterone levels contribute to systemic immunomodulation (Motomura *et al*., 2023). Nutritional deficiencies common in under-resourced systems may further impair immune competence. Understanding the interplay between bacterial virulence factors and pregnancy-associated immune modulation provides mechanistic insight into why *C. jejuni* preferentially induces late-term abortion. It also underscores the importance of supporting maternal immunity through optimal nutrition and stress reduction as part of comprehensive prevention strategies.

Emerging *Campylobacter jejuni* hyper-virulent clones

Molecular investigations have identified distinct hyper-virulent clones of *Campylobacter jejuni* associated with ovine abortion outbreaks (Sahin *et al.,* 2008; 2012; 2017). These strains differ genetically and phenotypically from enteric isolates typically recovered from asymptomatic carriers. Whole-genome sequencing has revealed enhanced expression of invasion-associated genes, increased cytolethal distending toxin production and adaptive mutations that favour systemic dissemination (Ghielmetti *et al*., 2023). Representative hyper-virulent *C. jejuni* clones, their virulence gene profiles, placental pathology and epidemiological impact are summarised in Table 2, illustrating the molecular determinants that contribute to abortion risk in small ruminants.

Hyper-virulent clones often demonstrate upregulated *ciaB* expression, promoting efficient epithelial translocation and higher rates of bacteraemia. Elevated *cdtABC* transcription results in intensified trophoblast apoptosis and more severe necrotising placentitis. Certain clones also exhibit genetic variations in capsular polysaccharide synthesis pathways that may enhance environmental survival and transmission efficiency during abortion storms (Rosario *et al*., 2025).

Comparative genomic analyses have shown clustering of abortion-associated strains within specific sequence types, suggesting clonal expansion in susceptible small ruminant populations (An *et al*., 2025). These clones may possess enhanced iron acquisition systems and oxidative stress tolerance, facilitating persistence within inflamed placental tissues. Some abortion-associated isolates also demonstrate increased resistance to fluoroquinolones mediated by mutations in the *gyrA* gene, complicating therapeutic interventions (Quiñones *et al*., 2008; An *et al*., 2025)

The emergence of hyper-virulent clones raises important animal and human health concerns. Their enhanced reproductive tropism may increase abortion incidence, while their zoonotic potential remains intact. Molecular surveillance and strain typing are therefore critical components of outbreak investigation. Understanding the evolutionary drivers behind clonal expansion may inform vaccine antigen selection and targeted control strategies. Continued genomic monitoring is essential to detect shifts in virulence profiles.

Table 2. Emerging hyper-virulent *Campylobacter jejuni* clones identified in small ruminants, including key virulence genes, associated placental pathology, epidemiological significance, and antimicrobial resistance profiles.

Clone / Sequence Type	Key Virulence Genes	Placental Pathology	Epidemiological Significance	Antimicrobial Resistance	References
ST-8	*cdtABC, ciaB, cadF, flaA.*	Severe necrotising placentitis with neutrophilic infiltration; foetal hypoxia.	Frequently associated with late-term abortion storms in sheep flocks.	Occasional fluoroquinolone resistance (*gyrA* mutation).	Sahin *et al*., 2012.
ST-21	*cdtABC, ciaB, LOS biosynthesis genes.*	Acute placentitis, chorionic oedema, moderate foetal compromise.	Outbreaks reported in mixed sheep–goat herds; high environmental persistence.	Susceptible to macrolides; variable quinolone resistance.	Sheppard *et al*., 2006; Sahin *et al*., 2012.
ST-50	*cdtABC, cadF, flaA, capsule genes.*	Necrotising and suppurative placentitis; weak neonates.	Linked to repeated abortions in intensive systems; enhanced colonisation of late-gestation dams.	Emerging macrolide resistance; enhanced biofilm formation aids survival.	Dasti *et al*., 2010; Sahin *et al*., 2012.
ST-353	*ciaB, LOS modification genes, flagella genes.*	Placental thrombosis, trophoblast apoptosis, foetal death.	Identified in multiple geographic regions; high virulence index in experimental infection.	Multi-drug resistance rare but increasing.	Parker *et al*., 2006; Sahin *et al*., 2012.
ST-257	*cdtABC, cadF, ciaB, iron acquisition genes.*	Extensive placental necrosis; foetal mummification.	Strongly associated with late gestation abortions and rapid flock-level transmission.	Low-level resistance to tetracyclines documented.	Hänninen *et al*., 2013; Sahin *et al*., 2012.

Differential diagnosis and comparison of abortifacient pathogens in small ruminants

Abortion in small ruminants is a multi-factorial syndrome with diverse infectious and non-infectious aetiologies. Accurate attribution of reproductive loss to *Campylobacter jejuni* requires careful differentiation from other major abortifacient pathogens that produce overlapping clinical and pathological features. Failure to establish a definitive diagnosis may result in inappropriate control measures and continued flock-level transmission.

Among bacterial causes, *Chlamydia abortus* remains one of the most common agents of enzootic abortion in ewes. It characteristically produces thickened, leathery placentas with intercotyledonary necrosis, whereas *C. jejuni*-associated abortion more frequently results in necrotising placentitis with oedema and suppurative inflammation. *Listeria monocytogenes* may induce septicaemia and multifocal hepatic necrosis in the foetus, often accompanied by maternal neurological signs. *Salmonella* spp. can produce enteritis and septicaemia with foetal autolysis. *Brucella melitensis*, of significant zoonotic concern, is associated with granulomatous placentitis and chronic reproductive loss (Khairullah *et al*., 2024).

Protozoal pathogens also complicate the diagnostic landscape. *Toxoplasma gondii* typically produces white necrotic foci within cotyledons, while *Neospora caninum* may cause abortion with minimal gross placental change but detectable foetal encephalitis (Buxton, 1988; Shaapan, 2016). Viral agents such as border disease virus can induce abortion, stillbirth or weak neonates accompanied by congenital abnormalities (Nettleton *et al*., 1988; Broaddus *et al*., 2009).

The distinguishing features of major abortifacient pathogens in small ruminants are summarised in Table 3, highlighting key lesions, transmission routes and differentiating characteristics relative to *C. jejuni*.

Definitive diagnosis of *C. jejuni*-associated abortion relies on isolation or molecular detection of the organism from placental tissues, abomasal contents or foetal liver. Histopathology demonstrating acute necrotising placentitis with neutrophilic infiltration supports bacterial involvement but is not pathognomonic (Orsaria *et al*., 2021). Polymerase chain reaction assays targeting species-specific genes such as *hipO* provide rapid confirmation, while detection of virulence genes including *cdtB* and *ciaB* strengthens causal inference by linking strain pathogenicity to placental damage (Njoga *et al*., 2023).

Given the frequent occurrence of mixed infections in communal systems, comprehensive abortion panels incorporating multiplex PCR and bacteriological culture are recommended. Integrating molecular diagnostics with gross pathology, histology and epidemiological context enhances diagnostic accuracy. Such differentiation is essential for targeted intervention, prevention planning and accurate estimation of the true burden of *C. jejuni* as an abortifacient pathogen in small ruminants.

Economic and food security implications

The economic consequences of *Campylobacter jejuni*-induced abortion in small ruminants are particularly severe in under-resourced rural settings where sheep and goats function as liquid assets, savings accounts and nutritional buffers for vulnerable households. In many pastoral and mixed crop–livestock systems, flock growth depends almost entirely on annual lambing and kidding performance. Reproductive loss directly reduces the number of replacement females entering the breeding population, thereby constraining flock expansion and delaying genetic progress. Even modest abortion rates of 10 – 20% can translate into substantial declines in herd growth trajectories, especially where baseline reproductive performance is already limited by nutritional stress and endemic disease (Shorten *et al*., 2021).

Beyond numerical losses, the economic burden includes reduced saleable stock for market, diminished milk yield and disruption of seasonal cash flow. Small ruminant milk, frequently allocated to children and lactating mothers, declines following abortion due to endocrine disturbance, shortened lactation curves and compromised maternal condition. In nutritionally marginal households, this reduction may exacerbate protein-energy malnutrition and micronutrient deficiencies. The nutritional impact is not trivial; animal-source protein from sheep and goats often represents the most accessible and affordable source of high biological value amino acids in rural diets (Shorten *et al*., 2021).

Table 3. Comparative features of key abortifacient pathogens in small ruminants, showing characteristic lesions, main transmission routes, and distinguishing features to aid differential diagnosis of *Campylobacter jejuni*-associated abortion.

Pathogen.	Typical lesions.	Main transmission route.	Distinguishing features compared to *C. jejuni*	References
Campylobacter jejuni	Necrotising placentitis, chorionic oedema, neutrophilic infiltration, foetal hypoxia.	Faecal-oral; contaminated feed/water; direct contact.	Often occurs in late gestation, abortion storms, minimal maternal signs, detectable by PCR of *cdtB* and *ciaB.*	Wu *et al*., 2014; Sahin *et al*., 2015; Nachamkin *et al*., 2019.
Chlamydia abortus	Thickened, leathery intercotyledonary placental necrosis; foetal mummification.	Oral ingestion of contaminated bedding, placenta, or vaginal discharges.	Gradual abortion pattern, older ewes commonly affected, intracellular pathogen detected by PCR or immunohistochemistry.	Longbottom and Coulter, 2003; Essig and Longbottom, 2015;
Toxoplasma gondii	Cotyledonary necrotic foci, focal mineralisation; occasionally autolysed foetuses.	Ingestion of oocysts from contaminated feed or water.	Protozoal cysts visible in placenta; chronic infection may cause early abortion.	Dubey, 2010; Dubey and Jones, 2008.
Listeria monocytogenes	Multifocal necrosis in foetal liver, septicaemia, occasional maternal neurological signs.	Oral ingestion of contaminated silage or feed.	Maternal encephalitis may precede abortion; bacterial culture from foetal liver or placenta confirms diagnosis.	Oevermann *et al*., 2010; WOAH, 2022.
***Salmonella* spp.**	Foetal autolysis, septicaemia, enteritis in dam.	Faecal–oral; contaminated feed/water.	Enteric signs in dam often prominent; isolation from foetal liver, spleen or intestine; serotyping distinguishes species.	Uzzau *et al*., 2000; WOAH, 2022.
Brucella melitensis	Granulomatous placentitis, chronic abortion, orchitis in males.	Direct contact, ingestion, venereal transmission.	Zoonotic significance; serology and culture critical; slower onset than Campylobacter abortion.	Seleem *et al.*, 2010; Godfroid *et al*., 2010.
Neospora caninum	Minimal placental change; foetal encephalitis and CNS lesions.	Transplacental, vertical transmission.	Neuropathology in foetus; protozoal cysts detected by PCR or immunohistochemistry.	Dubey and Schares, 2011; Lindsay and Dubey, 2020.
Border disease virus	Congenital malformations, mummification, stillbirth.	In utero via viraemic dam.	Viral aetiology; PCR and serology distinguish from bacterial causes.	Oguzoglu and Muz, 2017; WOAH, 2021.

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Indirect costs further compound financial strain. Veterinary consultation, antimicrobial therapy, laboratory diagnostics and biosecurity interventions impose additional expenditures that are rarely budgeted in subsistence systems. Labour associated with isolation of aborting dams and safe disposal of foetal tissues reduces time available for other agricultural activities. Recurrent abortion storms may erode market confidence, lowering sale prices and reducing buyer demand due to perceived disease risk (Kaurivi *et al*., 2020; Shorten *et al*., 2021)

In fragile rural economies, livestock losses frequently trigger distress sales of breeding females to meet immediate household needs, thereby undermining long-term productive capacity. This negative feedback loop perpetuates poverty cycles and reduces resilience to climatic or market shocks. Importantly, the cumulative effect of repeated reproductive losses extends beyond individual households to influence local meat supply, community-level protein availability and regional livestock trade dynamics (Kaurivi *et al*., 2020). Accordingly, *C. jejuni*-associated abortion must be viewed not solely as a veterinary reproductive disorder but as a driver of rural economic instability and food insecurity. Effective prevention and control strategies therefore carry implications not only for flock health but also for household nutrition, income stability and broader agricultural sustainability in resource-constrained settings.

Zoonotic and Public Health Significance

Campylobacter jejuni is recognised as one of the foremost bacterial causes of human gastroenteritis worldwide (Njoga *et al*., 2019; 2020; 2026), and its presence in small ruminant abortion events substantially amplifies its zoonotic risk. It has been reported that aborted foetuses, placentas and vaginal discharges from infected ewes and does contain high concentrations of viable organisms, often exceeding those detected in faecal shedding alone (Yaeger *et al*., 2021). During abortion storms, environmental contamination of bedding, soil and water troughs can be extensive, creating concentrated points of human exposure (Njoga *et al*., 2019; 2020; 2026). Individuals involved in assisting parturition, handling aborted materials or cleaning contaminated pens are therefore at considerable occupational risk.

Farmers, herdsmen, veterinarians and abattoir workers represent the most directly exposed groups, particularly in rural and peri-urban systems where personal protective equipment is inconsistently used (Njoga *et al*., 2023). In many smallholder settings, hand hygiene facilities are limited and contaminated clothing may be worn into domestic environments, increasing the likelihood of household transmission. The informal slaughter and on-farm processing of small ruminants further heighten exposure, especially when carcasses from recently aborting dams are handled without appropriate sanitary precautions (Njoga *et al.,* 2025b; 2025c).

Shared water sources represent an additional and often overlooked pathway. In communities where humans and livestock draw water from the same rivers, ponds or shallow wells, faecal contamination can facilitate indirect transmission. The ability of *C. jejuni* to persist in cold or moist environments allows survival long enough to infect susceptible individuals (Kreling et al., 2020). Consumption of unpasteurised milk and inadequately cooked meat derived from infected animals also remains an important route, particularly in pastoral communities where raw milk consumption is culturally embedded (Njoga *et al.,* 2023).

Clinically, human infection most commonly manifests as acute gastroenteritis characterised by diarrhoea, abdominal pain and fever (Njoga *et al*., 2025a, 2024b, 2025c). However, the public health implications extend beyond self-limiting illness. Post-infectious sequelae such as reactive arthritis and immune-mediated neuropathies, including Guillain–Barré syndrome, contribute to long-term morbidity and healthcare burden (Igwaran and Okoh, 2019). In immunocompromised individuals, invasive disease and bacteraemia may occur. The overlap between reproductive disease in livestock and human exposure pathways reinforces the necessity for integrated One Health approaches. Coordinated surveillance linking veterinary laboratory diagnosis of abortion cases with public health monitoring of enteric disease clusters can facilitate early detection of shared outbreaks. Strengthened biosecurity, education on safe handling of aborted materials and improved water sanitation are therefore critical not only for animal health but also for protecting vulnerable rural populations from zoonotic transmission.

Prevention and Control Strategies

Effective prevention and control of *Campylobacter jejuni*-associated abortion in small ruminants require coordinated interventions that address environmental contamination, host susceptibility and pathogen detection, as outlined in Table 4.

Because abortion events serve as amplification points for flock-level transmission, immediate isolation of aborting ewes or does is a critical first response. Rapid removal and secure disposal of foetuses, placentas and heavily contaminated bedding through deep burial or incineration substantially reduces environmental bacterial load and limits secondary exposure of susceptible pregnant animals (Bloom *et al*., 2022).

Environmental hygiene is particularly important in communal and semi-intensive systems. Protection of water sources through raised troughs, routine cleaning and prevention of direct faecal contamination interrupts the primary faecal-oral transmission cycle. Strategic pasture rotation and avoidance of overcrowding reduce stocking density-related exposure, while separation of late-gestation dams from replacement stock minimises contact between high-risk and potentially infected groups. These measures are especially relevant in resource-constrained rural systems where shared grazing and watering points facilitate rapid pathogen dissemination.

Host resilience represents an equally important control pillar. Adequate energy and protein supplementation during late gestation supports immune competence and may reduce the likelihood of systemic dissemination following intestinal colonisation. Minimisation of transport stress and abrupt dietary changes further reduces physiological immunosuppression that could predispose to bacteraemia and placental invasion.

Early and accurate diagnosis enhances outbreak containment. Molecular detection techniques, including polymerase chain reaction assays targeting virulence-associated genes such as *cdtB* and *ciaB*, enable identification of strains with heightened abortifacient potential (Njoga *et al*., 2023). Integration of molecular typing into routine abortion investigations strengthens surveillance and informs targeted control responses. Where antimicrobial therapy is considered during outbreak situations, treatment should be guided by culture and susceptibility testing to avoid indiscriminate use and to mitigate the risk of antimicrobial resistance development.

Table 4. Integrated prevention and control measures for *Campylobacter* jejuni-induced abortion.

Intervention.	Level of action.	Expected outcome.
Isolation of aborting animals.	Flock.	Limits spread of outbreak.
Safe disposal of foetal tissues.	Environmental.	Reduces contamination.
Water source protection.	Environmental.	Interrupts faecal–oral cycle.
Nutritional support in late gestation.	Host.	Enhances immunity.
Molecular surveillance.	Diagnostic.	Early detection of virulent strains.
Controlled antimicrobial therapy.	Clinical.	Reduces resistance risk.

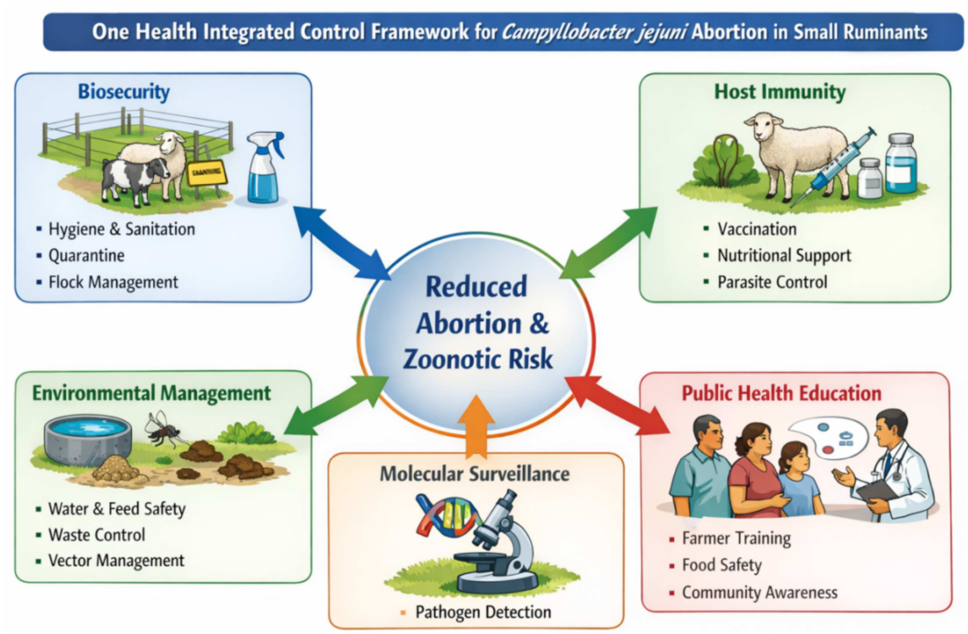

Figure 3. One Health integrated control framework for *Campylobacter jejuni* abortion in small ruminants. Integration of biosecurity, environmental hygiene, host immunity, molecular surveillance, and public health education collectively reduces reproduction losses and zoonotic transmission risk.

At present, commercially available vaccines specifically targeting *C. jejuni*-induced abortion in small ruminants remain limited. Nevertheless, reports on experimental immunisation strategies focusing on conserved antigens such as flagellin and the *CadF* adhesin show promise in reducing colonisation and systemic spread (Wu *et al.*, 2020; Kruglova *et al.*, 2024). Continued research into vaccine development, combined with robust farm-level biosecurity and surveillance, offer the most sustainable pathway to reducing reproduction losses and protecting both animal and public health. A One Health framework integrating biosecurity, environmental management, host immunity, molecular surveillance and public health measures to control *C. jejuni*-induced abortion in small ruminants is illustrated in Figure 3.

Conclusion

Campylobacter jejuni has emerged as a significant abortifacient pathogen in small ruminants, driven by coordinated expression of motility, adhesion, invasion and toxin-associated virulence genes. The molecular cascade from intestinal colonisation to necrotising placentitis explains the predominance of late-term abortion and outbreak dynamics. In rural, under-resourced systems, reproduction losses compromise food security, economic stability and nutritional adequacy. The zoonotic interface further elevates its importance within a One Health framework. Strengthened biosecurity, improved husbandry, molecular surveillance and vaccine development are essential to mitigate its impact on small ruminant production and public health.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

An JU, Lee J, Cho S and Song H (2025). Comparative genomic analysis of livestock-derived Campylobacter jejuni: Antimicrobial resistance, virulence, mobile genetic elements, and genetic relatedness. *Journal of Microbiology and Biotechnology*, 35: e2411044. https://doi.org/10.4014/jmb.2411.11044

Baltenweck I, Enahoro D, Frija A and Tarawali S (2020). Why is production of animal source foods important for economic development in Africa and Asia? *Animal Frontiers*, 10(4): 22 – 29.

Bloom MS, Varde M and Newman RB (2022). Environmental toxicants and placental function. *Best Practice & Research. Clinical Obstetrics & Gynaecology*, 85(Pt B): 105 – 120.

Broaddus CC, Lamm CG, Kapil S, Dawson L and Holyoak GR (2009). Bovine viral diarrhea virus abortion in goats housed with persistently infected cattle. *Veterinary Pathology*, 46(1): 45 – 53. https://doi.org/10.1354/vp.46-1-45

Buxton D (1998). Protozoan infections (Toxoplasma gondii, Neospora caninum and Sarcocystis spp.) in sheep and goats: recent advances. *Veterinary Research*, 29(3–4): 289 – 310.

Choudhury A, Mushi RA and Swai ES (2019). Constraints to effective tick control in smallholder livestock systems in developing countries: A review. *Tropical Animal Health and Production*, 51: 1001 – 1010.

Dasti JI, Tareen AM, Lugert R, Zautner AE and Groß U (2010). *Campylobacter jejuni* virulence mechanisms overview. *International Journal of Medical Microbiology*, 300(3): 205 – 211. https://doi.org/10.1016/j.ijmm.2009.08.002

Dubey JP (2010). *Toxoplasmosis of Animals and Humans*, 2nd ed. CRC Press, Boca Raton, FL.

Dubey JP and Jones JL (2008). Toxoplasma gondii infection in humans and animals. *International Journal for Parasitology*, 38(11): 1257 – 1278. https://doi.org/10.1016/j.ijpara.2008.03.007

Dubey JP and Schares G (2011). Neosporosis in animals—The last five years. *Korean Journal for Parasitology*, 49(1): 1 – 16. https://doi.org/10.3347/kjp.2011.49.1.1

Ekere SO, Njoga EO, Onunkwo JI and Njoga UJ (2018). Serosurveillance of *Brucella* antibody in food animals and role of slaughterhouse workers in spread of *Brucella* infection in Southeast Nigeria. *Veterinary World,* 11: 1171 – 1178.

Elmi A, Nasher F, Dorrell N, Wren B and Gundogdu O (2021). Revisiting Campylobacter jejuni virulence and fitness factors: Role in sensing, adapting, and competing. *Frontiers in Cellular and Infection Microbiology*, 10: 607704. https://doi.org/10.3389/fcimb.2020.607704

Essig A and Longbottom D (2015). *Chlamydia abortus*: New aspects of infectious abortion in sheep and goats. *The Veterinary Journal*, 205(2): 119 – 127. https://doi.org/10.1016/j.tvjl.2015.05.025

Gabbert AD, Mydosh JL, Talukdar PK, Gloss LM, McDermott JE, Cooper KK, Clair GC and Konkel ME (2023). The missing pieces: The role of secretion systems in *Campylobacter* jejuni virulence. *Biomolecules*, 13: 135. https://doi.org/10.3390/biom13010135

Ghielmetti G, Seth-Smith HMB, Roloff T, Cernela N, Biggel M, Stephan R and Egli A (2023). Whole-genome-based characterization of Campylobacter jejuni from human patients with gastroenteritis collected over an 18-year period reveals increasing prevalence of antimicrobial resistance. *Microbial Genomics,* 9: 000941. https://doi.org/10.1099/mgen.0.000941

Godfroid J, Nielsen K and Saegerman C (2010). Diagnosis of brucellosis in livestock and wildlife. *Veterinary Microbiology*, 140(3–4): 246 – 260.

Guerry P, Szymanski CM, Cameron A and Ketley JM (2006). Flagella and motility in Campylobacter. In: *Campylobacter*, 3rd ed., Nachamkin I, Szymanski CM, Blaser MJ (eds). ASM Press, Washington DC.

Haiko J and Westerlund-Wikström B (2013). The role of the bacterial flagellum in adhesion and virulence. *Biology*, 2: 1242 – 1267.

Hänninen ML and Rautelin H (2013). Campylobacter jejuni epidemiology and resistance. In: *Campylobacter*, 5th ed. ASM Press. https://doi.org/10.1128/9781555816942

Hoo R, Nakimuli A and Vento-Tormo R (2020). Innate immune mechanisms to protect against infection at the human decidual-placental interface. *Frontiers in Immunology*, 11: 2070. https://doi.org/10.3389/fimmu.2020.02070

Hu L, Bray MD, Osorio M and Kopecko DJ (2006). Campylobacter jejuni induces maturation and cytokine production in human dendritic cells. *Infection and Immunity,* 74: 2697 – 2705. https://doi.org/10.1128/IAI.74.5.2697-2705.2006

Hussain T, Murtaza G, Kalhoro DH, Kalhoro MS, Yin Y, Chughtai MI, Tan B, Yaseen A and Rehman ZU (2022). Understanding the immune system in fetal protection and maternal infections during pregnancy. *Journal of Immunology Research*, 2022: 7567708. https://doi.org/10.1155/2022/7567708

Igwaran A and Okoh AI (2019). Human campylobacteriosis: A public health concern of global importance. *Heliyon*, *5*(11), e02814. https://doi.org/10.1016/j.heliyon.2019.e02814

Janssen R, Krogfelt KA, Cawthraw SA, van Pelt W, Wagenaar JA and Owen RJ (2008). Host-pathogen interactions in Campylobacter infections: the host perspective. *Clinical Microbiology Reviews*, 21(3): 505 – 518. https://doi.org/10.1128/CMR.00055-07

Jinadasa RN, Bloom SE, Weiss RS and Duhamel GE (2011). Cytolethal distending toxin: a conserved bacterial genotoxin that blocks cell cycle progression, leading to apoptosis of a broad range of mammalian cell lineages. *Microbiology (Reading, England)*, 157(Pt 7): 1851 – 1875. https://doi.org/10.1099/mic.0.049536-0

Kaurivi YB, Laven R, Parkinson T, Hickson R and Stafford K (2020). Effect of animal welfare on the reproductive performance of extensive pasture-based beef cows in New Zealand. *Veterinary Sciences*, 7(4): 200. https://doi.org/10.3390/vetsci7040200

Khairullah AR, Kurniawan SC, Puspitasari Y, Aryaloka S, Silaen OSM, Yanestria SM, Widodo A, Moses IB, Effendi MH, Afnani DA, Ramandinianto SC, Hasib A and Riwu KHP (2024). Brucellosis: Unveiling the complexities of a pervasive zoonotic disease and its global impacts. *Open Veterinary Journal*, 14(5): 1081 – 1097.

Konkel ME, Kim BJ, Rivera-Amill V and Garvis SG (2004). Secretion of Campylobacter jejuni proteins required for host cell invasion. *Molecular Microbiology*, 57(2): 518 – 531.

Konkel ME, Larson CL and Flanagan RC (2005). *Campylobacter jejuni* Cia proteins and host cell interactions. *Microbes and Infection*, 6(1): 93 – 101.

Kreling V, Falcone FH, Kehrenberg C and Hensel A (2020). *Campylobacter* sp.: Pathogenicity factors and prevention methods—new molecular targets for innovative antivirulence drugs? *Applied Microbiology and Biotechnology*, 104: 10409 – 10436.

Kruglova M, Nikitin N, Evtushenko E, Matveeva I, Mazurov A, Pavlenko I, Popova V, Bogomolova O, Vasilyev S, Markova E and Fedorov Y (2024). Inactivated flagellin-containing vaccine efficacy against ovine enzootic abortion. *Pathogens*, 13: 277.

Larson CL, Samuelson DR, Eucker TP, O'Loughlin JL and Konkel ME (2013). The fibronectin-binding motif within FlpA facilitates *Campylobacter* jejuni adherence to host cell and activation of host cell signaling. *Emerging Microbes & Infections*, 2: e65. https://doi.org/10.1038/emi.2013.65

Lindsay DS and Dubey JP (2020). Neospora caninum and neosporosis. *Microorganisms*, 8(4): 571. https://doi.org/10.3390/microorganisms8040571

Longbottom D and Coulter LJ (2003). Animal chlamydioses and zoonotic implications. *Journal of Comparative Pathology*, 128(4): 217 – 244. https://doi.org/10.1053/jcpa.2002.0629

Louwen R, van Marrewijk A and Jackman TH (2005). Cytolethal distending toxin of *Campylobacter jejuni*. *Infection and Immunity*, 73(10): 7060 – 7069.

Motomura K, Miller D, Galaz J, Liu TN, Romero R and Gomez-Lopez N (2023). The effects of progesterone on immune cellular function at the maternal-fetal interface and in maternal circulation. *The Journal of Steroid Biochemistry and Molecular Biology*, 229: 106254. https://doi.org/10.1016/j.jsbmb.2023.106254

Muema J, Mutono N, Kisaka S, Ogoti B, Oyugi J, Bukania Z, Daniel T, Njuguna J, Kimani I, Makori A, Omulo S, Boyd E, Osman AM, Gwenaelle L, Jost C and Thumbi SM (2023). The impact of livestock interventions on nutritional outcomes of children younger than 5 years old and women in Africa: a systematic review and meta-analysis. *Frontiers in Nutrition*, 10: 1166495. https://doi.org/10.3389/fnut.2023.1166495

Nachamkin I, Szymanski CM and Blaser MJ (2019). *Campylobacter*, 4th ed. ASM Press, Washington, DC. https://doi.org/10.1128/9781555818892

Nettleton PF, Gilray JA, Russo P and Dlissi E (1998). Border disease of sheep and goats. *Veterinary Research*, 29(3–4): 327 – 340.

Njoga EO, Chah KF and Oguttu JW (2026). Molecular epidemiology and genetic relatedness of zoonotic Campylobacter species from human and food-producing animal sources in Enugu State, Nigeria. *Scientific Reports*, 16: 3948. https://doi.org/10.1038/s41598-025-34067-3

Njoga EO, Ezenduka EV and Nwanta JA (2020). Surveillance of Campylobacter infection in indigenous poultry reared at Nsukka, Nigeria. *Notulae Scientia Biologicae*, 12: 242 – 250. https://doi.org/10.15835/nsb12210724

Njoga EO, Mshelbwala PP, Ogugua AJ, Enemuo-Edo EC, Onwumere-Idolor OS, Ogunniran TM, Bernard SN, Ugwunwarua JC, Anidobe EC, Okoli CE, Godwin E, Enem SI and Oguttu JW (2025b). Campylobacter colonisation of poultry slaughtered at Nigerian slaughterhouses: Prevalence, antimicrobial resistance, and risk of zoonotic transmission. *Tropical Medicine and Infectious Disease*, 10: 265. https://doi.org/10.3390/tropicalmed10090265

Njoga EO, Nnaemeka VC, Jaja IF, Oguttu JW, Nwanta JA and Chah KF (2025c). Systematic review and meta-analysis of Campylobacter species infections in humans and food-producing animals in Nigeria, 2002 – 2023: The imperative of a One Health control approach. *One Health*, 20: 101029. https://doi.org/10.1016/j.onehlt.2025.101029

Njoga EO, Nwankwo IO and Ugwunwarua JC (2019). Epidemiology of thermotolerant Campylobacter infection in poultry in Nsukka agricultural zone, Nigeria. *International Journal of One Health*, 5: 92 – 98. https://www.onehealthjournal.org/Vol.5/14.pdf

Njoga EO, Nwanta JA and Chah KF (2023). Detection of multidrug-resistant *Campylobacter* species from food-producing animals and humans in Nigeria: Public health implications and one health control measures. *Comparative Immunology, Microbiology and Infectious Diseases*, 103: 102083. https://doi.org/10.1016/j.cimid.2023.102083

Njoga EO, Ochi E, Nwobi OC, Ugwunwarua JC, Anidobe EC, Onwumere-Idolor OS, Kperegbeyi JI, Atadiose EO, Ogunniran TM, Ezenduka EV and Oguttu JW (2025a). Fecal carriage and multidrug resistance profiles of zoonotic Campylobacter species isolated from broilers in Nsukka, Nigeria. *Veterinary World*, 18: 3409 – 3419. https://doi.org/10.14202/vetworld.2025.3409-3419

Oevermann A, Di Palma S, Doherr MG, Abril C, Zurbriggen A and Vandevelde M (2010). Neuropathogenesis of *Listeria monocytogenes* in ruminants: Selective bacterial tropism and interaction with the blood-brain barrier. *Veterinary Research*, 41(5): 63. https://doi.org/10.1051/vetres/2010030

Oguzoglu TC and Muz D (2017). Pestiviruses in small ruminants: Pathogenesis, diagnosis and control. *Veterinary Microbiology*, 201: 121 – 128. https://doi.org/10.1016/j.vetmic.2017.02.011

Omole Z, Dorrell N, Elmi A, Nasher F, Gundogdu O and Wren BW (2024). Pathogenicity and virulence of *Campylobacter jejuni*: What do we really know? *Virulence*, 15: 2436060. https://doi.org/10.1080/21505594.2024.2436060

Orsaria M, Liviero S, Rossetti E, Pittini C, Driul L, Londero AP and Mariuzzi L (2021). Placental acute inflammation infiltrates and pregnancy outcomes: a retrospective cohort study. *Scientific Reports*, 11(1): 24165. https://doi.org/10.1038/s41598-021-03655-4

Otto B, Haag LM, Fischer A, Plickert R, Kühl AA, Göbel UB, Heimesaat MM and Bereswill S (2012). Campylobacter jejuni induces extra-intestinal immune responses via Toll-like-receptor-4 signaling in conventional IL-10 deficient mice with chronic colitis. *European Journal of Microbiology & Immunology*, 2: 210 – 219.

Parker CT, Gilbert M and Yuki N. (2005). Characterization of *Campylobacter jejuni* lipooligosaccharides. *Journal of Clinical Microbiology*, 43(1): 166 – 172.

Parker CT, Horn ST and Gilbert M (2006). MLST and virulence-associated gene variation in *Campylobacter jejuni*. *Journal of Clinical Microbiology*, 44(5): 1751 – 1762.

Quiñones B, Guilhabert MR, Miller WG, Mandrell RE, Lastovica AJ and Parker CT (2008). Comparative genomic analysis of clinical strains of *Campylobacter* jejuni from South Africa. *PLoS One*, 3: e2015.

Rosario GX, Brown S, Karmakar S, Rumi MAK and Nayak NR (2025). Super-enhancers in placental development and diseases. *Journal of Developmental Biology*, 13(2): 11. https://doi.org/10.3390/jdb13020011

Sahin O, Fitzgerald C, Stroika S, Zhao S, Sippy RJ, Kwan P, Plummer PJ, Han J, Yaeger MJ and Zhang Q (2012). Molecular evidence for zoonotic transmission of an emergent, highly pathogenic Campylobacter jejuni clone in the United States. *Journal of Clinical Microbiology*, 50: 680 – 687.

Sahin O, Kassem II, Shen Z, Lin J, Rajashekara G and Zhang Q (2015). Campylobacter in poultry: Ecology and potential interventions. *Avian Diseases*, 59(2): 185 – 200. https://doi.org/10.1637/11072-032315-Review

Sahin O, Plummer PJ, Jordan DM, Sulaj K, Pereira S, Robbe-Austerman S, Wang L, Yaeger MJ, Hoffman LJ and Zhang Q (2008). Emergence of a tetracycline-resistant Campylobacter jejuni clone associated with outbreaks of ovine abortion in the United States. *Journal of Clinical Microbiology*, 46: 1663 – 1671. https://doi.org/10.1128/JCM.00031-08

Sahin O, Terhorst SA, Burrough ER, Shen Z, Wu Z, Dai L, Tang Y, Plummer PJ, Ji J, Yaeger MJ and Zhang Q (2017). Key role of capsular polysaccharide in the induction of systemic infection and abortion by hypervirulent *Campylobacter* jejuni. *Infection and Immunity*, 85: e00001-17. https://doi.org/10.1128/IAI.00001-17

Satué K, La Fauci D, Medica P, Damiá Gímenez E, Cravana C and Fazio E (2025). Shifts between pro-inflammatory and anti-inflammatory profiles in pregnant mares: a review of physiological functions. *Frontiers in Veterinary Science*, 12: 1660759. https://doi.org/10.3389/fvets.2025.1660759

Seleem MN, Boyle SM and Sriranganathan N (2010). Brucellosis: A re-emerging zoonosis. *Veterinary Microbiology*, 140(3–4): 392 – 398.

Shaapan RM (2016). The common zoonotic protozoal diseases causing abortion. *Journal of Parasitic Diseases*, 40(4): 1116 – 1129. https://doi.org/10.1007/s12639-015-0661-5

Sharp AN, Heazell AE, Crocker IP and Mor G (2010). Placental apoptosis in health and disease. *American Journal of Reproductive Immunology*, 64: 159 – 169.

Sheppard SK, Dallas JF and MacRae M (2006). Campylobacter genotyping and source attribution. *Clinical Infectious Diseases*, 48(8): 1072 – 1078.

Shorten PR, Edwards SJ and Juengel JL (2021). The role of reproductive loss on flock performance: a comparison of nine industry flocks. *Translational Animal Science*, 5(1): txab013. https://doi.org/10.1093/tas/txab013

Tikhomirova A, McNabb ER, Petterlin L, Bellamy GL, Lin KH, Santoso CA, Daye ES, Alhaddad FM, Lee KP and Roujeinikova A (2024). *Campylobacter* jejuni virulence factors: update on emerging issues and trends. *Journal of Biomedical Science*, 31: 45. https://doi.org/10.1186/s12929-024-01033-6

Tremblay W, Mompart F, Lopez E, Quaranta M, Bergoglio V, Hashim S, Bonnet D, Alric L, Mas E, Trouche D, Vignard J, Ferrand A, Mirey G and Fernandez-Vidal A (2021). Cytolethal distending toxin promotes replicative stress leading to genetic instability transmitted to daughter cells. *Frontiers in Cell and Developmental Biology*, 9: 656795. https://doi.org/10.3389/fcell.2021.656795

Uzzau S, Brown DJ, Wallis T, Rubino S, Leori G, Bernard S, Casadesús J, Platt DJ and Olsen JE (2000). Host adapted serotypes of *Salmonella enterica*. *Epidemiology and Infection*, 125(2): 229 – 255. https://doi.org/10.1017/S0950268899004379

Veronese P and Dodi I (2024). *Campylobacter jejuni/coli* infection: Is it still a concern? *Microorganisms*, 12: 2669. https://doi.org/10.3390/microorganisms12122669

WOAH (2022). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. World Organisation for Animal Health (WOAH), Paris. https://www.woah.org

Wu Z, Sahin O, Shen Z, Liu P, Miller WG, Zhang Q (2014). Multilocus sequence typing and virulence gene analysis of *Campylobacter jejuni* isolates from sheep abortions. *Journal of Clinical Microbiology*, 52(8): 2683 – 2689. https://doi.org/10.1128/JCM.00734-14

Wu Z, Yaeger MJ, Sahin O, Xu C, Beyi AF, Plummer PJ, Meral Ocal M and Zhang Q (2020). A homologous bacterin protects sheep against abortion induced by a hypervirulent *Campylobacter jejuni* clone. *Vaccines*, 8: 662. https://doi.org/10.3390/vaccines8040662

Yaeger MJ, Sahin O, Plummer PJ, Wu Z, Stasko JA and Zhang Q (2021). The pathology of natural and experimentally induced *Campylobacter jejuni* abortion in sheep. *Journal of Veterinary Diagnostic Investigation*, 33: 1096–1105. https://doi.org/10.1177/10406387211033293.