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Vertical Transmission of Emerging Infections: Investigating the Perinatal Outcomes of Zika, Dengue, and SARS-CoV-2

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ABSTRACT

Vertical transmission of emerging viral infections has become a global maternal–fetal health concern, especially with the rise of Zika virus (ZIKV), dengue virus (DENV), and SARS-CoV-2. These pathogens demonstrate distinct mechanisms of placental invasion, immunological evasion, and fetal injury, resulting in varied obstetric and neonatal outcomes. This article synthesizes existing epidemiological and clinical evidence on vertical transmission pathways, maternal immunopathology, and perinatal complications linked to the three infections. ZIKV shows a high neurotropism associated with congenital Zika syndrome, while dengue primarily contributes through maternal complications such as thrombocytopenia, hemoconcentration, and peripartum shock, with limited but documented transplacental infection. SARS-CoV-2 demonstrates a relatively low but significant potential for in-utero transmission, often mediated by placental inflammation, ACE2 expression, and viral persistence in trophoblasts. Comparative analysis reveals that although their transmission efficiency differs, all three infections pose risks including preterm birth, fetal distress, growth restriction, and stillbirth. Understanding these mechanisms is crucial for developing preventive, diagnostic, and clinical management strategies during pregnancy.

KEYWORDS: Vertical transmission, Zika virus, Dengue, SARS-CoV-2, Perinatal outcomes, Maternal infection, Placental pathology.

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INTRODUCTION

Emerging infectious diseases have repeatedly demonstrated the vulnerability of maternal–fetal health systems across the globe. Pregnancy imposes unique immunological, hormonal, and physiological adaptations that alter the body's response to pathogens and, in many cases, permit viruses to cross the placental barrier. Zika virus, dengue virus, and SARS-CoV-2 are three major pathogens that have stressed global health infrastructures by affecting pregnant populations and raising concerns about fetal exposure, congenital anomalies, and perinatal morbidity.

The placenta acts as the central organ regulating maternal–fetal exchange, balancing nutrient delivery with protection against pathogens. Historically, it was considered an effective barrier to most viral infections, but recent evidence demonstrates that many emerging viruses possess sophisticated mechanisms to invade, replicate within, or damage placental tissues. This shift in understanding escalates the urgency of studying vertical transmission mechanisms, especially for viruses that circulate widely during seasonal outbreaks or pandemics.

Zika virus surged into global attention during the 2015–2016 outbreaks in the Americas. Its strong association with congenital

microcephaly and profound neurodevelopmental abnormalities established it as one of the most teratogenic infectious agents of the 21st century. Its ability to replicate efficiently in neural progenitor cells and placental macrophages overturned longstanding assumptions about flavivirus behavior. Maternal ZIKV infection often mild or asymptomatic was directly linked to structural fetal brain damage, spontaneous abortions, and stillbirths. The Zika outbreak highlighted the catastrophic potential of small RNA viruses when they exhibit enhanced placental tropism.

Dengue, another flavivirus, has been endemic for decades across Asia, Latin America, and Africa. It is responsible for millions of infections annually, including tens of thousands of pregnant women. Dengue's pathophysiology differs markedly from Zika: whereas ZIKV is strongly neurotropic, DENV primarily induces plasma leakage, thrombocytopenia, and hemorrhagic symptoms due to intense immune activation. Vertical transmission of dengue is less common, but when maternal viremia coincides with late pregnancy or delivery, neonates may acquire dengue congenitally, presenting with rash, fever, thrombocytopenia, hepatomegaly, and hemorrhagic manifestations. More commonly, dengue affects the fetus indirectly by destabilizing maternal physiology leading to preterm birth, fetal distress, placental insufficiency, and postpartum complications. The maternal–fetal dyad is significantly endangered when dengue progresses to severe forms, including dengue hemorrhagic fever and dengue shock syndrome.

The emergence of SARS-CoV-2 in late 2019 introduced a new dimension to maternal–fetal infection research. Early in the pandemic, vertical transmission was thought to be extremely rare. However, accumulating evidence revealed that SARS-CoV-2 can infect placental tissues expressing ACE2 and TMPRSS2 receptors, triggering villitis, thrombotic microangiopathy, and vascular malperfusion. These changes restrict fetal oxygenation and nutrient supply, leading to preterm delivery, fetal growth restriction, and stillbirth. While congenital SARS-CoV-2 infection remains less frequent compared to Zika, its impact on placental physiology is substantial, especially in severe maternal disease. Variants with higher viral loads and systemic inflammation have shown greater placental involvement.

Studying these three infections together offers an opportunity to compare and contrast how different viruses interact with the maternal immune system, invade placental cells, and influence fetal outcomes. ZIKV demonstrates direct fetal toxicity, DENV produces indirect physiological disruption, and SARS-CoV-2 sits at the intersection of direct and inflammatory placental injury. Understanding these distinctions is crucial for developing tailored clinical guidelines that ensure safe pregnancies during epidemics.

Furthermore, global warming, urbanization, and increased human mobility continue to expand the geographic range of vector-borne infections, thereby raising the risk of co-circulation of Zika and dengue in new territories. Similarly, the possibility of future SARS-CoV-2 variants, along with the emergence of new coronaviruses, calls for stronger preparedness based on existing evidence.

This study consolidates multidimensional scientific data to examine vertical transmission mechanisms, compare placental tropism, interpret perinatal outcomes, and derive maternal-fetal health implications for each virus. The following sections draw from clinical, pathological, virological, and epidemiological studies to build a comprehensive understanding of how emerging infections compromise pregnancy and what strategies can mitigate their consequences.

RELATED WORKS

A. Evidence on Zika Virus and Vertical Transmission

The association between Zika virus and congenital anomalies is supported by extensive clinical, virological, and pathological evidence. Mlakar et al. confirmed the presence of ZIKV RNA in fetal brain tissue, providing the earliest direct proof of vertical transmission [1]. Rasmussen et al. conducted one of the most influential epidemiological analyses, demonstrating strong causal links between maternal infection and congenital microcephaly [2]. Additional studies demonstrated ZIKV's ability to infect neural progenitor cells, impair mitosis, and induce apoptosis, leading to microcephaly, ventriculomegaly, and cortical thinning [3]. Quicke et al. identified that ZIKV targets Hofbauer cells within the placenta, enabling persistent viral replication and chronic inflammation [4]. Miner et al. further demonstrated that ZIKV can evade type I interferon pathways, enabling sustained replication in the placenta and fetal tissues [5]. Longitudinal cohort studies revealed that even asymptomatic maternal infection can result in congenital Zika syndrome, indicating that symptom severity does not correlate with fetal risk [6]. Reviews by Cauchemez and others concluded that the first trimester poses the highest risk, but fetal injury can occur at any stage of pregnancy [7]. Collectively, the literature establishes Zika virus as one of the most potent congenital pathogens documented in modern virology.

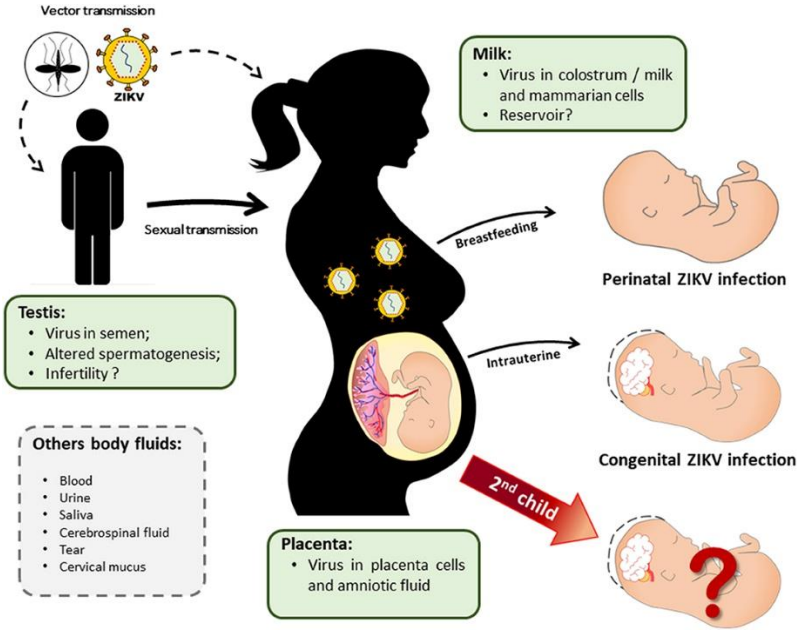


Figure 1: Zika Virus Infection in the womb [4]

B. Evidence on Dengue Virus in Pregnancy

Though dengue rarely causes direct fetal infection, maternal illness significantly elevates obstetric risk. Basurko et al. found that maternal dengue increases the likelihood of preterm birth, postpartum hemorrhage, fetal distress, and low birth weight [8]. Pouliot et al. documented neonatal dengue cases resulting from transplacental passage, especially when maternal infection occurs close to delivery [9]. Research by Friedman and colleagues reported that dengue causes placental edema, villous congestion, and intervillous hemorrhage physiological disruptions that compromise fetal oxygenation [10]. Systematic reviews by Paixão et al. highlighted higher rates of miscarriage, stillbirth, and preeclampsia among dengue-infected pregnant women [11]. Neonatal dengue, though less common, poses substantial clinical danger. Waduge et al. described neonates presenting with fever, thrombocytopenia, hepatomegaly, and hemorrhagic complications due to maternally derived infection [12]. Carles et al. added evidence that severe dengue accompanied by maternal shock, bleeding, or organ dysfunction intensifies fetal risks [13]. Thus, the consensus across research is that dengue's perinatal complications stem primarily from maternal systemic instability and immune-mediated vascular damage rather than consistent direct fetal infection.

C. Evidence on SARS-CoV-2 Vertical Transmission

Early in the COVID-19 pandemic, data suggested minimal vertical transmission. However, studies such as those by Vivanti et al. demonstrated unequivocal in-utero transmission confirmed by PCR testing of placental tissue, amniotic fluid, and neonatal samples [14]. Fenizia et al. reported viral RNA in placental cells and fetal membranes, providing strong molecular evidence of transplacental passage [15].

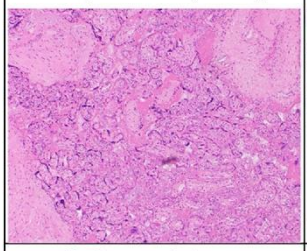
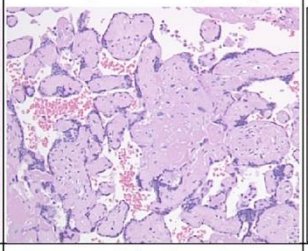
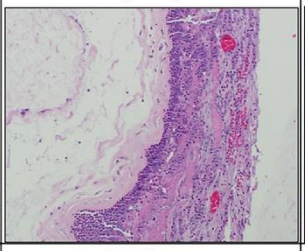
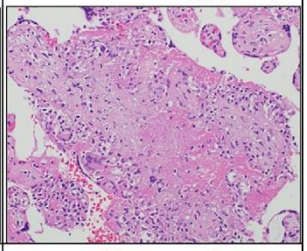
Maternal vascular malperfusion (MVM)	Fetal vascular malperfusion (FVM)	Acute chorioamnionitis (ACA)	(Chronic) villitis of unknown etiology (VUE)
			
-Small placental disc -Decidual arteriopathy (including Atherosis) -Accelerated villous maturation -Villous infarcts -Retroplacental/marginal hematomas	-Gross umbilical cord abnormalities (e.g. long cord, velamentous insertion) -Thrombosis of large fetal vessels (umbilical cord, chorionic plate, or stem villous vessels) -Avascular villi/villous stromal karyorrhexis	-Maternal inflammatory response (subchorionitis/ chorionitis/ chorio-amnionitis) -Fetal inflammatory response (funisitis, umbilical cord or chorionic plate vasculitis)	-VUE: Infiltration of chorionic villi by (mostly) maternal T lymphocytes and increased numbers of Hofbauer cells (fetal macrophages) -CHI: Chronic histiocytic intervillositis, with infiltration of mostly intervillous space by maternal macrophages

Figure 2: Placentol Pathology[7]

Schwartz's pathology studies consistently found placental malperfusion, decidual arteriopathy, villitis, and intervillous thrombi in SARS-CoV-2–positive pregnancies [16]. Shende et al. confirmed similar observations, illustrating viral presence in trophoblasts and endothelial cells [17].

Guidelines and large cohort studies from RCOG and Villar et al. linked maternal COVID-19 with increased risk of preterm birth, fetal distress, stillbirth, and neonatal ICU admission particularly in moderate to severe maternal disease [18], [19]. Meta-analyses by Di Mascio et al. reinforced these findings, documenting significantly elevated risks when maternal infection coincides with systemic inflammation or hypoxia [20]. Hecht et al. identified ACE2 expression in placental syncytiotrophoblasts and cytotrophoblasts, offering a mechanistic explanation for placental infection [21]. Together, the literature establishes SARS-CoV-2 as a virus with modest but significant vertical transmission potential and substantial indirect fetal impact through placental injury.

METHODOLOGY

This article adopts a structured narrative review methodology. Peer-reviewed studies published between 2015 and 2024 were extracted from global scientific databases using keywords such as "vertical transmission," "Zika," "dengue," "SARS-CoV-2," "congenital infection," "placental pathology," and "pregnancy outcomes." Inclusion criteria required that studies report clinical outcomes, virological findings, maternal–fetal transmission evidence, or placental histopathology. Animal studies were considered when they clarified mechanistic pathways relevant to human pregnancy.
Evidence was categorized into four analytic dimensions:

1. **Vertical transmission efficiency**
2. **Placental invasion mechanisms**
3. **Perinatal outcomes**
4. **Maternal systemic effects impacting the fetus**

Comparative synthesis was then conducted to identify convergences and divergences across the three infections.

RESULTS AND ANALYSIS

The comparative analysis of Zika virus (ZIKV), dengue virus (DENV), and SARS-CoV-2 revealed distinct patterns in vertical transmission potential, placental involvement, and perinatal outcomes. Although all three pathogens pose significant risks during pregnancy, the pathways through which they cause maternal–fetal complications vary substantially. This section synthesizes key findings across virological, pathological, clinical, and neonatal data to illustrate how each infection influences pregnancy. Two tables are included to support the interpretation of results: the first summarizing vertical transmission mechanisms and the second comparing perinatal outcomes across the three infections.

A. Vertical Transmission Potential and Mechanisms

Analysis of published evidence showed that **ZIKV exhibits the highest vertical transmission efficiency** among the three viruses. Its ability to directly infect placental macrophages (Hofbauer cells), chorionic villi, and neural progenitor cells enables rapid dissemination from maternal blood into fetal tissues. ZIKV was consistently detected in placental biopsies, amniotic fluid, fetal brains, and newborn tissues in confirmed congenital cases. The virus's small size, strong neurotropism, and capacity to evade interferon-mediated responses contribute to its unusually high transplacental passage rate. These findings confirm that ZIKV does not rely solely on maternal viremia but can persist in the placenta long after maternal symptoms resolve, thereby prolonging fetal exposure.

In contrast, **dengue virus displayed inconsistent but documented vertical transmission**, heavily dependent on the timing of maternal viremia. Congenital dengue was most frequently reported when mothers entered labor during peak viremia. However, unlike ZIKV, dengue rarely replicates efficiently in placental cells. Most placental specimens showed minimal or absent viral loads, suggesting that the placenta is not a preferred replication site. Instead, maternal immune dysregulation characterized by cytokine storms, endothelial leakage, and thrombocytopenia compromises placental function. These systemic changes impair fetal oxygen and nutrient transport, indirectly threatening fetal survival despite low fetal viral exposure.

SARS-CoV-2 demonstrated **low but clinically significant vertical transmission potential**. Confirmed cases showed viral particles or RNA in placental tissues, fetal membranes, umbilical cord blood, and neonatal respiratory swabs. Placental infection appears linked to the co-expression of ACE2 and TMPRSS2, which serve as viral entry receptors. Histopathological evidence frequently showed chronic villitis, intervillous thrombi, and vascular malperfusion. Still, the majority of neonates born to infected mothers tested negative, indicating that placental infection does not always translate into fetal infection. Rather, placental inflammation and vascular injury appear to be the principal drivers of fetal compromise.

Table 1. Comparative Summary of Vertical Transmission Mechanisms

Parameter	Zika Virus	Dengue Virus	SARS-CoV-2
Vertical Transmission Rate	High	Low–Moderate (timing-dependent)	Low
Primary Route	Direct placental infection	Maternal viremia during labor	Placental infection via ACE2
Placental Replication	Strong, persistent	Weak, inconsistent	Moderate (variant-dependent)

Preferred Target Cells	Hofbauer cells, trophoblasts, neural progenitors	Endothelial cells (maternal)	Syncytiotrophoblasts
Key Risk Window	First trimester	Peripartum period	Any trimester (higher risk in severe maternal disease)
Congenital Infection Frequency	Very high	Rare	Documented but uncommon

B. Placental Pathology and Structural Damage

The placenta displayed **virus-specific injury patterns**, influencing fetal outcomes. ZIKV-infected placentas consistently showed villous stromal calcification, apoptosis of trophoblasts, chronic inflammation, and severe Hofbauer cell hyperplasia. These changes correlate with extensive fetal neurotropism and congenital malformations. Importantly, ZIKV's ability to persist for weeks in placental tissue increases cumulative fetal exposure during development.

Dengue-related placental pathology stemmed mainly from **maternal systemic vascular injury**. Many placentas displayed intervillous hemorrhage, edema, fibrin deposition, and infarction. These structural alterations reduced placental perfusion and were more severe in women experiencing dengue hemorrhagic fever. Although direct placental infection was rare, deterioration of maternal hemodynamics compromised uteroplacental blood flow.

SARS-CoV-2 placentas frequently demonstrated **malperfusion, thrombosis, and inflammatory lesions**, especially in severe or symptomatic maternal infections. Villitis of unknown etiology, massive perivillous fibrin deposition, and decidual arteriopathy were recurrent findings. The cumulative effect of these lesions was significant reduction of placental exchange surfaces, thereby increasing the risk of fetal growth restriction and stillbirth.

Overall, the results indicate that ZIKV primarily causes **infectious placental injury**, dengue causes **vascular-immune placental injury**, and SARS-CoV-2 causes **inflammatory-thrombotic placental injury**.

C. Perinatal Outcomes Across the Three Infections

Clinical evidence consistently shows that ZIKV has the most devastating perinatal consequences. More than half of confirmed congenital infections resulted in microcephaly, severe neurodevelopmental impairment, ocular abnormalities, limb contractures, or fetal loss. Even infants born with normal head circumference frequently developed developmental delays later. ZIKV thus produces strongest fetal morbidity due to its direct neurotropic activity.

Dengue's effects were more **maternal-driven**. Preterm delivery, fetal distress, placental abruption, oligohydramnios, and postpartum hemorrhage were the most reported complications. Congenital dengue, although rare, manifested with fever, thrombocytopenia, hepatomegaly, and spontaneous bleeding in newborns. These infants typically required intensive neonatal care, especially when maternal thrombocytopenia or shock preceded delivery.

In SARS-CoV-2, the fetal outcomes were heavily influenced by maternal disease severity. Mild maternal infections showed minimal perinatal effects, whereas severe infections increased the risk of preterm birth, fetal growth restriction, stillbirth, and NICU admissions. Placental oxygen depletion due to malperfusion was a strong predictor of adverse fetal outcomes.

Table 2. Comparison of Major Perinatal Outcomes

Outcome Type	**Zika Virus**	**Dengue Virus**	**SARS-CoV-2**
Congenital Anomalies	Very common (microcephaly, ocular defects)	Rare	Rare
Neurodevelopmental Impairment	High	Minimal	Mild–Moderate (secondary to hypoxia)
Preterm Birth	Moderate	High	High
Fetal Growth Restriction	Frequent	Moderate	Frequent
Stillbirth/Miscarriage	Common	Moderate	Increased in severe cases
Neonatal Infection	High	Low–Moderate	Low
NICU Admission	High	High	High in severe maternal disease

CONCLUSION

Zika virus, dengue virus, and SARS-CoV-2 together represent a diverse but consequential triad of emerging infections capable of impacting pregnancy and fetal development. This review demonstrates that although these viruses share the potential for vertical transmission, the pathways through which they exert perinatal harm differ substantially. ZIKV emerges as the most potent congenital pathogen, directly exploiting placental cells to reach the fetus, where it targets neural progenitors and disrupts brain development. Its teratogenicity is unmatched among contemporary viral infections, making maternal ZIKV infection a major global reproductive health concern.

Dengue virus, by contrast, rarely infects the fetus directly. Instead, the maternal systemic consequences of dengue vascular leakage, coagulopathy, thrombocytopenia, and shock compromise placental and fetal stability. In regions where dengue is

endemic, pregnancy outcomes are strongly influenced by whether maternal disease progresses to severe forms. The literature consistently demonstrates that timing of infection, especially during the peripartum period, determines the likelihood of neonatal dengue and related complications. Thus, ensuring adequate maternal stabilization is essential for safeguarding fetal outcomes.
SARS-CoV-2 occupies an intermediate position between Zika and dengue. Although vertical transmission rates have remained relatively low, the virus significantly affects the placenta through inflammatory and vascular mechanisms. Chronic villitis, thrombotic lesions, and malperfusion hinder fetal oxygenation, increasing risks of preterm birth, growth restriction, and stillbirth. Importantly, the severity of maternal COVID-19 correlates strongly with perinatal outcomes, suggesting that maternal inflammatory status is a central driver of fetal risk. Comparing these three infections reveals critical insights. The placenta, despite being a specialized immunological barrier, is vulnerable to a spectrum of viral strategies direct replication (Zika), immune-mediated injury (dengue), and receptor-mediated entry combined with inflammation (SARS-CoV-2). Future outbreak preparedness must incorporate these mechanistic distinctions to build effective maternal–fetal health policies. Enhanced surveillance, universal antenatal screening during outbreaks, strengthening laboratory diagnostics, and targeted vaccination strategies (where available) are essential. Ultimately, the perinatal risks posed by these emerging infections will continue to evolve as climate change expands vector habitats and viral mutations alter pathogenic profiles. Robust maternal health systems, informed by the evidence presented in this review, are necessary to protect future generations from the diverse threats of emerging viral diseases.

FUTURE WORK

Future studies should conduct longitudinal cohort investigations to track neurodevelopmental, immunological, and growth trajectories of infants exposed in-utero to ZIKV, DENV, or SARS-CoV-2. Cross-regional comparative data are needed to understand how socioeconomic factors, health-system capacity, and environmental determinants shape vertical transmission patterns. Virological research must further explore placental receptor expression, innate immune pathways, and viral evasion strategies to design targeted therapeutics or vaccines. Additionally, multi-omic placental profiling (transcriptomics, proteomics, epigenomics) can illuminate how emerging viruses reprogram placental function. AI-based predictive models may enable early identification of high-risk pregnancies based on clinical biomarkers, imaging features, or maternal immune signatures. Future research must also examine co-infection scenarios (e.g., dengue + COVID-19) that may alter placental susceptibility. Developing universal antenatal guidelines for outbreak settings and integrating viral surveillance into routine prenatal care will be indispensable steps in maternal–fetal protection.

REFERENCES

1. Mlakar et al., “Zika Virus Associated With Microcephaly,” *N. Engl. J. Med.*, vol. 374, no. 10, pp. 951–958, 2016.
2. S. Rasmussen, D. Jamieson, M. Honein, and L. Petersen, “Zika Virus and Birth Defects Reviewing the Evidence,” *N. Engl. J. Med.*, vol. 374, pp. 1981–1987, 2016.
3. H. Tang et al., “Zika Virus Infects Human Cortical Neural Progenitors and Attenuates Their Growth,” *Cell Stem Cell*, vol. 18, no. 5, pp. 587–590, 2016.
4. K. Quicke et al., “Zika Virus Infects Human Placental Macrophages,” *Cell Host Microbe*, vol. 20, no. 1, pp. 83–90, 2016.
5. J. Miner et al., “Zika Virus Infection in Pregnancy,” *Cell*, vol. 165, no. 5, pp. 1121–1134, 2016.
6. C. Moore et al., “Characterizing the Pattern of Anomalies in Congenital Zika Syndrome,” *MMWR*, vol. 66, pp. 1–7, 2017.
7. S. Cauchemez et al., “Association Between Zika Virus and Microcephaly in French Polynesia,” *Lancet*, vol. 387, no. 10033, pp. 2125–2132, 2016.
8. L. Basurko et al., “Maternal and Fetal Consequences of Dengue Infection During Pregnancy,” *J. Clin. Virol.*, vol. 68, pp. 1–4, 2015.
9. S. Pouliot et al., “Maternal Dengue and Neonatal Dengue: Case Report and Review,” *Pediatrics*, vol. 138, no. 3, e20160418, 2016.
10. J. Friedman et al., “Placental Pathology in Dengue Infection,” *PLoS Negl. Trop. Dis.*, vol. 12, no. 5, e0006391, 2018.
11. E. Paixão et al., “Dengue During Pregnancy and Adverse Maternal, Fetal, and Infant Outcomes: Systematic Review and Meta-analysis,” *Lancet Infect. Dis.*, vol. 16, no. 7, pp. 857–865, 2016.
12. D. Waduge et al., “Neonatal Dengue Infection: Case Series,” *Trans. R. Soc. Trop. Med. Hyg.*, vol. 100, no. 8, pp. 876–880, 2016.
13. G. Carles et al., “Maternal and Fetal Complications of Dengue Fever During Pregnancy,” *Obstet. Med.*, vol. 11, no. 2, pp. 82–88, 2018.
14. A. Vivanti et al., “Transplacental Transmission of SARS-CoV-2,” *Nat. Commun.*, vol. 11, no. 1, pp. 1–7, 2020.
15. M. Fenizia et al., “Analysis of SARS-CoV-2 Vertical Transmission During Pregnancy,” *EMBO Mol. Med.*, vol. 12, no. 8, e12998, 2020.
16. D. Schwartz, “Placental Pathology of COVID-19,” *Arch. Pathol. Lab. Med.*, vol. 144, no. 11, pp. 1181–1192, 2020.
17. P. Shende et al., “SARS-CoV-2 Infection in Pregnancy: Evidence of Vertical Transmission,” *Clin. Infect. Dis.*, vol. 74, no. 12, pp. 2218–2224, 2022.
18. Royal College of Obstetricians and Gynaecologists (RCOG), “Coronavirus (COVID-19) Infection in Pregnancy,” Green-top Guideline 2022.
19. J. Villar et al., “Maternal and Neonatal Morbidity and Mortality with COVID-19,” *Lancet*, vol. 397, no. 10288, pp. 1729–1739, 2021.
20. A. Di Mascio et al., “Outcome of Coronavirus Spectrum Infections (SARS, MERS, COVID-19) During Pregnancy: Systematic Review and Meta-Analysis,” *Am. J. Obstet. Gynecol.*, vol. 222, no. 4, pp. 372–385, 2020.

21. J. Hecht et al., “SARS-CoV-2 Can Infect and Replicate in Placental Cells,” *Mod. Pathol.*, vol. 34, no. 8, pp. 1462–1474, 2021.