



Early-onset group B streptococcal disease: Rethinking intrauterine and intrapartum transmission pathways

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ABSTRACT

Article info:

Received: 29 Jul 2026

Accepted: 16 Sep 2026

Keywords:

Group B Streptococcus
Early-onset disease
Intrauterine infection
Intrapartum transmission
Neonatal sepsis

Early-onset group B streptococcal disease (EOGBS) remains an important cause of invasive neonatal infection and is traditionally attributed predominantly to vertical transmission occurring during labor or following rupture of membranes. Although this model provides the basis for current screening and intrapartum antibiotic prophylaxis strategies, increasing clinical, pathological, experimental, and molecular observations have raised the possibility that fetal exposure to Group B Streptococcus (GBS) may occur before the onset of labor in a subset of pregnancies. In this narrative review, the biological and clinical pathways linking maternal GBS colonization to neonatal infection are examined, with particular attention to the temporal distinction between intrauterine and intrapartum transmission. Evidence concerning ascending infection, intraamniotic exposure, placental and fetal inflammatory responses, and molecular characterization of matched maternal, intraamniotic, placental, and neonatal isolates is considered. The established intrapartum transmission model and the evidence supporting its prevention through intrapartum antibiotic prophylaxis are also reviewed. The strengths and limitations of the available evidence for intrauterine acquisition are discussed, with emphasis on the difficulty of distinguishing antenatal infection from exposure occurring immediately before or during delivery. Finally, the potential implications of a broader temporal model for prevention are considered, including the complementary roles of intrapartum prophylaxis, antenatal risk recognition, and maternal vaccination. By integrating these perspectives, this review aims to provide a framework for understanding EOGBS as a potentially heterogeneous process in which maternal colonization, ascending infection, peripartum exposure, and neonatal invasion may occur along a continuum rather than as mutually exclusive transmission pathways.

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<https://jcbior.com>

<https://doi.org/10.66224/jcbior.7.3.366>

1. Introduction

1.1 Clinical importance of GBS

Group B *Streptococcus* (GBS), or *Streptococcus agalactiae*, is an important cause of invasive infection during pregnancy and the neonatal period. The organism commonly colonizes the gastrointestinal and genitourinary tracts of healthy individuals, and maternal rectovaginal colonization represents the principal recognized risk factor for neonatal early-onset disease (EOD) [1]. Although colonization is frequently asymptomatic, maternal carriage can be associated with transmission to the fetus or newborn and, in some circumstances, with maternal upper genital tract infection, intraamniotic infection, preterm birth, and other adverse pregnancy outcomes. Current epidemiological evidence indicates that maternal GBS colonization remains common worldwide, although prevalence varies substantially between populations. A recent systematic review of matched maternal-neonatal pairs estimated pooled maternal colonization at 17.1% and demonstrated a markedly increased likelihood of neonatal colonization among infants born to colonized mothers [2].

1.2 Conventional model of EOGBS transmission

Early-onset group B streptococcal disease (EOGBS) is conventionally defined by invasive disease occurring during the first six days of life and is predominantly associated with maternal colonization and vertical transmission around the time of labor and delivery. In the established model, GBS present in the maternal genital tract is exposed to the fetus or newborn during labor, particularly following membrane rupture, and may subsequently colonize the neonatal respiratory or mucosal surfaces before progressing to bacteremia and sepsis [1,3]. This model provides the biological rationale for antenatal screening and intrapartum antibiotic prophylaxis (IAP), which remains the principal strategy for reducing EOGBS. Contemporary obstetric guidance continues to describe transmission during labor or following rupture of membranes as the predominant pathway [1,4].

1.3 Why reconsider the timing of transmission?

The conventional intrapartum model, however, does not necessarily imply that all EOGBS infections are acquired after the onset of labor. Increasing clinical, microbiological, and pathological observations indicate that GBS may reach the intraamniotic compartment before delivery in a subset of pregnancies. Intraamniotic GBS infection and chorioamnionitis have been documented, while neonatal cases associated with fetal inflammatory responses or clinical evidence of infection before birth suggest that the biological process may sometimes begin antenatally [5,6]. More recently,

comparative genomic analysis has provided preliminary molecular evidence linking GBS recovered from the maternal vaginal ecosystem, amniotic fluid, chorioamniotic membranes, and neonatal blood in an individual case of early neonatal sepsis. These observations do not establish that intrauterine acquisition accounts for a large proportion of EOGBS, but they support consideration of EOGBS as a potentially heterogeneous process in which the timing of microbial invasion may vary [7].

This narrative review aims to critically examine the currently recognized pathways leading from maternal GBS colonization to early-onset neonatal disease, with particular emphasis on the timing and biological plausibility of intrauterine and intrapartum transmission. The review is intended to clarify the evidence supporting ascending GBS infection and fetal or intraamniotic exposure before labor, while considering these observations alongside the well-established intrapartum transmission model. The potential implications of this framework for current and emerging prevention strategies, including intrapartum antibiotic prophylaxis, antenatal risk assessment, and maternal vaccination, are also considered.

2. EOGBS: From maternal colonization to neonatal infection

2.1 Maternal colonization as the starting point

Maternal colonization is the principal epidemiological starting point for most recognized cases of perinatal GBS transmission. Colonization is dynamic rather than uniformly persistent, and detection depends partly on the timing and method of sampling. The 2024 meta-analysis of matched maternal-neonatal pairs estimated that approximately 4.5% of maternal-neonatal pairs experienced documented vertical transmission, while neonatal colonization was substantially more frequent among infants born to colonized mothers than among those born to non-colonized mothers [2]. These findings indicate that maternal carriage is an important prerequisite for transmission in many cases but also demonstrate that colonization and invasive disease are not equivalent events. Most colonized mothers do not give birth to infants who develop invasive EOGBS.

2.2 From colonization to fetal or neonatal exposure

Transmission from maternal colonization to neonatal disease involves several biological steps. GBS may ascend from the lower genital tract into the upper genital tract and intraamniotic compartment, or the fetus may encounter the organism during labor and delivery. After exposure, the organism must overcome epithelial and innate immune defenses before establishing invasive infection. Prematurity, prolonged rupture of

membranes, intraamniotic infection, and other maternal or obstetric factors may modify this process. Importantly, the distinction between exposure and invasive infection is critical: detection of GBS in maternal or neonatal specimens demonstrates colonization or transmission, whereas bacteremia, pneumonia, meningitis, or other invasive manifestations indicate progression beyond simple exposure [3,6,8].

2.3 Clinical spectrum

EOGBS most commonly presents as a rapidly progressive systemic infection, with sepsis and respiratory disease among the principal clinical manifestations. Severe disease may develop shortly after birth, and some affected neonates demonstrate physiological abnormalities that are compatible with fetal infection already occurring around the time of delivery [9,10]. In a cohort of neonates with culture-confirmed early-onset GBS sepsis, most cases were diagnosed during the first hour of life, and affected neonates had higher frequencies of preterm delivery, low Apgar scores, severe umbilical cord acidemia, fetal distress, and clinical chorioamnionitis than comparison neonates [11].

3. Intrauterine GBS infection: Emerging evidence and biological plausibility

3.1 Ascending infection and intraamniotic exposure

An intrauterine pathway requires GBS to move beyond maternal colonization and reach the fetal environment before delivery. Ascending infection from the lower genital tract represents a biologically plausible mechanism, particularly when the normal barriers of the cervicovaginal environment are altered. GBS has been identified in association with intraamniotic infection and inflammation, and clinical observations have linked GBS-associated intraamniotic infection with adverse maternal and neonatal outcomes. The possibility of ascending infection is also consistent with the observation that GBS can be associated with preterm labor, rupture of membranes, and chorioamnionitis. Nevertheless, the frequency with which this pathway results in invasive fetal infection remains incompletely defined [6,12,13].

Recent molecular evidence provides an additional perspective. In a 2024 study, whole-genome and comparative genomic analyses were performed on GBS isolates obtained from the maternal vaginal ecosystem, amniotic fluid, chorioamniotic membranes, and neonatal blood in a case of early neonatal sepsis. The investigators reported highly related organisms across these anatomical compartments, supporting the possibility of an ascending route from the maternal genital tract into the intraamniotic environment and subsequently to the newborn [7].

3.2 Placental and fetal inflammatory responses

Intraamniotic exposure to GBS may generate maternal and fetal inflammatory responses before delivery. Clinical chorioamnionitis, intraamniotic inflammation, and fetal inflammatory activation have all been associated with microbial exposure during pregnancy. Experimental work has additionally demonstrated that GBS-induced placental inflammation can influence fetal development and inflammatory injury, although experimental observations cannot be directly equated with the clinical course of human EOGBS. The biological significance of this inflammatory response may therefore extend beyond the simple presence or absence of bacteremia at birth [12,14].

Evidence from clinical cohorts also supports a relationship between EOGBS and inflammatory conditions surrounding delivery. The high frequency of chorioamnionitis among neonates with early-onset GBS sepsis in observational studies suggests that at least some infections may involve substantial fetal or intraamniotic exposure before or during labor [11]. However, clinical chorioamnionitis is not synonymous with proven fetal infection, and its presence alone cannot determine whether microbial invasion occurred hours before delivery or immediately during the intrapartum period [15].

3.3 What constitutes evidence for intrauterine infection?

The term “intrauterine infection” should therefore be applied cautiously. The strongest evidence would involve concordant microbiological identification of the same organism in maternal genital specimens, intraamniotic fluid or placental tissues, and fetal or neonatal samples, preferably supported by molecular characterization. Placental histopathology and evidence of fetal inflammatory responses may strengthen the interpretation but do not necessarily establish microbial invasion by themselves.

Conversely, a positive neonatal blood culture obtained shortly after birth cannot, in isolation, distinguish between acquisition immediately before delivery and acquisition during labor [7,11,12]. This distinction is important because the timing of infection has implications for prevention. If GBS invasion has already occurred before labor, an intervention administered only during labor may have limited ability to reverse established fetal infection. Conversely, the established effectiveness of IAP in reducing EOGBS supports the conclusion that a substantial proportion of disease remains preventable by interrupting transmission around delivery. The available evidence therefore favors a model in which intrauterine and intrapartum exposure may represent overlapping points along a temporal continuum rather than mutually exclusive categories [14,16].

4. Intrapartum transmission: The established pathway

4.1 Exposure during labor and delivery

Intrapartum transmission remains the best-established pathway for EOGBS. Maternal colonization of the vagina and rectum provides the immediate reservoir, and exposure increases during labor and after rupture of membranes. The organism may contaminate the newborn during passage through the birth canal or may ascend following membrane rupture. The strong association between maternal colonization and neonatal colonization, together with the effectiveness of IAP, provides substantial support for this model [2,3,16].

4.2 Neonatal colonization to invasive disease

Exposure does not invariably result in invasive infection. After contact with GBS, the neonate must progress from mucosal colonization to tissue invasion and bloodstream infection [17]. Host susceptibility, bacterial characteristics, and the intensity and duration of exposure may influence this transition [18]. Premature and very-low-birth-weight infants are particularly vulnerable, although term infants also develop EOGBS. The clinical phenotype can progress rapidly, which is consistent with a short interval between acquisition and invasive disease in some neonates [3,8,19].

4.3 Evidence supporting the intrapartum model

The principal evidence supporting intrapartum transmission is epidemiological and interventional. Maternal GBS colonization is associated with neonatal colonization, and IAP substantially reduces the occurrence of EOGBS. In a large observational analysis, β -lactam prophylaxis administered for at least four hours before delivery was associated with high effectiveness against EOGBS, although shorter exposure was less effective [14]. Contemporary guidelines consequently continue to recommend IAP for women meeting established indications rather than replacing it with strategies directed exclusively at antenatal or postnatal periods [16].

5. Rethinking EOGBS: Intrauterine versus intrapartum transmission

5.1 Timing and biological pathway

The available evidence suggests that EOGBS should not necessarily be conceptualized as a single transmission event occurring exclusively during labor. Instead, maternal colonization may precede an ascending intrauterine process in some pregnancies, whereas in others exposure may occur predominantly during labor and delivery. The distinction is therefore temporal as well as anatomical. An intrauterine pathway

implies microbial access to the fetal environment before birth, while the intrapartum pathway primarily involves exposure around labor, membrane rupture, and delivery. Current evidence is insufficient to assign precise proportions of EOGBS to either pathway [6-8,11,12].

5.2 Clinical consequences

The timing of exposure may influence the clinical phenotype. Antenatal infection may contribute to intraamniotic inflammation, preterm labor, fetal inflammatory responses, or fetal infection before delivery [18,20]. Intrapartum acquisition may instead result in neonatal colonization followed by rapid progression to bacteremia or respiratory disease after birth. These categories should not, however, be interpreted as clinically discrete syndromes. A fetus exposed shortly before delivery may develop findings that resemble both antenatal infection and conventional intrapartum EOGBS [11,21].

5.3 Strength and limitations of the evidence

The evidence supporting intrapartum transmission is considerably stronger at the population level because it is supported by epidemiological associations and intervention studies demonstrating that IAP reduces EOGBS [14]. Evidence for intrauterine transmission is biologically and clinically credible but more heterogeneous. It includes cases of intraamniotic infection, associations with chorioamnionitis, pathological observations, experimental models, and recent molecular characterization of matched isolates. The latter provides an important proof-of-concept but cannot establish the prevalence of this pathway. Consequently, the intrauterine hypothesis should be regarded as a complementary explanation for a subset of EOGBS rather than as a replacement for the established intrapartum model [7,12,14].

5.4 A spectrum rather than mutually exclusive pathways

A spectrum model may better accommodate the available evidence. Maternal colonization can persist without fetal exposure, progress to ascending intrauterine infection, or remain localized until labor provides an opportunity for neonatal exposure. Between these extremes, infection may begin shortly before delivery and become clinically apparent immediately after birth. This conceptual continuum also explains why the distinction between intrauterine and intrapartum infection can be difficult to establish retrospectively using routine clinical specimens. The practical value of the distinction therefore lies less in assigning every case to a single category and more in identifying which preventive strategies address exposure before labor and which interrupt transmission during delivery [6,7]. The available evidence supporting intrauterine and intrapartum transmission pathways is summarized in Table 1.

Table 1. Evidence supporting intrauterine and intrapartum transmission pathways in early-onset group B streptococcal disease

Feature	Intrauterine transmission	Intrapartum transmission
Timing of exposure	Before delivery, potentially before onset of labor	Mainly during labor and delivery, particularly after rupture of membranes
Proposed pathway	Ascending spread from the lower genital tract to the upper genital tract and intraamniotic compartment	Exposure to GBS in the maternal genital tract during labor, delivery, or after membrane rupture
Evidence described in the review	Intraamniotic infection, chorioamnionitis, fetal inflammatory responses, pathological observations, experimental findings, and molecular characterization of matched isolates	Epidemiological association between maternal colonization and neonatal colonization, together with evidence from intervention studies
Molecular evidence	Highly related GBS isolates identified in maternal vaginal ecosystem, amniotic fluid, chorioamniotic membranes, and neonatal blood in a reported case	Molecular evidence is not presented as the principal basis for this pathway
Clinical associations	Intraamniotic inflammation, chorioamnionitis, preterm labor, fetal inflammatory responses, and possible fetal infection before delivery	Rapid neonatal colonization and progression to invasive disease after exposure around delivery
Strength of current evidence	Biologically and clinically credible, but heterogeneous and insufficient to determine its population-level frequency	Stronger population-level evidence supported by epidemiological and interventional studies
Main limitation	Difficult to distinguish antenatal infection from exposure occurring immediately before or during delivery	Cannot explain every EOGBS case, particularly when infection may have been established before labor
Prevention implications	May require prevention or recognition strategies acting before labor, including maternal vaccination and improved antenatal risk recognition	Supports continued use of screening and intrapartum antibiotic prophylaxis
Overall interpretation	Considered a complementary pathway that may account for a subset of cases	Remains the established and predominant transmission pathway

6. Implications for prevention

6.1 What IAP can and cannot prevent

The continuing effectiveness of IAP supports its role as the principal intervention for preventing transmission occurring during labor and delivery. Current obstetric guidance recommends universal screening at 36 0/7–37 6/7 weeks of gestation and administration of appropriate prophylaxis to women with positive screening results or established clinical indications [3].

Nevertheless, IAP cannot be expected to prevent every case of EOGBS. Infection that has already developed before labor, rapidly progressing infection in which there is insufficient time for prophylaxis, and cases arising when maternal colonization is not detected remain potential limitations of a delivery-centered strategy [14].

6.2 Maternal vaccination and antenatal prevention

The possibility of antenatal GBS infection provides a rationale for preventive strategies acting before labor. Maternal vaccination is particularly relevant because transplacentally transferred antibodies could potentially provide protection during the period in which the fetus is exposed and during the early neonatal period. Vaccine development remains under clinical investigation, and current evidence supports continued evaluation rather than assuming that vaccination will replace established screening and IAP strategies [22,23].

More broadly, improved antenatal identification of women at risk for intraamniotic infection may eventually complement delivery-based prevention, although evidence for specific approaches remains under development [6,12].

7. Conclusion

EOGBS is traditionally understood as a vertically transmitted infection acquired predominantly during labor or after rupture of membranes. This model remains strongly supported by epidemiological and interventional evidence and provides the basis for current screening and IAP strategies. At the same time, clinical, pathological, experimental, and emerging molecular observations indicate that GBS can reach the intraamniotic environment before delivery and may occasionally contribute to fetal infection before the onset of labor. The present evidence does not justify replacing the established intrapartum model with an exclusively intrauterine explanation. Rather, it supports viewing EOGBS as a temporal continuum in which maternal colonization, antenatal ascending infection, peripartum exposure, and neonatal invasion may interact. Defining the relative contribution of these pathways remains an important research priority because a clearer understanding of when fetal exposure occurs could identify opportunities for prevention that are not addressed by intrapartum prophylaxis alone.

Declaration of artificial intelligence (AI) in the writing process

The authors declare whether AI or AI-assisted technologies were used during the preparation of this manuscript. If used, AI tools were employed solely to improve language quality, grammar, readability, and organizational structure. The authors carefully reviewed and edited all AI-generated content and take full responsibility for the accuracy, integrity, and originality of the final manuscript. No AI tool was used to generate, analyze, or interpret scientific data or images, or to draw scientific conclusions. The use of AI-assisted

technologies complies with current publication ethics recommendations and journal policies.

Authors' contributions

Conceptualization, Methodology, Supervision, Project administration, Writing - review & editing: RS, EK, SK. Investigation, Data curation, Formal analysis, Writing - original draft: FA, NSP, SN, RK, EMJ. All authors have read and approved the final version of the manuscript.

Conflict of interest

No potential conflict of interest was reported by the authors.

Ethical declarations

Not applicable.

Financial support

Self-funded.

References

- Delara M, Vadlamudi NK, Sadarangani M. Strategies to Prevent Early and Late-Onset Group B Streptococcal Infection via Interventions in Pregnancy. *Pathogens*. 2023;12(2):229. DOI: [10.3390/pathogens12020229](https://doi.org/10.3390/pathogens12020229) PMID: [36839501](https://pubmed.ncbi.nlm.nih.gov/36839501/)
- Lee KW, Yap SF, Murdan S, Zainudin Z, Abdul Hamid H, Emamjomeh M, et al. Maternal and neonatal group B streptococcus colonisation: A systematic review and the meta-analysis of matched-pair studies. *Acta Paediatr*. 2024;113(5):892-911. DOI: [10.1111/apa.17152](https://doi.org/10.1111/apa.17152) PMID: [38351491](https://pubmed.ncbi.nlm.nih.gov/38351491/)
- Prevention of Group B Streptococcal Early-Onset Disease in Newborns: ACOG Committee Opinion, Number 797. *Obstet Gynecol*. 2020;135(4):978-979. DOI: [10.1097/AOG.0000000000003824](https://doi.org/10.1097/AOG.0000000000003824) PMID: [31977795](https://pubmed.ncbi.nlm.nih.gov/31977795/)
- Campbell P, Broderick C, Fenton C, Davis B, Xu W, Todhunter-Brown A, et al. Benefits and Harms of Antenatal/Intrapartum Screening for Maternal Group B Streptococcus and Use of Intrapartum Antibiotic Prophylaxis Versus Risk-Based Protocols or No Intervention: A Rapid Review. *Acta Paediatr*. 2026;115(8):1598-1610. DOI: [10.1111/apa.70568](https://doi.org/10.1111/apa.70568) PMID: [42060466](https://pubmed.ncbi.nlm.nih.gov/42060466/)
- Consonni S, Salmoiraghi E, Vaglio T, Pintucci A, Vitale V, Calzi P, et al. Intra-Amniotic Inflammation or Infection: Suspected and Confirmed Diagnosis of "Triple I" at Term. *Children (Basel)*. 2023;10(7):1110. DOI: [10.3390/children10071110](https://doi.org/10.3390/children10071110) PMID: [37508607](https://pubmed.ncbi.nlm.nih.gov/37508607/)
- Manuel G, Twentyman J, Noble K, Eastman AJ, Aronoff DM, Seepersaud R, et al. Group B streptococcal infections in pregnancy and early life. *Clin Microbiol Rev*. 2025;38(1):e0015422. DOI: [10.1128/cmr.00154-22](https://doi.org/10.1128/cmr.00154-22) PMID: [39584819](https://pubmed.ncbi.nlm.nih.gov/39584819/)
- Pongchaikul P, Romero R, Wongsurawat T, Jenjaroenpun P, Kruasuwat W, Mongkolsuk P, et al. Molecular evidence that GBS early neonatal sepsis results from ascending infection: comparative hybrid genomics analyses show that microorganisms in the vaginal ecosystem, amniotic fluid, chorioamniotic membranes, and neonatal blood are the same. *J Perinat Med*. 2024;52(9):977-990. DOI: [10.1515/jpm-2024-0310](https://doi.org/10.1515/jpm-2024-0310) PMID: [39405032](https://pubmed.ncbi.nlm.nih.gov/39405032/)
- Mei JY, Silverman NS. Group B Streptococcus in Pregnancy. *Obstet Gynecol Clin North Am*. 2023;50(2):375-387. DOI: [10.1016/j.ogc.2023.02.009](https://doi.org/10.1016/j.ogc.2023.02.009) PMID: [37149317](https://pubmed.ncbi.nlm.nih.gov/37149317/)
- Tavares T, Pinho L, Bonifácio Andrade E. Group B Streptococcal Neonatal Meningitis. *Clin Microbiol Rev*. 2022;35(2):e0007921. DOI: [10.1128/cmr.00079-21](https://doi.org/10.1128/cmr.00079-21) PMID: [35170986](https://pubmed.ncbi.nlm.nih.gov/35170986/)
- Heath PT, Jardine LA. Neonatal infections: group B streptococcus. *BMJ Clin Evid*. 2014;2014:0323. PMID: [24580886](https://pubmed.ncbi.nlm.nih.gov/24580886/)
- Tudela CM, Stewart RD, Roberts SW, Wendel GD Jr, Stafford IA, McIntire DD, et al. Intrapartum evidence of early-onset group B streptococcus. *Obstet Gynecol*. 2012;119(3):626-9. DOI: [10.1097/AOG.0b013e31824532f6](https://doi.org/10.1097/AOG.0b013e31824532f6) PMID: [22353962](https://pubmed.ncbi.nlm.nih.gov/22353962/)
- Afsari M, White A, Adhikari EH. Group B Streptococcus and Intraamniotic Inflammation and Infection. *Clin Obstet Gynecol*. 2024;67(3):576-588. DOI: [10.1097/GRF.0000000000000884](https://doi.org/10.1097/GRF.0000000000000884) PMID: [39061126](https://pubmed.ncbi.nlm.nih.gov/39061126/)
- Vornhagen J, Adams Waldorf KM, Rajagopal L. Perinatal Group B Streptococcal Infections: Virulence Factors, Immunity, and Prevention Strategies. *Trends Microbiol*. 2017;25(11):919-931. DOI: [10.1016/j.tim.2017.05.013](https://doi.org/10.1016/j.tim.2017.05.013) PMID: [28633864](https://pubmed.ncbi.nlm.nih.gov/28633864/)
- Fairlie T, Zell ER, Schrag S. Effectiveness of intrapartum antibiotic prophylaxis for prevention of early-onset group B streptococcal disease. *Obstet Gynecol*. 2013;121(3):570-577. DOI: [10.1097/AOG.0b013e318280d4f6](https://doi.org/10.1097/AOG.0b013e318280d4f6) PMID: [23635620](https://pubmed.ncbi.nlm.nih.gov/23635620/)
- Jung E, Romero R, Suksai M, Gotsch F, Chaemsaihong P, Erez O, et al. Clinical chorioamnionitis at term: definition, pathogenesis, microbiology, diagnosis, and treatment. *Am J Obstet Gynecol*. 2024;230(3S):S807-S840. DOI: [10.1016/j.ajog.2023.02.002](https://doi.org/10.1016/j.ajog.2023.02.002) PMID: [38233317](https://pubmed.ncbi.nlm.nih.gov/38233317/)
- Dhudasia MB, Flannery DD, Pfeifer MR, Puopolo KM. Updated Guidance: Prevention and Management of Perinatal Group B Streptococcus Infection. *Neoreviews*. 2021;22(3):e177-e188. DOI: [10.1542/neo.22-3-e177](https://doi.org/10.1542/neo.22-3-e177) PMID: [33649090](https://pubmed.ncbi.nlm.nih.gov/33649090/)
- Greenfield KG, Harlow OS, Witt LT, Dziekan EM, Tamar CR, Meier J, et al. Neonatal intestinal colonization of Streptococcus agalactiae and the multiple modes of protection limiting translocation. *Gut Microbes*. 2024;16(1):2379862. DOI: [10.1080/19490976.2024.2379862](https://doi.org/10.1080/19490976.2024.2379862) PMID: [39042143](https://pubmed.ncbi.nlm.nih.gov/39042143/)
- Daskalakis G, Psarris A, Koutras A, Fasoulakis Z, Prokopakis I, Varthaliti A, et al. Maternal Infection and Preterm Birth: From Molecular Basis to Clinical Implications. *Children (Basel)*. 2023;10(5):907. DOI: [10.3390/children10050907](https://doi.org/10.3390/children10050907) PMID: [37238455](https://pubmed.ncbi.nlm.nih.gov/37238455/)
- Benitz WE, Gould JB, Druzin ML. Risk factors for early-onset group B streptococcal sepsis: estimation of odds ratios by critical literature review. *Pediatrics*. 1999;103(6):e77. DOI: [10.1542/peds.103.6.e77](https://doi.org/10.1542/peds.103.6.e77) PMID: [10353974](https://pubmed.ncbi.nlm.nih.gov/10353974/)
- Chen X, Zhang X, Xue R, Mi L, Liu Y, Zhang G, et al. Intrauterine inflammation exposure may increase the risk of late-onset sepsis in premature infants: a retrospective cohort study. *Ital J Pediatr*. 2025;51(1):218. DOI: [10.1186/s13052-025-02040-5](https://doi.org/10.1186/s13052-025-02040-5) PMID: [40640885](https://pubmed.ncbi.nlm.nih.gov/40640885/)
- Allard MJ, Brochu ME, Bergeron JD, Segura M, Sébire G. Causal role of group B Streptococcus-induced acute chorioamnionitis in intrauterine growth retardation and cerebral palsy-like impairments. *J Dev Orig Health Dis*. 2019;10(5):595-602. DOI: [10.1017/S2040174418001083](https://doi.org/10.1017/S2040174418001083) PMID: [30626456](https://pubmed.ncbi.nlm.nih.gov/30626456/)
- Kobayashi M, Vekemans J, Baker CJ, Ratner AJ, Le Doare K, Schrag SJ. Group B Streptococcus vaccine development: present status and future considerations, with emphasis on perspectives for low and middle income countries. *F1000Res*. 2016;5:2355. DOI: [10.12688/f1000research.9363.1](https://doi.org/10.12688/f1000research.9363.1) PMID: [27803803](https://pubmed.ncbi.nlm.nih.gov/27803803/)
- Schrag SJ, Verani JR. Intrapartum antibiotic prophylaxis for the prevention of perinatal group B streptococcal disease: experience in the United States and implications for a potential group B streptococcal vaccine. *Vaccine*. 2013;31 Suppl 4:D20-6. DOI: [10.1016/j.vaccine.2012.11.056](https://doi.org/10.1016/j.vaccine.2012.11.056) PMID: [23219695](https://pubmed.ncbi.nlm.nih.gov/23219695/)