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Born by the knife? Mode of delivery, the neonatal gut microbiome and necrotising enterocolitis

Abstract

Background: Caesarean rates are rising worldwide and in India, where provisional national data show over a quarter of births are operative. Necrotising enterocolitis (NEC), a leading surgical emergency in preterm newborns, is increasingly seen as a disorder of host–microbe interaction.

Aim: To review, from a paediatric surgical standpoint, evidence linking mode of delivery, early gut colonisation, and the occurrence, severity and surgical outcome of NEC.

Methods: PubMed, Embase and Scopus were searched, supplemented by citation chasing, for this narrative review.

Findings: Term caesarean-born babies show disrupted transfer of maternal gut bacteria and early colonisation by hospital-associated organisms; in preterm babies, neonatal unit exposures appear to matter more. Proteobacteria-rich dysbiosis often precedes NEC, but no single causal organism is known. Clinical data are inconsistent: pooled analyses and propensity-matched preterm cohorts show no clear effect, whereas national case–control data show odds rising with gestational age, and term babies with congenital heart disease showed higher odds. Labour before birth was linked to lower NEC odds in one very low birth weight cohort. Confounding by indication and selection (collider) bias probably explain much of this. Data on delivery mode and surgical outcome are almost absent.

Conclusion: Caesarean birth is best seen as one early hit in a cumulative dysbiosis pathway, not an independent cause of NEC. Human milk remains the most dependable protection; probiotics may reduce NEC but their regulation is unsettled; maternal microbial transfer remains experimental. Registries linking obstetric and surgical data are needed.

Keywords: necrotising enterocolitis, caesarean section, gut microbiome, dysbiosis, neonatal surgery, probiotics, human milk

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Abbreviations: aOR, adjusted odds ratio; AREDF, absent or reversed end-diastolic flow; CHD, congenital heart disease; CI, confidence interval; FGR, fetal growth restriction; NEC, necrotising enterocolitis; NFHS, National Family Health Survey; NICU, neonatal intensive care unit; OR, odds ratio; PN, parenteral nutrition; RR, risk ratio; SGA, small for gestational age; VLBW, very low birth weight

Introduction

Necrotising enterocolitis (NEC) has been with us for as long as we have kept very small babies alive, and it still takes a heavy toll. It is one of the most common and devastating gastrointestinal emergencies in the neonatal intensive care unit (NICU), it strikes hardest at the most premature and for the babies who need an operation the outlook remains poor:[1] in high-income countries about a third of infants die after surgery for NEC, and many survivors develop intestinal failure or neurodevelopmental impairment.[2,3] Every paediatric surgeon knows the pattern: a distended, discoloured abdomen, free gas on the film, a laparotomy that finds more dead bowel than hoped and, for survivors, months of stoma care, parenteral nutrition and uncertainty.

Over the same decades, the way babies are born has changed. About one in five births worldwide (21.1% in 2010–2018) is by caesarean section, and projections suggest that nearly three in ten (28.5%) will be by 2030.[4] India has followed the same curve and probably faster. The national caesarean rate rose from 17.2% in the fourth National Family Health Survey (2015–16).[5] to 21.5% in the fifth (2019–21),[6] and, according to provisional figures, 27.2% in the sixth (2023–24), when 54.1% of births in private facilities were by caesarean section against 16.9% in public facilities (Figure 1).[7] In Telangana and Andhra Pradesh, more than half of all babies are now born by caesarean section.[7]

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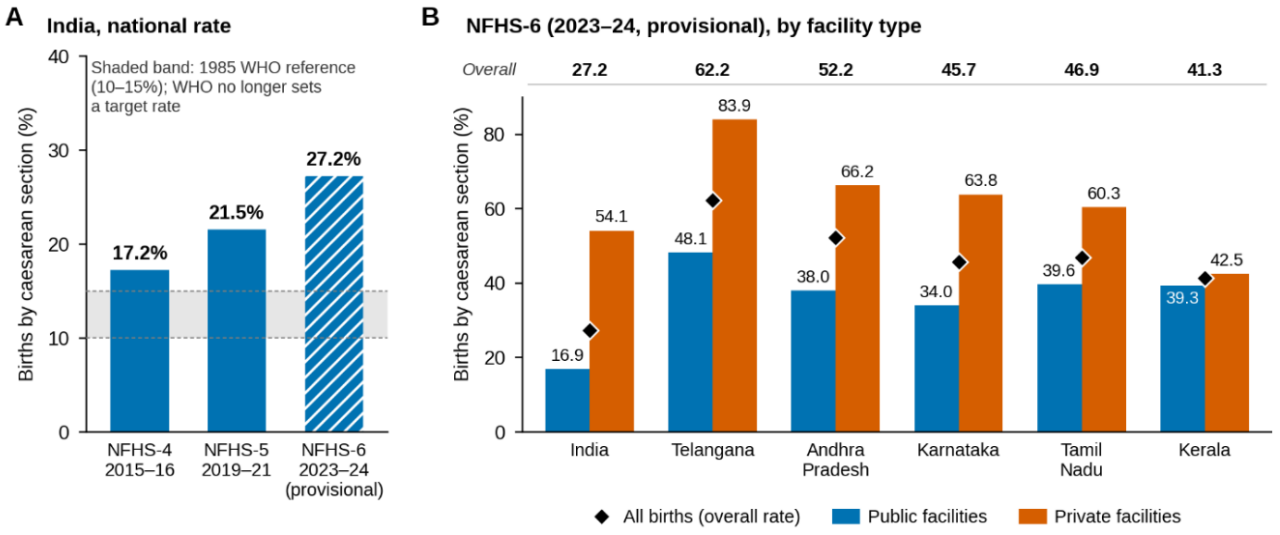


Figure 1 Caesarean births in India.

[A] National caesarean rate across three rounds of the National Family Health Survey (NFHS): NFHS-4, 2015–16 (17.2%);[5] NFHS-5, 2019–21 (21.5%);[6] and NFHS-6, 2023–24 (27.2%; provisional).[7] [B] Caesarean rates in NFHS-6 (2023–24; provisional fact-sheet estimates) for India and the five large southern states (Telangana, Andhra Pradesh, Karnataka, Tamil Nadu and Kerala): bars show births in public and private facilities; diamonds show the overall rate for all births.[7] The shaded band marks the historical 10–15% population reference range from the 1985 WHO consensus meeting; the 2015 WHO statement found that rates above about 10% were not associated with further reductions in maternal and newborn mortality, and advised against aiming for a specific rate.[8] NFHS, National family health survey; WHO, World health organization.

The link between these two trends is the gut microbiome. A vaginal birth gives the newborn a first inoculum of maternal bacteria; a caesarean birth largely does not. Because NEC is increasingly thought of as a disease of disordered host–microbe interaction, it is reasonable to ask whether birth by caesarean section raises the risk of NEC, makes it more severe or changes what happens after surgery.[8]

This question matters to paediatric surgeons for practical reasons. Infection remains a major cause of neonatal death in India: in a Delhi cohort of more than 13,000 NICU admissions, most sepsis isolates were Gram-negative, many were multidrug-resistant and nearly a quarter of deaths were attributable to sepsis,[9] and in one North Indian audit sepsis was the main cause of death in a public-sector neonatal surgical intensive care unit.[10] Paediatric surgeons are also increasingly involved before birth, counselling families alongside obstetricians and fetal medicine specialists. Structured tools such as the Management, Outcome, Risk and Expectation (MORE) classification were developed partly to address whether a fetal anomaly should alter obstetric management, including the timing and mode of delivery,[11] and its reliability has since been tested prospectively.[12] When parents ask whether a planned caesarean will harm their baby's gut, we should be able to answer honestly, without either dismissing the question or feeding unnecessary anxiety.[13]

This review sets out what is known and, just as importantly, what is not. It follows the baby from the birth canal (or the operating table) through colonisation, dysbiosis and NEC, to the laparotomy and beyond, and ends with the interventions that might change that course.

Specifically, we address three questions. First, does birth by caesarean section, or birth without labour, change the incidence of NEC? Second, does mode of delivery influence the severity of NEC, including progression to perforation and the need for surgery? Third, does it affect post-operative outcome and the recovery of the gut microbiome after laparotomy?

Material and methods

This narrative review is based on searches of PubMed, Embase and Scopus for articles published in English, using combinations of the terms necrotising (necrotizing) enterocolitis, caesarean (cesarean) section, mode of delivery, microbiome, microbiota, dysbiosis, probiotics, human milk, vaginal seeding, faecal microbiota transplantation, peritoneal drainage, laparotomy, enterostomy and short bowel syndrome. Reference lists of key papers were hand-searched. Titles and abstracts were screened by two reviewers independently, and studies were selected for their relevance to delivery mode, early gut colonisation, the occurrence or severity of NEC, and its surgical management and outcome. Priority was given to systematic reviews, meta-analyses, randomised trials, large cohort studies and studies using culture-independent sequencing. Because the evidence is heterogeneous and largely observational, no pooled analysis was attempted; the aim was a critical synthesis with a surgical emphasis.

Google Scholar was used only for forward and backward citation chasing and was not searched systematically. The certainty of each key finding was labelled by study design (randomised trial, systematic review or meta-analysis, cohort, case–control or mechanistic sequencing study) and, where available, by the GRADE certainty reported in Cochrane reviews;[14–16] where studies conflicted, the discrepancy and its likely explanations are discussed explicitly rather than resolved by counting studies.

Evidence synthesis

How mode of delivery shapes the first microbiome

The first clear demonstration that birth mode sets the starting point of the infant microbiome came from a small study (n = 10) by Dominguez-Bello and colleagues, who showed that vaginally born

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babies acquire bacterial communities resembling their mother's vaginal flora, whereas babies born by caesarean section are colonised by communities resembling skin, dominated by genera such as *Staphylococcus*, *Corynebacterium* and *Propionibacterium*.[17]

The most informative work since then is the *Baby Biome Study*. Shao and colleagues sequenced stool from 596 babies born in British hospitals, 314 vaginally and 282 by caesarean section.[18] In the caesarean group, transmission of maternal *Bacteroides* strains was disrupted and there was heavy colonisation by opportunistic organisms associated with the hospital environment, including *Enterococcus*, *Enterobacter* and *Klebsiella* species.[18] Over 800 isolates of these opportunistic pathogens were grown and sequenced, and many carried virulence factors and clinically relevant antimicrobial resistance genes. Two caveats from the same study deserve equal weight. The authors could not link the altered colonisation to any clinical outcome, and most of the differences diminished with age; they were still detectable at infancy sampling (mean age about 9 months), when a *Bacteroides* deficit persisted in about 60% of caesarean-born babies.

Other cohorts point the same way. In a small Swedish cohort (n = 24), infants born by caesarean had lower microbial diversity, delayed colonisation by Bacteroidetes and reduced Th1 responses over the first two years.[19] An older Finnish study found that colonisation rates of *Bifidobacterium* and *Lactobacillus* in caesarean-born babies took about a month and about ten days, respectively, to catch up with vaginally born controls and that the flora could remain disturbed for up to six months.[20]

It is worth being precise about what 'caesarean section' means in these studies, because it is not one exposure. It usually travels with intrapartum antibiotic prophylaxis, delayed initiation of breastfeeding, less skin-to-skin contact and a longer hospital stay. Elective caesarean before labour and emergency caesarean after rupture of membranes may expose the baby to quite different microbial environments. At birth, and sampled across several body sites rather than the gut alone, babies delivered by caesarean after labour had begun carried bacteria of both maternal vaginal and skin origin, whereas those delivered before labour carried mainly skin-type flora; by 6 weeks these differences were no longer detectable.[21] For the gut itself the evidence is less clear: the Baby Biome Study found no difference between elective and emergency caesarean,[18] and in a US cohort both planned and in-labour caesarean-born infants lacked *Bacteroides* at 2 weeks.[22] Most clinical studies do not make this distinction, which limits how far we can attribute effects to the operation itself. Table 1 summarises how these routes of birth differ.

Table 1 How the route of birth shapes the first gut microbiota

Route of birth	Main source of first bacteria	Early pattern reported	Usual co-exposures
Vaginal birth	Maternal vaginal and faecal flora	Communities resembling maternal vaginal flora; maternal *Bacteroides* strains transmitted	Earlier skin-to-skin contact and breastfeeding; intrapartum antibiotics in some mothers
Caesarean after labour began	Maternal skin and vagina	At birth, mixed vaginal- and skin-type flora across body sites; in gut studies, *Bacteroides* deficit similar to pre-labour caesarean	Intrapartum antibiotics; ruptured membranes; often emergency indication
Caesarean before labour	Mainly maternal skin and hospital environment	Skin-type genera at birth; delayed *Bacteroides*, *Bifidobacterium* and *Lactobacillus*; more *Enterococcus*, *Klebsiella*, *Enterobacter* (gut findings largely shared with emergency caesarean)	Pre-incision antibiotics; delayed breastfeeding; frequently a maternal or fetal indication

Most differences narrow over the first months; in the Baby Biome Study they had diminished by infancy (mean age about 9 months), although a Bacteroides deficit persisted in about 60% of caesarean-born babies.

Why should any of this matter to the gut wall? Commensal anaerobes ferment dietary substrates into short-chain fatty acids that feed colonocytes and support tight-junction integrity; they also shape bile acid metabolism and help train mucosal immunity. When this ecosystem is disturbed, barrier function weakens and bacterial products reach the portal circulation more easily, a mechanism now well described along the gut–liver axis.[23] In the newborn, whose epithelium and immune signalling are immature, the same mechanism provides a plausible route from abnormal colonisation to intestinal injury.

The preterm gut and the dysbiosis that precedes NEC

For preterm babies the picture is different again. They are separated from their mothers at birth, often receive antibiotics in the first days, may start feeds late and spend weeks in an environment full of hospital organisms. Under these conditions the NICU, rather than the birth canal, appears to be the dominant source of colonising bacteria: strains that colonise preterm infants are found on NICU sinks and surfaces, often before they appear in the gut,[24] and in longitudinal preterm cohorts birth mode altered at most the pace, not the pattern, of gut colonisation, which followed postconceptional age.[25,26] Timing matters as well. The weeks in which caesarean-born and vaginally born babies differ most are also the weeks in which preterm babies are most at risk of NEC (Figure 2) .[27]

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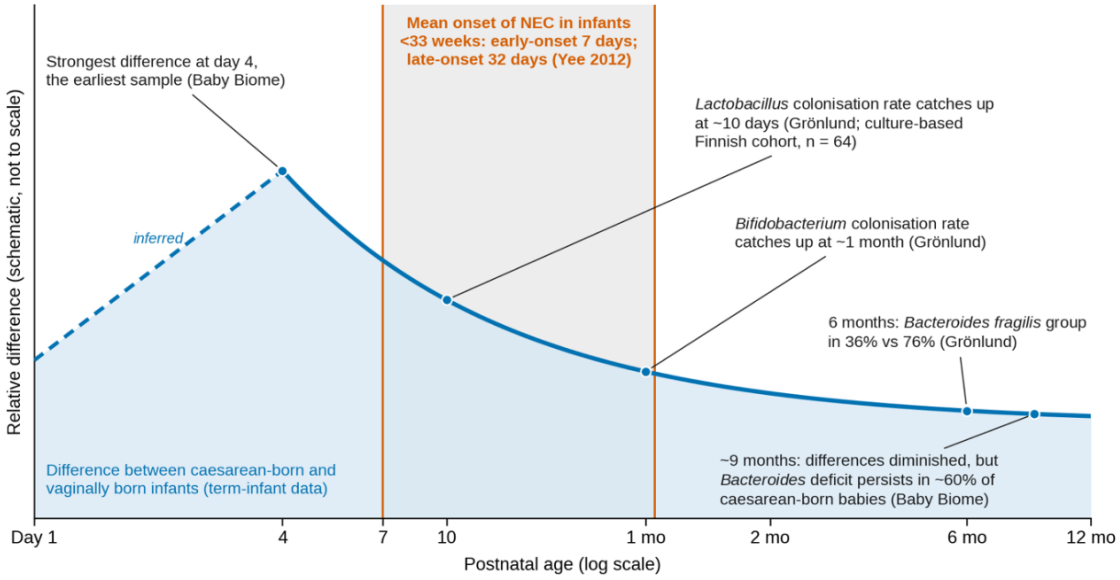


Figure 2 Schematic timeline of early gut colonisation by route of birth, set against the usual timing of necrotising enterocolitis (NEC) in preterm infants.

The curve is illustrative and has no units; it represents the difference between caesarean-born and vaginally born infants. The marked points come from studies of healthy term infants: a culture-based Finnish cohort of 64 infants, in which Lactobacillus colonisation in caesarean-born infants caught up after about 10 days and Bifidobacterium after about 1 month,[20] and the Baby Biome Study, in which the delivery-mode effect was strongest at day 4 and had largely resolved by infancy (mean age about 9 months), except for a Bacteroides deficit that persisted in about 60% of caesarean-born babies.[18] The shaded band spans the mean age at onset of early-onset NEC (<14 days; mean 7 days) and late-onset NEC (≥14 days; mean 32 days) in a Canadian national cohort of 16,669 infants born before 33 weeks; late onset occurred mainly in less mature infants.[27] Because the colonisation data are from term babies and NEC mainly affects preterm babies, the overlap is indicative only. In preterm infants, dysbiosis has been shown to precede the onset of NEC.[28] NEC, necrotising enterocolitis.

What is now well established is that NEC does not arise from a sterile or normal gut. In the systematic review and meta-analysis by Pammi et al., which reprocessed 16S rRNA sequence data from eight cohorts (106 NEC cases, 278 controls) through a single pipeline, stool from preterm infants who later developed NEC showed increased relative abundance of Proteobacteria and decreased Firmicutes and Bacteroidetes before the disease began.[28] The largest prospective study, of 3,586 stools from 166 very low birth weight infants, found the same direction of change, more Gammaproteobacteria and fewer strict anaerobes before NEC, most clearly in babies born before 27 weeks.[29] Diversity measures, by contrast, were not consistent across studies. No single organism has been identified as the cause; the evidence points to a dysbiotic community rather than a classical pathogen.

This fits the prevailing model of NEC pathogenesis, in which an immature intestine with exaggerated inflammatory signalling meets an abnormal microbial community, often under conditions of reduced mesenteric perfusion.[1,3] It also provides a mechanistic bridge to delivery mode, because the Proteobacteria and hospital-associated genera enriched before NEC overlap with the organisms over-represented in caesarean-born babies;[18,28] *Klebsiella*, for example, is both more common after caesarean birth[18] and enriched, with increased bacterial replication, in the days before NEC.[30] Mechanistic plausibility, however, is not the same as clinical proof.

Does caesarean birth raise the risk of NEC? The clinical evidence

When we turn from microbiology to clinical outcomes, the association becomes much less secure. The key studies are summarised in Table 2 and the available effect estimates are plotted in Figure 3.

Table 2 Selected clinical studies on mode of delivery, labour and NEC

Study	Population and design	Main finding	Comment
Su et al.,[31]	Meta-analysis of risk-factor studies in neonates	Caesarean section not significantly associated with NEC (OR 1.31, 0.86–2.00; 4 studies, I^2 80.7%)	Pooled observational studies; caesarean indication not reported
Ahle et al.,[32]	720 NEC cases and 3,567 controls matched for gestational age and birth year; Swedish national registers, 1987–2009; case–control	Caesarean section: aOR 1.54 (1.25–1.89) overall; <28 weeks 0.93 (0.66–1.29); 28–31 weeks 1.42 (0.96–2.09); 32–36 weeks 2.07 (1.23–3.48); term 3.72 (2.15–6.45)	Population-based; association rises with gestational age; pre-eclampsia was inversely associated with NEC, consistent with selection (collider) bias in preterm strata
Son et al.,[33]	2,012 singletons at imminent risk of delivery at 24–31+6 weeks; secondary analysis of a US multicentre randomised trial of antenatal magnesium sulfate (MFMU BEAM)	Caesarean birth (36% of infants): NEC 8.1% vs 8.7% after vaginal birth; any NEC aOR 0.74 (0.52–1.04); NEC stage 2–3 aOR 0.73 (0.46–1.16)	Prospectively collected trial data; no significant association; restricted to very preterm infants

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Study	Population and design	Main finding	Comment
Riskin et al.,[34]	20,223 infants of 24–31 weeks and ≤1,500 g; Israel national VLBW database, 1995–2015	NEC stage 2–3: no association in singletons; multiple births OR 1.31 (1.01–1.69)	Population-based
Wang et al.,[35]	1,375 infants <32 weeks and <1,500 g (390 propensity-matched pairs); single-centre retrospective cohort, China, 2016–2021	No difference in NEC or surgical NEC	Caesarean birth linked to lower 3-day mortality below 28 weeks; 52% of caesareans were emergency procedures
Dhawan et al.,[36]	1,951 VLBW infants of 23–34+6 weeks; single-centre retrospective cohort, USA, 2012–2023	Labour before birth: aOR 0.61 (0.38–0.98) for NEC; surgical NEC aOR 0.73 (0.38–1.39), not significant	Described by the authors as the first study of labour exposure; not significant in the 2018–23 subset adjusted for maternal diabetes (aOR 0.63, 0.36–1.10)
Ihle et al.,[37]	952 term neonates undergoing cardiac surgery at ≤28 days, 2007–2017; 60 NEC cases, 178 matched controls analysed; Melbourne	Caesarean birth: aOR 2.64 (1.31–5.29)	aOR 2.00 (1.04–3.86) in the less-adjusted model; pre-caesarean antibiotics not captured; labour status and caesarean urgency not reported; 72% of NEC post-operative; planned caesarean common after prenatal CHD diagnosis

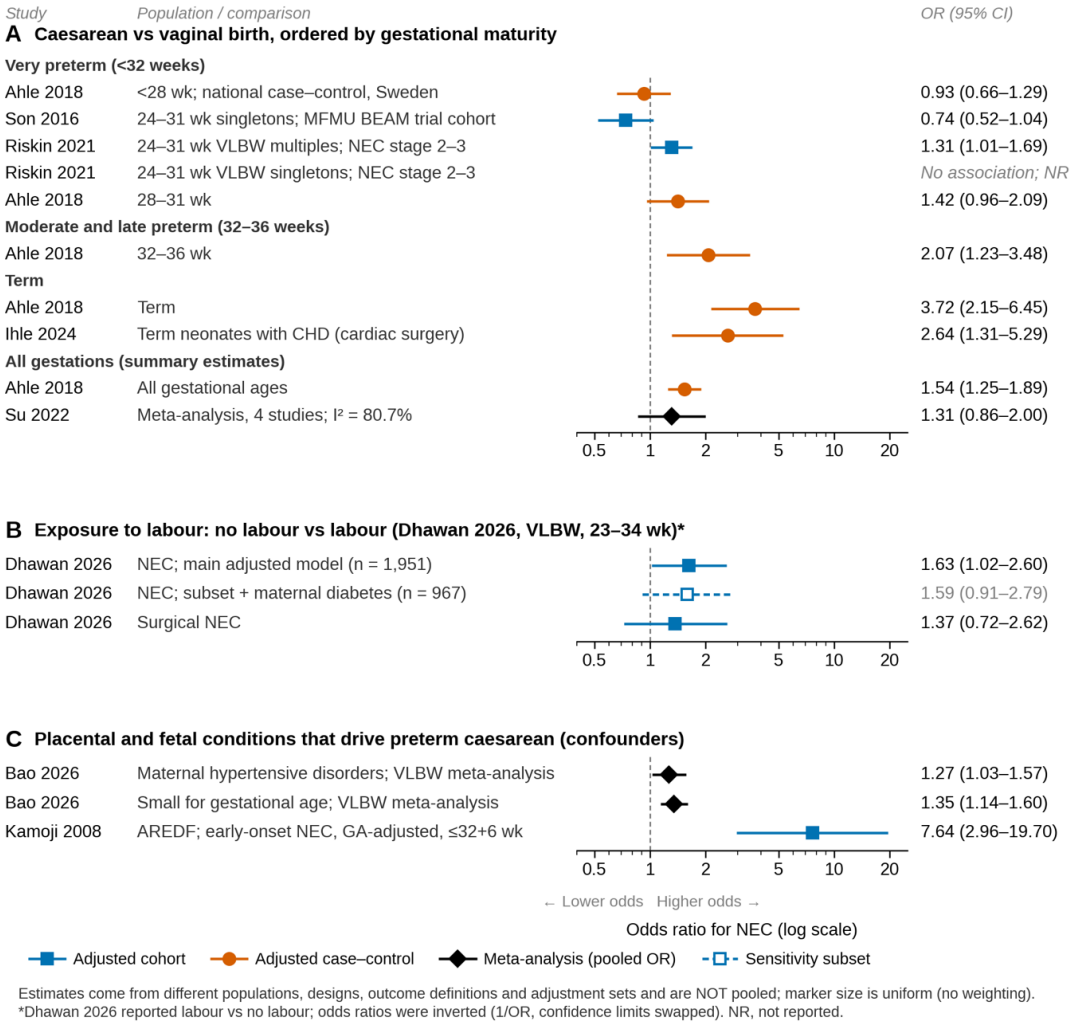


Figure 3 Reported associations of birth route, labour and placental–fetal conditions with necrotising enterocolitis (NEC).

[A] Caesarean versus vaginal birth, ordered by gestational maturity. In a Swedish national case–control study, the adjusted odds ratio rose from 0.93 below 28 weeks to 3.72 at term; its overall estimate is shown with the summary estimates.[32] Very preterm estimates are from a secondary analysis of 2,012 singletons at 24–31 weeks in the MFMU BEAM trial[33] and from the Israel national VLBW database for NEC stage 2–3, which found an association in multiple births but

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none in singletons (no singleton estimate reported).[34] The term estimate for congenital heart disease is from neonates undergoing cardiac surgery.[37] The pooled odds ratio is from a random-effects meta-analysis of four studies (I^2 = 80.7%).[31] A propensity-matched cohort of 1,375 very preterm infants found no difference in NEC but reported no odds ratio and is not plotted.[35] [B] Exposure to labour in VLBW infants of 23–34 weeks.[36] The published adjusted odds ratios for labour versus no labour were inverted *(1/OR, with confidence limits swapped) so* that they express the odds associated with no labour: NEC, main adjusted model (n = 1,951), 1.63 (1.02–2.60); surgical NEC, 1.37 (0.72–2.62). The open marker and dashed line show the 2018–2023 subset (n = 967) also adjusted for maternal diabetes, in which the association was no longer significant (1.59, 0.91–2.79). [C] Placental and fetal conditions that commonly lead to preterm caesarean delivery and act as confounders: pooled odds ratios for maternal hypertensive disorders and small for gestational age from a meta-analysis of VLBW infants,[42] and AREDF as a risk factor for early-onset NEC in infants born at or before 32+6 weeks, adjusted for gestational age.[43] Squares show adjusted cohort estimates, circles adjusted case–control estimates, diamonds pooled estimates from meta-analyses and the open square a sensitivity subset; horizontal lines show 95% CIs on a log scale. Estimates come from different populations, designs, outcome definitions and adjustment sets; they were not pooled, and marker size does not reflect study weight. aOR, adjusted odds ratio; AREDF, absent or reversed end-diastolic flow in the umbilical artery; CHD, congenital heart disease; CI, confidence interval; MFMU BEAM, Maternal–Fetal Medicine Units Network Beneficial Effects of Antenatal Magnesium Sulfate trial; NEC, necrotising enterocolitis; NR, not reported; OR, odds ratio; VLBW, very low birth weight (≤1,500 g); wk, weeks.

A 2022 meta-analysis of risk factors for neonatal NEC found no significant association with caesarean section (pooled OR 1.31, 95% CI 0.86–2.00; four studies, I^2 80.7%),[31] and an earlier systematic review of multivariable prognostic studies found caesarean birth associated with a lower risk of NEC in at least two studies.[38] A 2025 single-centre cohort of 1,375 very preterm infants (below 32 weeks and 1,500 g) found, after propensity matching (390 pairs), no difference in NEC or in NEC requiring surgery between vaginal and caesarean deliveries.[35] In that cohort, caesarean birth was actually associated with lower three-day mortality in babies below 28 weeks, at the cost of more respiratory morbidity at 28–31 weeks.[35] A secondary analysis of 2,012 singletons born at 24–31 weeks in a US multicentre trial likewise found no association (adjusted OR 0.74, 95% CI 0.52–1.04).[33]

Two further cohorts add nuance. In the Israel national cohort of 20,223 very preterm, very low birth weight infants, caesarean delivery was not associated with NEC in singletons, but in multiple births it carried modestly higher odds (OR 1.31, 95% CI 1.01–1.69).[34] More recently, a single-centre American cohort of 1,951 very low birth weight infants looked at labour rather than the route of delivery. Babies exposed to labour before birth, a group that includes all vaginal births and caesareans performed after labour had begun, had lower odds of NEC (adjusted OR 0.61, 95% CI 0.38–0.98) and the direction of effect was the same in every gestational age band.[36] The authors suggest that exposure to maternal vaginal flora during labour may explain the difference. Strain-tracking studies urge caution: maternal vaginal and skin strains colonise the infant gut only transiently, whereas maternal gut strains persist,[39] and in one cohort both planned and in-labour caesarean-born infants lacked *Bacteroides* at 2 weeks.[22] Labour also marks a less compromised pregnancy (see below), and the association lost significance in the subset adjusted for maternal diabetes.[36] If confirmed, this would be the most useful clinical signal so far, because it points to a biologically meaningful distinction that simple '*caesarean versus vaginal*' comparisons blur.

The picture changes in term babies with congenital heart disease. In a case–control study from Melbourne of 952 term neonates who had cardiac surgery, 60 developed NEC (6.3%); after adjustment, caesarean birth was associated with more than twice the odds of NEC (adjusted odds ratio 2.64, 95% CI 1.31–5.29; 2.00, 1.04–3.86 in the less-adjusted model).[37] Most cases (43 of 60, 72%) arose after cardiac surgery, a different exposure window from the perinatal period.[37] This matters to surgeons, because NEC in this group is not trivial. In a separate Irish series of 25 term infants with CHD and NEC (including Bell stage 1 cases), just over a third needed surgical management, 30-day mortality was 20% and long-term mortality reached 40%.[40] In a US multi-institutional cohort of 38,770 neonates with CHD, NEC occurred in 3.7% and was associated with 24.4% in-hospital mortality, against 11.8% without NEC.[41]

Why should the association be clear in term cardiac babies and not in preterm ones? The pattern is not confined to cardiac babies: in a Swedish national case–control study of 720 NEC cases, the caesarean–NEC association rose steadily with maturity, from null below 28 weeks (aOR 0.93, 95% CI 0.66–1.29) to 3.72 (2.15–6.45) at term.[32] The most plausible explanation is confounding by indication. A preterm caesarean is rarely elective in the usual sense. It is commonly done for pre-eclampsia, fetal growth restriction or abnormal umbilical artery Doppler studies; these conditions predispose to NEC through gut ischaemia. In a geographically defined cohort of 206 preterm babies, absent or reversed end-diastolic flow was an independent risk factor for early-onset NEC, with an odds ratio of about 7.6 (95% CI 3.0–19.7) after adjustment for gestational age.[43] Babies exposed to such flow patterns also show persisting abnormalities of splanchnic blood flow after birth.[44] Pooled data point the same way: in a 2026 meta-analysis of very low birth weight infants, maternal hypertensive disorders (OR 1.27) and being small for gestational age (OR 1.35) were both associated with NEC.[42] The labour study described above shows how tightly the route of birth is bound to its reason: babies born without labour were more than three times as likely to be small for gestational age (29.8% versus 9.0%).[36] These relationships are summarised in Table 3. In preterm cohorts, therefore, caesarean birth marks a sick placenta as much as a surgical route of delivery, and any microbial effect is buried under much larger haemodynamic and gestational effects. Restricting analyses to very preterm or very low birth weight infants adds a second problem, selection (collider) bias: almost every such baby is born early because of some pathology, so an exposure that is harmful in the whole population can appear null or even protective.[45,46] This may explain the null or protective caesarean estimates in preterm-restricted cohorts and the inverse association of pre-eclampsia with NEC in the Swedish data.[32] In term cardiac babies, where these obstetric confounders are fewer, a microbial contribution may become visible. That is a hypothesis, not a finding, but it is testable. Figure 4 sets out these relationships as a directed acyclic graph: placental dysfunction is a common cause of both pre-labour caesarean delivery and NEC, and restricting a study to preterm or very low birth weight infants conditions on a collider that opens a second, non-causal path between them.[47]

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Table 3 Obstetric and fetal conditions that confound the caesarean–NEC association

Condition	Link to caesarean delivery	Association with NEC	Likely mechanism
Maternal hypertensive disorders, including pre-eclampsia	Frequently leads to planned preterm delivery	OR 1.27 (1.03–1.57) in VLBW infants[42]	Reduced fetal blood supply from placental insufficiency[42]
Fetal growth restriction / small for gestational age	SGA in 29.8% of infants born without labour vs 9.0% after labour[36]	OR 1.35 (1.14–1.60);[42] OR 1.85 (1.15–2.97)[31]	Placental insufficiency; weaker gut barrier and immune function[42]
Absent or reversed end-diastolic flow	Elective preterm delivery is common[44]	Early-onset NEC: OR 7.64 (2.96–19.70), adjusted for gestational age[43]	Antenatal gut ischaemia; abnormal splanchnic flow persisting after birth[43,44]
Maternal diabetes, including gestational diabetes	Commoner in infants born without labour (7.5% vs 5.2%)[36]	Gestational diabetes: OR 3.62 (1.77–7.41)[31]; not associated in one VLBW cohort (aOR 0.85, 0.37–1.97)[36]	Uncertain
Congenital heart disease, often diagnosed before birth	Prenatal diagnosis often followed by planned caesarean[37]	OR 2.73 (1.10–6.78)[31]	Mesenteric hypoperfusion[37]

aOR, adjusted odds ratio; NEC, necrotising enterocolitis; OR, odds ratio; SGA, small for gestational age; VLBW, very low birth weight. Values in parentheses are 95% confidence intervals. Reference 31 pooled studies of all neonates; reference 42 pooled studies of VLBW infants only. Odds ratios are shown as reported in the source studies.

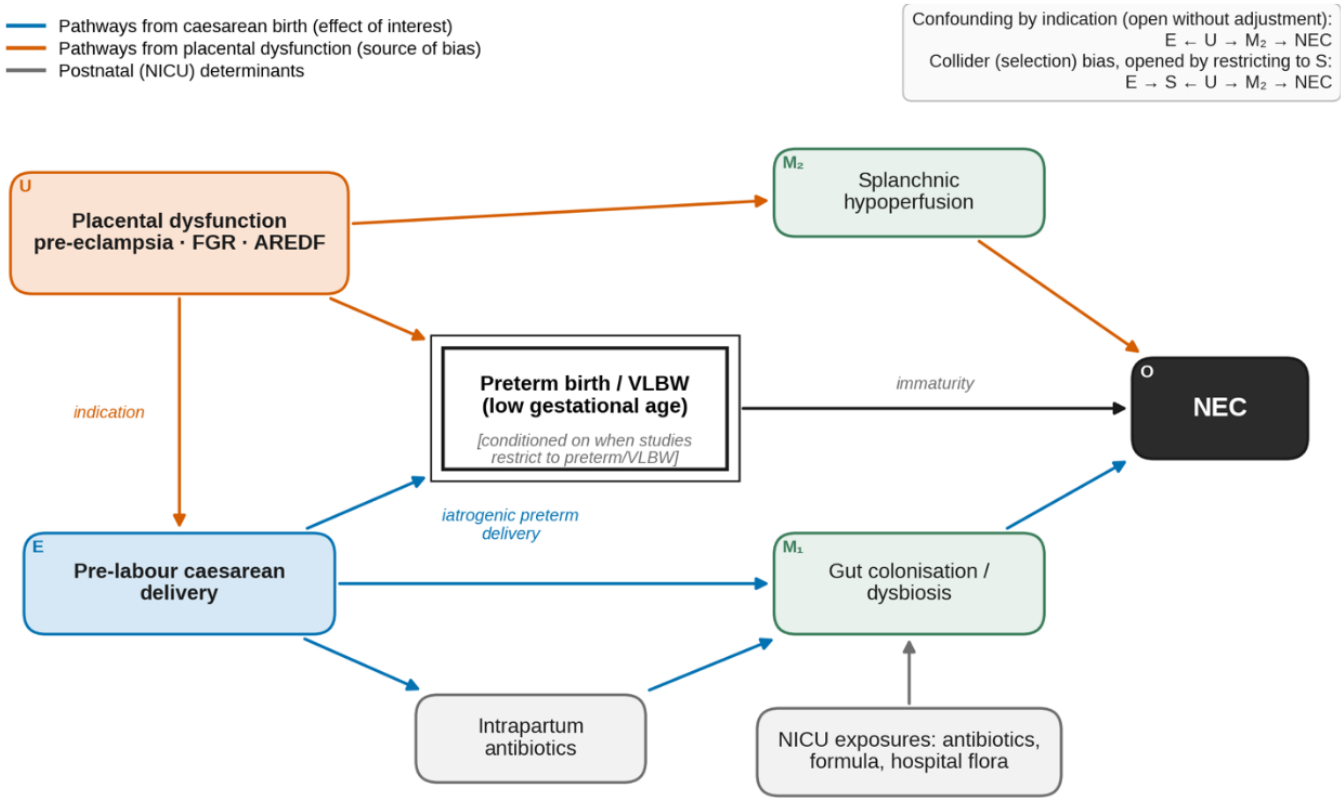


Figure 4 Directed acyclic graph of how birth route may relate to necrotising enterocolitis (NEC), and why observational estimates can be biased.

Placental dysfunction (U; pre-eclampsia, FGR, AREDF) is a common indication for pre-labour caesarean delivery (E) and independently predisposes to NEC (O) through splanchnic hypoperfusion (M2). Caesarean birth may affect NEC through altered gut colonisation and dysbiosis (M1), both directly and through intrapartum antibiotics, and through iatrogenic preterm delivery; in preterm infants, NICU exposures (antibiotics, formula feeding, hospital flora) are major further determinants of colonisation. Two sources of bias are shown. Confounding by indication: the path E ← U → M2 → NEC remains open unless the indication is measured and adjusted for.[48] Collider (selection) bias: preterm birth or VLBW (S; double-bordered box) is caused by both caesarean delivery and placental dysfunction, so it is a collider; restricting a study to preterm or VLBW infants conditions on S and opens the non-causal path E → S ← U → M2 → NEC, which can make caesarean birth appear harmless or even protective.[45,46] Blue arrows show pathways from caesarean birth, vermillion arrows pathways from placental dysfunction and grey arrows postnatal determinants. The graph encodes causal assumptions, not effect sizes, and was drawn following current recommendations for reporting directed acyclic graphs.[47] AREDF, absent or reversed end-diastolic flow; E, exposure; FGR, fetal growth restriction; M1 and M2, mediators; NEC, necrotising enterocolitis; NICU, neonatal intensive care unit; O, outcome; S, selection (collider) node; U, common cause; VLBW, very low birth weight.

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Mode of delivery and surgical NEC

For the surgeon, the more pressing questions are narrower. Does delivery mode predict which babies perforate? Does it influence whether peritoneal drainage succeeds or fails? Does it affect stricture, short bowel syndrome or death after laparotomy? At present we cannot answer any of these with confidence, although the baseline rates are known: after surgery for NEC, strictures develop in about a quarter of survivors and intestinal failure in about one in eight.[49] Direct data are scant. In the labour study described above, surgery for NEC was needed in 2.4% of labour-exposed and 3.5% of unexposed infants, a difference that was not significant after adjustment;[36] in a small retrospective cohort of 56 preterm infants with NEC, delivery mode did not differ between those managed surgically and medically.[50] No adequately powered study has examined birth mode as a predictor of perforation, operative choice or post-operative outcome, and surgical cohorts that record delivery mode generally report it only as a baseline characteristic.

The main randomised evidence in surgical NEC concerns the operation, not the birth. Laparotomy and primary peritoneal drainage produced similar survival in very low birth weight infants with perforated NEC in a North American multicentre trial,[51] while an international multicentre trial found drainage ineffective as a definitive procedure in most infants.[52] The largest trial, in 310 extremely low birth weight infants, found no overall difference in death or neurodevelopmental impairment, but a 97% Bayesian probability of benefit from initial laparotomy when the preoperative diagnosis was NEC rather than isolated intestinal perforation;[53] a 2025 Cochrane review of the three trials found little or no difference in death or neurodevelopmental impairment between the two approaches.[16] The recent multicentre STAT trial found that infants randomised to primary anastomosis after resection came off parenteral nutrition earlier than those given a stoma.[54] National cohort studies such as the UK-wide BAPS-CASS study of surgical NEC show that prospective registries of every infant needing surgery are feasible.[55] Adding a handful of obstetric variables to such registries (delivery mode, labour status, indication for caesarean and intrapartum antibiotics) would cost little and, if analyses were framed as emulations of a target trial (for example, planned pre-labour caesarean versus planned labour, with time zero at the decision to deliver),[56,57] could go a long way towards answering the question within a few years; a proposed minimum dataset is given in Table 4.

Table 4 Proposed obstetric–surgical minimum dataset for NEC registries

Domain	Variable	Suggested definition	Why it matters
Infant	Gestational age, birth weight, birth-weight z-score	Completed weeks; grams; z-score or centile	Strongest confounders
Infant	Multiple birth; congenital heart disease	Singleton or multiple; CHD yes/no, with diagnosis	Associations reported in multiples and in term CHD
Pregnancy	Maternal hypertension, diabetes and Doppler findings	Pre-eclampsia; pre-existing or gestational diabetes; AREDF	Confounders linked to both caesarean and NEC
Birth	Mode of delivery	Vaginal (spontaneous or assisted); caesarean, planned or unplanned	Primary exposure
Birth	Labour before delivery	Contractions with cervical change before birth; ruptured membranes (yes/no)	Proposed proxy for exposure to vaginal flora
Birth	Indication for caesarean	Pre-eclampsia, FGR, abnormal Doppler, fetal distress, malpresentation, previous caesarean, maternal request, other	Controls for confounding by indication
Birth	Intrapartum antibiotics	Agent; timing (for caesarean: before incision or after cord clamping)	Affects early colonisation
Birth	Rupture of membranes and chorioamnionitis	Duration of rupture; clinical or histological chorioamnionitis	Infection and inflammation exposure
Neonatal	First enteral feed	Age at first feed; own milk, donor milk or formula	Colonisation; formula feeding increases NEC risk
Neonatal	Early antibiotics and probiotics	Days of empirical antibiotics before NEC; probiotic product and start date	Modifiable exposures
Diagnosis	NEC definition and stage	Staging system used; stage; isolated intestinal perforation recorded separately	Separates two distinct diseases

AREDF, absent or reversed end-diastolic flow; CHD, congenital heart disease; FGR, fetal growth restriction; NEC, necrotising enterocolitis.

The post-operative gut: a further disruption

Whatever the starting microbiome, a laparotomy for NEC adds to the damage. These babies have already had broad-spectrum antibiotics, prolonged bowel rest and often a period of profound illness; surgery adds resection, often a stoma, and further weeks of parenteral nutrition.

The limited data suggest that recovery of the microbiota is slow. In a prospective study of 13 preterm infants with surgical NEC and 15 matched controls, alpha diversity in the NEC group (11 of whom had an enterostomy) was still lower than in controls after full enteral feeding had been re-established.[58] Short bowel physiology then favours bacterial overgrowth, D-lactic acidosis and translocation; in a small study, *Enterobacteriaceae* dominated the faecal microbiota of four of five children with neonatal-onset short bowel syndrome who remained on parenteral nutrition.[59] The gut–liver axis, well described in adult fatty liver disease,[23] offers a plausible route to intestinal failure-associated liver disease: in children with intestinal failure,

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Proteobacteria-dominated microbiota have been linked to prolonged parenteral nutrition and histological liver steatosis.[60,61]

Stomas themselves bring complications that prolong this phase: in one 10-year series, 86% of infants with a stoma for NEC had at least one stoma complication, and prolapse occurred in 38% of all neonatal enterostomies.[62] Simple techniques that avoid another laparotomy have a place in reducing further surgical insult.[63] Earlier restoration of intestinal continuity, where safe, may also shorten the period of abnormal luminal ecology; the STAT data on parenteral nutrition are consistent with this, although the trial was not designed to study the microbiome.[54] Mucous fistula refeeding is used in many units with the same aim; in a meta-analysis of observational studies it was associated with about 37 fewer days of parenteral nutrition and less cholestasis (low-certainty evidence),[64] but its effect on the microbiota has not been studied.

Can we change the course? Microbiome-directed interventions

The interventions discussed below are summarised in Table 5.

Table 5 Microbiome-directed interventions and their status in surgical infants

Intervention	Evidence of benefit for NEC	Main concerns	Studied after NEC surgery?
Mother's own milk; donor milk when maternal milk is insufficient	Donor milk vs formula: NEC RR 0.53 (0.37–0.76); 11 trials, 2,261 infants; high certainty (Cochrane).[15] Benefit of mother's own milk supported by observational data	Supply; donor milk banking capacity	None identified
Probiotics	NEC RR 0.54 (0.46–0.65), low certainty; mortality RR 0.77 (0.66–0.90), moderate certainty; extremely preterm/ELBW infants: NEC RR 0.92 (0.69–1.22), low certainty (Cochrane)[14]	Product quality; invasive infection from probiotic organisms (FDA warning);[65] AAP: evidence does not support routine use, particularly below 1,000 g[66]	None identified
Maternal faecal microbiota transfer	No NEC data. In 7 term caesarean-born infants, microbiota resembled that of vaginally born infants from about day 7; no adverse effects to 3 months[67]	Pathogen transmission; intensive maternal screening	None identified
Vaginal seeding	No NEC data. Partial shift in microbiota development in one observational cohort;[68] no effect at 1 or 3 months in a pilot RCT of 25 infants.[69] Systematic review of 10 studies: efficacy uncertain[70]	Case report of possible HSV transmission[71]	None identified

Human milk. Mother's own milk remains the most dependable protection against NEC: in a whole-population study in England, infants born before 32 weeks who received it within the first week had a lower risk of severe NEC (RR 0.69, 95% CI 0.60–0.78),[72] and WHO makes a strong recommendation for it in preterm and low-birth-weight infants.[73] Where it is not available in sufficient quantity, donor human milk is preferable to formula; the 2024 Cochrane review found that donor milk, compared with formula, reduces the risk of NEC by about half (high-certainty evidence).[15] Human milk carries secretory IgA, oligosaccharides that selectively feed *Bifidobacterium*, and a microbial community of its own; in preterm infants, low concentrations of one oligosaccharide, disialyllacto-N-tetraose, in mother's own milk have been linked to NEC.[74] For a caesarean-born baby who missed the maternal inoculum at birth, early expressed colostrum is perhaps the simplest supportive measure available and one that needs no regulatory approval.

Probiotics. The 2023 Cochrane review found that probiotics may reduce the risk of NEC (RR 0.54, 95% CI 0.46–0.65; 57 trials, 10,918 infants; low-certainty evidence) and probably reduce mortality slightly (RR 0.77, 95% CI 0.66–0.90; moderate certainty).[14] In extremely preterm or extremely low birth weight infants, however, the group most at risk of surgical NEC, it found little or no effect on NEC (RR 0.92, 95% CI 0.69–1.22; low certainty).[14] Network meta-analyses suggest that combinations of *Lactobacillus* and *Bifidobacterium* species,[75] and multi-strain products more generally,[76] are the most effective. ESPGHAN has made conditional recommendations for specific strains,[77] and WHO conditionally recommends that probiotics be considered for human-milk-fed infants born before 32 weeks.[73] The American Academy of Pediatrics, however, concluded that the evidence does not support routine, universal use, particularly in babies below 1,000 g,[66] and in 2023 the US Food and Drug Administration warned of invasive, potentially fatal infection caused by organisms contained in probiotic products given to hospitalised preterm infants;[65] ESPGHAN and the European Foundation for the Care of Newborn Infants responded by calling for a more nuanced and supportive approach to the use of certain probiotics.[78] Since then, a propensity-matched national cohort of infants born before 32 weeks in England and Wales has reported fewer cases of severe NEC, confirmed at laparotomy or post-mortem, among those given probiotics (3.3% vs 4.2%; OR 0.80, 95% CI 0.72–0.89), including infants born before 28 weeks.[79] For surgeons the key gap is simple: almost none of these studies included babies after NEC surgery, and it is not known whether probiotics are safe or useful in a baby with a fresh anastomosis or a stoma.

Restoring maternal microbes after caesarean birth. The most direct approach to the caesarean problem is to give back what the baby missed. In a Finnish proof-of-concept study, 17 mothers were screened and seven were judged eligible; their term caesarean-born babies received a small dose of their mother's own faecal material in the first milk feed.[67] From about the first week the babies' microbiota resembled that of vaginally born infants and none had adverse effects over three months of follow-up.[67] 'Vaginal seeding', in which caesarean-born babies are swabbed with maternal vaginal fluid, partly restored vaginal-type bacterial communities in a first pilot study[80] and partly shifted the developmental trajectory of the microbiota in a larger observational cohort,[68] whereas a small randomised pilot of

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oral maternal vaginal microbes did not show a meaningful effect;[69] a later blinded trial in 68 infants reported faster gut microbiota maturation and better neurodevelopmental scores at 6 months.[81] These are experimental procedures in healthy term babies. They carry real infective risk, illustrated by a reported case of possible herpes simplex transmission after vaginal seeding,[71] and none has been tested in preterm or surgical infants. A 2026 systematic review of ten studies of maternal microbial transfer after elective caesarean delivery, four of them randomised trials, concluded that its efficacy in restoring the neonatal microbiota and improving health outcomes remains uncertain; only three of the included studies reported any neonatal clinical outcome, and we found no study of maternal microbial transfer that examined NEC.[70] They should not be offered outside research protocols, as ACOG has advised for vaginal seeding.[82] A less invasive option is probiotic supplementation: in a double-blind trial, a multispecies probiotic eliminated or reduced the birth-mode differences in the infant microbiota, but only in infants who were at least partly breastfed.[83]

Implications for counselling and practice

What should we say to a mother who asks whether her planned caesarean will harm her baby's gut? On the present evidence, an honest answer is that birth by caesarean section changes the bacteria a baby first acquires, that these differences largely diminish during the first year, and that there is no convincing evidence that caesarean birth, on its own, causes NEC in a healthy term baby. Where a caesarean is medically indicated, the benefits of timely delivery outweigh any theoretical microbial cost. Breastfeeding, early skin-to-skin contact and avoiding unnecessary antibiotics are practical steps that support healthy colonisation whatever the route of birth.

This illustrates a broader challenge in perinatal medicine: a new finding presented without context can generate more anxiety than benefit. Frameworks that separate management, outcome, risk and expectation can help clinicians keep counselling proportionate.[11,12] The balance may be different for specific high-risk groups. For a fetus with a prenatally diagnosed ductal-dependent cardiac lesion, where planned caesarean delivery is common and a link with NEC has been reported,[37] the question of delivery mode deserves more deliberate discussion among the obstetric, cardiac and surgical teams than it usually receives.

For the surgeon, three practical points follow. First, delivery mode, labour status and the indication for caesarean should be recorded in every surgical NEC database. Second, antibiotic stewardship and early human milk feeding should be treated as part of peri-operative care, not only as neonatal concerns: in preterm infants without culture-proven sepsis, longer courses of empirical antibiotics have been associated with higher odds of NEC or death[84] and of death or major morbidity.[85] Third, when a baby with NEC comes to theatre, the choice between drainage, stoma and primary anastomosis, and the timing of stoma closure, affect how long the gut remains in an abnormal state, and that consideration belongs in operative decision-making.

A unifying model

We find it useful to think of NEC as the product of successive hits rather than a single cause (Figure 5). The first hit falls before and at birth: the obstetric indication, the mode of delivery and intrapartum antibiotics. The second falls in the first weeks of NICU care: hospital colonisers, empirical antibiotics and delayed or formula feeding. Together these can produce a dysbiotic, Proteobacteria-dominated community in an immature gut, which in a proportion of babies progresses to NEC and, in some, to perforation. Surgery then delivers a third hit, with its own prolonged dysbiosis. Caesarean birth sits early in this chain. Its independent weight is probably small in preterm babies, in whom the obstetric indication and NICU exposures dominate, and may be larger in selected term populations; because it acts first, however, it may shape what follows.

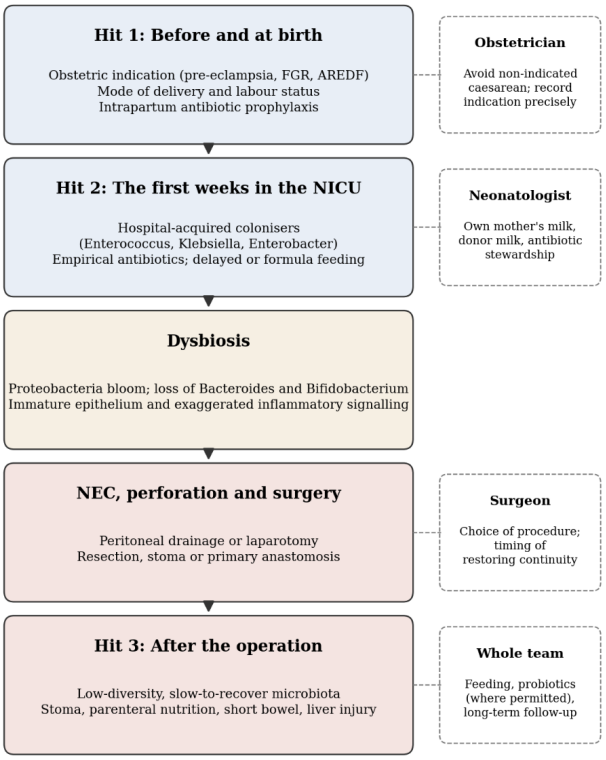


Figure 5 Proposed multiple-hit model linking birth, neonatal care, necrotising enterocolitis (NEC) and surgery.

First hit (before and at birth): the obstetric indication (for example, pre-eclampsia or fetal growth restriction with abnormal umbilical Doppler flow), mode of delivery, labour status and intrapartum antibiotics shape the initial microbial inoculum. Second hit (first weeks of neonatal intensive care): colonisation by hospital-associated organisms, empirical antibiotics and delayed or formula feeding favour a low-diversity, Proteobacteria-dominated community in an immature, hyper-reactive intestine, from which NEC and, in some infants, perforation may develop. Third hit (surgery): resection, stoma formation, further antibiotics and prolonged parenteral nutrition extend the dysbiosis into the post-operative period. Potentially modifiable points include mother's own or donor milk, antibiotic stewardship, probiotics in selected infants, the choice of operation and the timing of restoration of intestinal continuity. The model is a hypothesis-generating framework: the weight of each hit, and in particular the independent contribution of caesarean birth, has not been quantified and probably differs between preterm and term infants.

Gaps and future directions

Several questions deserve priority. We need prospective studies that separate elective from emergency caesarean section, and pre-labour from in-labour delivery, and that account properly for the obstetric indication, with the adjustment set pre-specified in a causal diagram that also makes explicit the selection bias introduced by restricting analyses to very preterm or very low birth weight infants,[47] and with analyses framed as emulations of a target trial;[56, 57] the recent signal around labour exposure makes this distinction more than academic.[36]

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We need microbiome sampling at the moments that matter to surgeons: at diagnosis, at laparotomy, from stoma effluent and before closure. Trials of probiotics and human milk strategies should include infants recovering from NEC surgery rather than excluding them. Finally, most of the existing literature comes from high-income countries. India, with its high and unevenly distributed caesarean rates,[6] large preterm population and heavy burden of neonatal sepsis, much of it caused by Gram-negative organisms with high rates of multidrug resistance,[9,10] is well placed to contribute studies that would change practice in India and elsewhere.

Box 1. Perspective from India

Provisional NFHS-6 data show that 27.2% of births in India are now by caesarean section, rising to 54.1% in private facilities against 16.9% in public facilities, and exceeding half of all births in Telangana and Andhra Pradesh.[7] Indian neonatal units carry a heavy burden of hospital-associated Gram-negative organisms: in the Delhi Neonatal Infection Study, 64% of sepsis isolates were Gram-negative, and multidrug resistance was found in 82% of *Acinetobacter* and 54% of *Klebsiella* isolates.[9] For many preterm babies, this second hit may outweigh the route of birth. Human milk is the most dependable protection, yet of 16 Indian human milk banks surveyed, only two were government-funded and 63% reported gaps between supply and demand.[86] Probiotic benefit is strain-specific: a recent Indian randomised trial of single-strain *Lactobacillus rhamnosus* GG in very low birth weight infants showed no reduction in sepsis, NEC or death.[87]

Limitations of this review

This is a narrative rather than a systematic review. Although three databases were searched and supplemented by citation chasing, study selection was not exhaustive, may have been shaped by our surgical perspective, and relevant studies may have been missed. Only articles published in English were included. We did not formally assess the risk of bias of individual studies or pool their estimates; certainty labels were derived from study design and, where available, from published GRADE assessments. NEC was defined inconsistently across studies, and modified Bell staging, other definitions and isolated intestinal perforation were not always distinguished,[88] which limits comparison between studies. Most microbiome data come from term infants in high-income countries, and few clinical studies separated elective from emergency, or pre-labour from in-labour, caesarean section. Finally, the review cites some previous work by the authors; these studies were given no more weight than other evidence of similar design.

Conclusions

The biological case that caesarean birth alters the neonatal gut is strong. The clinical case that it causes NEC is not. In preterm infants, confounding by the indication for caesarean, compounded by the selection bias inherent in cohorts restricted to very preterm or very low birth weight infants, probably explains more of the reported associations than the operation itself. The association appears to strengthen with increasing gestational age and is clearest in term babies, particularly those with congenital heart disease, in whom it deserves prospective study. For surgical NEC, the relationship between birth mode and outcome has scarcely been studied. For now, the most useful thing a paediatric surgeon can do is to treat dysbiosis as a continuous process that begins before birth and continues well after the laparotomy, and to work with obstetric and neonatal colleagues at each point where it can be changed, including by ensuring that surgical NEC registries capture the obstetric data needed to answer the question.

In answer to the first question, caesarean birth has not been shown to increase the incidence of NEC in preterm infants, whereas higher odds have been reported at term, including in neonates with congenital heart disease, and birth without labour is an emerging but unconfirmed signal. Second, whether mode of delivery influences the severity of NEC, progression to perforation or the need for surgery is unknown, because no adequately powered study has examined it. Third, post-operative outcome has not been studied in relation to mode of delivery, and the only available data suggest that gut microbial diversity remains reduced for weeks after surgery for NEC.

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Conflict of interest

The authors declare no conflict of interest.

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