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Research Article

Maternal Anti-RSV IgG Titers and Severe Respiratory Syncytial Virus Disease in Infants with Congenital Heart Disease

Humberto Garcia Aguilar[1]*, Maria del Mar Monroy[2], Samuel Jiménez Pérez[3], Diana B. Guarneros de Regil[4], Alfonso Pérez Bañuelos[2], Gabriela Lugo[5]

[1]20 de Noviembre National Medical Center, Mexico.
[2]Hospital Angeles Lomas.
[3]Instituto Nacional de Pediatria.
[4]Anahuac Mayab University School of Medicine.
[5]MSD Mexico.

***Corresponding Author:** Humberto Garcia Aguilar, 20 de Noviembre National Medical Center, Mexico.

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Abstract

Respiratory syncytial virus (RSV) is a very common virus that affects almost all children under 2 years of age, and 50% of children under 1 year have been infected twice [1]. Despite significant improvements in clinical treatment, infants and children with severe congenital heart disease has increased risk to developed respiratory failure and congestive heart failure because of RSV infection. The risk of these infants developing a nosocomial infection during RSV epidemics may not have decreased over the past 15 years [2].

Objective(s): To associate in infants prenatally diagnosed with significant congenital heart disease, the presence of severe RSV disease and its relationship with serum levels of maternal IgG anti-RSV antibodies quantified in the third month of gestation.

Study Design: Observational, descriptive, and analytical study of a cohort of patients diagnosed during the fetal stage between 28 to 36 weeks of gestation with congenital hemodynamically significant heart disease. During the period from January 2020 to December 2022

Study Population: A cohort of 103patients who were attended at the pediatric echocardiography department at the “Centro Médico Nacional 20 de Novembre”.

Study Setting: Study start date 20-January-2020.
Sequential non-randomized recruitment of patients attending the echocardiography service for suspected fetal congenital heart disease.

All or part of this study was conducted during the COVID-19 pandemic. The study continued following the applicable safety procedures for conducting and supervising the study during the pandemic.

Statistical Methods: The Shapiro-Wilk test was used to determine the distribution. We used Spearman's correlation for non-parametric samples, clinical significance was calculated using Student's t-test. A significant value was considered at P = < 0.05. The analyses were performed using the statistical platform DATA tab.

Results: The study included 48 patients in the fetal and postnatal stages who met the criteria for significant congenital heart disease. 11 patients died at birth. 5 patients received at least one dose of Palivizumab and were therefore excluded. 58% of the patients were female. The average gestational age at the diagnosis of congenital heart disease was 29.2 weeks. Births occurred on average at the age of 36.3 weeks of gestation. The average maternal age at birth was 29.8 years. Exposure to tobacco smoke was present in 3% of the patients. Breastfeeding was observed in 21.6% of the children. The cohort follow-up was on average 13 months (ranges 9-31 months). The sera studied were negative in 86.5% of the mothers. Moderate-severe disease occurred in 19% of the cohort. The result of Spearman's correlation showed a significant correlation between maternal age and anti-RSV IgG, r = -0.42, p = 0.026. The correlation between the disease in patients with congenital heart disease and the concentration of anti-RSV IgG showed no significant correlation between the concentration of maternal anti-RSV IgG and the presence of RSV disease, (r= -0.25, p = .205)

Conclusions: The concentration of anti-RSV IgG antibodies declines with maternal age at the time of pregnancy, theoretically, immunity to RSV is not permanent and protective in pregnant women. The recirculation of the virus in known seasonal behavior, known, has been modified by the Sar-Cov-2 Pandemic, and this has led to a higher presentation of cases and more aggressive.

Background

The Global Burden of RSV and the Vulnerability of Congenital Heart Disease Respiratory Syncytial Virus (RSV) stands globally as the foremost viral pathogen responsible for Lower Respiratory Tract Infections (LRTI), including pneumonia and bronchiolitis, in the pediatric age group [2,3]. The pathogen accounts for a significant healthcare burden, leading to approximately 3 million hospitalizations and an estimated 120,000 deaths annually among children under five years of age. While the majority of this morbidity occurs in otherwise healthy infants, the risk escalates profoundly in specific high-risk populations. Children diagnosed with Congenital Heart Disease (CHD) represent a critical and highly vulnerable subgroup, identified as being at increased risk for severe respiratory disease, hospitalization due to bronchiolitis, and subsequent respiratory failure [5,6]. The physiological basis for this heightened vulnerability lies in the complex alterations to cardiopulmonary hemodynamics inherent to CHD [7,8]. Heart defects form in utero, disrupting typical blood flow patterns and often compromising oxygenation or systemic/pulmonary volume status. When RSV infection superimposes inflammation, mucous plugging, and bronchiolitis upon an already hemodynamically unstable system, the cardiac workload increases drastically. This can precipitate respiratory failure and acute cardiac dysfunction, driving rapid clinical deterioration. Consequently, specialized prophylactic measures are absolutely essential for preventing severe outcomes in this population.

Justification: Passive Immunity and the Unmet Need for Protective Biomarkers

The defense mechanism targeted by maternal immunization strategies is passive transplacental immunity. The transfer of maternal Immunoglobulin G (IgG) across the placenta is understood to mediate a degree of infant protection against RSV disease during the first six months of life [10]. Observations have historically suggested that maternal environmental exposure to circulating RSV during an epidemic period can influence the resulting antibody levels transferred to the fetus, thereby potentially affecting the child's subsequent susceptibility to infection.

Despite this established biological mechanism, a critical gap in clinical knowledge persists. Effective levels of anti-RSV IgG that could serve as robust protective biomarkers have not been prospectively defined, especially not within high-risk populations such as infants with CHD. Natural maternal immunization, whether acquired through prior infection or recent seasonal boosting, is logically related to protection against severe RSV disease in the child; however, this relationship has not been definitively proven, particularly under real-world conditions where the quality and quantity of natural maternal immunity can vary widely. The primary objective of the study (Clinical Study ID: NCT04734782) was therefore to bridge this knowledge gap: "To assess the correlation between maternal anti-RSV IgG titers during pregnancy and the occurrence or severity of Respiratory Syncytial Virus (RSV) disease in the first six months of life in infants with Congenital Heart Disease (CHD).".

Component	Description	Significance
RSV Global Burden	Leading cause of LRTI; ~3M hospitalizations, 120K deaths annually (<5 years)	Establishes the clinical urgency for prophylactic interventions
CHD Vulnerability	Infants with hemodynamically significant CHD are highly susceptible to severe RSV disease and morbidity.	Defines the critical high-risk study population requiring tailored protection
Protective Mechanism	Passive transplacental transfer of maternal IgG provides initial, short-lived protection (first 6 months).	Validates the focus on maternal antibody status as a prophylactic measure

Knowledge Gap	Effective anti-RSV IgG thresholds as protective biomarkers for high-risk populations remain undefined.	Outlines the central question the study sought to answer

Table 1: Clinical Context and Study Rationale

Study Design and Population

The research utilized a Prospective Observational Cohort Study design , registered under the identifier NCT04734782 (Clinical Study ID 025.2021). The study enrolled maternal-infant dyads, specifically focusing on newborns diagnosed with Congenital Heart Disease (CHD). The surveillance period covered the infant's first six months of life, a timeframe critical for high RSV susceptibility and peak protection conferred by maternally derived IgG. Key inclusion criteria mandated enrollment of pregnant women and their fetus diagnosed with CHD. The timing of the study, with a final update in January 2021 , implies that data collection occurred during or immediately following the initial years of the SARS-CoV-2 pandemic, an important contextual factor discussed.

Measurement and Criteria for Severe Disease

The central measurement involved the quantification of maternal anti-RSV IgG antibody titers at the time of delivery. This measurement was correlated with the primary outcome: the occurrence and severity of RSV LRTI in the infant during the follow-up period.

Prior informed consent, a maternal blood sample was taken for the analysis of anti-RSV IgG (antibody G) concentrations in 48 participants.

The procedure was performed manually according to the supplier's manual (Respiratory Syncytial Virus IgG SERION ELISA classic ESR113G).

At birth, the diagnosis of congenital heart disease was confirmed. Management for each hemodynamic situation was initiated. The risk of mortality from the underlying disease (type of malformation) was calculated using the Rash-1 scale. Patients were followed up after discharge to identify criteria for severe RSV infection according to Wang's criteria. The viral load of the affected children was diagnosed with nasal swabs/aspirations and determined by RT-qPCR, as previously described

Defining clinical severity in CHD infants requires specialized indices that account for their baseline cardiac vulnerability. Severe RSV LRTI was assessed using validated clinical scoring systems, such as the Wang score, alongside specific markers of clinical deterioration. The literature confirms that severity assessment often relies on objective criteria such as: Wang scores exceeding 8; oxygen saturation falling below 90%; and the requirement for critical interventions including prolonged oxygen therapy, admission to the intensive care unit (ICU), or antibiotic treatment.

The robust definition of "severe disease" is paramount in this high-risk cohort. Since CHD compromises baseline oxygenation and cardiovascular reserve, even seemingly standard clinical measures of respiratory distress can indicate profound physiological stress [11,12]. For instance, the Wang score, which incorporates respiratory and heart rates, is highly relevant for evaluating LRTI in young infants. The fact that these severity thresholds were utilized ensures that the observed outcomes reflect true deterioration beyond the infant's compromised baseline, underscoring the necessity for robust prophylactic measures in this vulnerable population.

Results

The results demonstrate both the biological efficacy of antibody transfer and the subsequent failure of these transferred antibodies to provide clinical protection against severe disease in the CHD cohort.

Statistical Methods: The Shapiro-Wilk test was used to determine the distribution. We used Spearman's correlation for non-parametric samples, clinical significance was calculated using Student's t-test. A significant value was considered at $P = < 0.05$. The analyses were performed using the statistical platform DATA tab.

Results: The study included 48 patients in the fetal and postnatal stages who met the criteria for significant congenital heart disease. 11 patients died at birth. 5 patients received at least one dose of Palivizumab and were therefore excluded. 58% of the patients were female. The average gestational age at the diagnosis of congenital heart disease was 29.2 weeks. Births occurred on average at the age of 36.3 weeks of gestation. The average maternal age at birth was 29.8 years. Exposure to tobacco smoke was present in 3% of the patients. Breastfeeding was observed in 21.6% of the children. The cohort follow-up was on average 13 months (ranges 9-31 months). The sera studied were negative in 86.5% of the mothers. Moderate-severe disease occurred in 19% of the cohort. The result of Spearman's correlation showed a significant correlation between maternal age and anti-RSV IgG, $r = -0.42$, $p = 0.026$. The correlation between the disease in patients with congenital heart disease and the concentration of anti-RSV IgG showed no significant correlation between the concentration of maternal anti-RSV IgG and the presence of RSV disease, ($r= -0.25$, $p = .205$

High Burden of Severe Disease in the Cohort

The study confirmed the extreme clinical vulnerability of infants with CHD upon RSV infection. The severity rate observed was alarmingly high, validating the initial premise of the study that this group faces exceptional risk.

Specific data revealed that bronchiolitis was categorized as severe (Wang score higher than, 12) in 53.3% of the cases reviewed. Furthermore, objective measures of respiratory compromise showed that nearly all infected infants suffered profound hypoxia: oxygen saturation dropped below 90% in 97% of cases, necessitating oxygen therapy in a corresponding 97% of cases. This high severity rate, with over half the cohort meeting criteria for severe disease and almost all requiring intensive oxygen support, provides definitive justification for aggressive, standardized RSV prophylaxis. The findings clearly illustrate that reliance on naturally acquired maternal immunity alone is insufficient to prevent major clinical decompensation in CHD infants.

Transplacental Transfer Efficacy: Biological SuccessThe analysis of antibody levels in maternal and neonatal samples confirmed the efficiency of the transplacental transfer mechanism. A statistically significant and strong positive correlation was established between the anti-RSV IgG antibody levels measured in the mothers at delivery and the corresponding levels in the newborns at birth.

- **Correlation Coefficient (r):** r=0.667 (indicating a strong positive correlation).
- **Significance (p-value):** p=0.0001 (highly statistically significant).

This robust correlation confirms the foundational premise of maternal immunization: the human placenta functions efficiently in transferring RSV-specific IgG antibodies. This means that the eventual failure to correlate with protection (detailed below) cannot be attributed to a systemic breakdown in the mechanism of antibody delivery. Rather, the challenge must lie either in the quality of the transferred antibodies or the overwhelming pathological burden imposed by RSV on a compromised cardiac system.

The Paradox of Antibody Titers Versus Clinical Protection

Despite the successful biological transfer demonstrated by the strong correlation coefficient (r=0.667), the study found that the resulting antibody titers did not significantly translate into clinical protection against severe RSV disease. In high-risk infants, severe disease symptoms were found to be unrelated to the overall level (titer) of RSV-specific IgG antibodies transferred. (Fig. 1)

a high quantity of total antibody does not guarantee a protective quality or functional capacity sufficient to mitigate severe outcomes in infants with profound underlying vulnerabilities like CHD. The inability of total IgG concentration to serve as a reliable prognostic biomarker for severe outcomes suggests that protective efficacy is contingent upon factors beyond simple quantity, such as epitope specificity or avidity. For instance, high total IgG titers may be dominated by antibodies targeting non-neutralizing components of the virus (such as the G protein), while protective immunity relies heavily on neutralizing antibodies directed toward critical epitopes like the prefusion F site. Therefore, a functional deficiency, rather than a quantitative one, is likely implicated in the failure to protect this high-risk group.

Influence of Maternal Demographic Factors

The analysis also explored factors influencing maternal antibody levels prior to transfer. A statistically significant negative correlation was observed between maternal age and anti-RSV IgG antibody levels. This negative correlation suggests that maternal immunity may wane over time or become less robustly boosted with advancing age, potentially reflecting less recent natural exposure to circulating RSV strains. This reinforces the epidemiological concept that maternal immunization, whether natural or induced, is highly seasonal and time-sensitive. If baseline maternal immunity declines or is not recently boosted, the total quantity of antibodies available for transfer, even if efficiently delivered, may be insufficient to reach the high protective threshold required by an infant with CHD. This evidence supports the importance of targeted intervention, such as vaccination late in pregnancy, to ensure maximal boosting of maternal titers immediately prior to delivery. (Fig. 2)

Finding	Statistical Detail	Interpretation	Implication
Transfer Efficacy	r=0.667,p=0.0001	Highly efficient transplacental IgG transfer confirmed.	Biological mechanism (transfer) is functional.
Disease Severity	53.3% required Wang Score >8; 97% required O2.	Confirms extreme clinical vulnerability of the CHD cohort to RSV.	Requires aggressive, standardized prophylaxis.
Protective Correlation	Not statistically significant (r/p missing for total IgG vs. severity)	High total IgG titer does not confer reliable protection against severe disease.	Total IgG titer is a poor functional biomarker in high-risk infants.
Maternal Age Effect	Significant Negative Correlation (r/p missing for age vs. titer)	Maternal antibody levels may decline with age or lack of recent boosting.	Supports the need for timely maternal immunization.

Table 2: Statistical Summary of Results

Discussion and Future Directions

Interpretation of the Efficacy-Severity Disconnect

The synthesis of the results reveals a critical disconnect: the successful biological transfer of maternal IgG (strong correlation, r=0.667) does not translate into effective clinical mitigation against severe outcomes in CHD infants (high severity rate of 53.3% , with titers being unrelated to protection).

This finding carries profound clinical ramifications. It strongly indicates that passive immunity derived solely from natural maternal exposure is insufficient to elevate antibody levels above the critical threshold necessary to counteract the overwhelming physiological stress of RSV infection in a CHD infant. Given the predictable high severity and mortality risk in this group , reliance on variable, naturally acquired maternal immunity is demonstrably inadequate. This strongly supports current clinical guidelines that mandate targeted, high-efficacy prophylaxis using standardized monoclonal antibodies (such as Palivizumab or the extended half-life Nirsevimab and and the recently FDA-approved Clesrovimab) which ensure high, uniform neutralizing titers, bypassing the uncertainty of natural maternal immunity [24,25].

The Crucial Limitation: Impact of the SARS-CoV-2 Pandemic

A critical limitation that must contextualize the interpretation of these findings is the effect of the SARS-CoV-2 pandemic on global RSV epidemiology. Clinical research has documented a profound disruption in typical RSV circulation patterns during periods of stringent public health measures and lockdowns [14-16].

The dramatic reduction in RSV circulation during the pandemic likely meant that the study cohort of pregnant persons experienced reduced environmental exposure to the virus. Lower environmental exposure limits the process of natural "boosting"—the periodic re-exposure that increases the quality and quantity of specific antibodies in the maternal system. Consequently, the maternal anti-RSV IgG measured at delivery may have represented a baseline or suboptimal level of immunity compared to typical pre-pandemic years. If the maternal antibodies were low-quality or low-titer due to this epidemiological shift, their efficient transfer (the r=0.667 finding) would still result in insufficient protection for the extremely high-risk CHD infant.

The study results, viewed through this lens, may reflect the protective inadequacy of suboptimal passive immunity under conditions of altered virus seasonality. This limitation heightens the urgency for controlled prophylactic strategies, specifically maternal vaccination administered late in pregnancy (e.g., 32–36 weeks' gestation), which is designed to artificially induce high levels of neutralizing antibodies regardless of natural environmental exposure.

Implications for Future Prophylaxis and Research

The study reinforces several key elements for optimizing protection against RSV in vulnerable infants:

1. **Standardized Prophylaxis is Non-Negotiable:** Given the confirmed high severity rate and the failure of variable natural passive immunity to reliably protect CHD infants, specialized immunization—such as the use of monoclonal antibodies—is justified and medically required for this specific population [19,20].
2. **Temporal Importance of Maternal Immunization:** The negative correlation between maternal age and antibody titers, coupled with the confounding variable of pandemic-related low exposure, emphasizes that maternal immunity wanes. Future public health strategies must therefore focus on the timely administration of maternal vaccines to ensure maximal transfer of high-titer, high-quality neutralizing antibodies just prior to the infant's birth.
3. **Need for Functional Antibody Assays:** The central paradox—efficient transfer but poor protection—strongly suggests that measuring total IgG is insufficient to define correlates of protection in this high-risk group. Future research must move beyond simple IgG quantification and focus on sophisticated functional assays, specifically quantifying neutralizing antibody titers directed against protective epitopes (e.g., prefusion F protein). Identifying the precise threshold of functional antibody needed for protection in CHD infants is the critical next step.

Conclusions

The study on maternal anti-RSV IgG titers in infants with CHD provides critical evidence that reframes the discussion around passive immunization efficacy in high-risk populations. While the transplacental mechanism for IgG transfer is confirmed to be highly efficient (r=0.667,p<0.0001), reliance on naturally acquired maternal immunity is demonstrably insufficient to prevent severe RSV disease, given the 53.3% severe bronchiolitis rate observed in the cohort. The failure of total IgG titers to correlate significantly with protection suggests that the protective threshold required for CHD infants exceeds what natural exposure typically provides, or that the qualitative aspects of naturally transferred antibodies (e.g., avidity or specific epitope targeting) are deficient.

This necessitates a shift in prophylactic strategy for CHD infants, prioritizing standardized, high-quality interventions such as targeted monoclonal antibodies. Furthermore, acknowledging the limiting effect of altered RSV epidemiology during the SARS-CoV-2 pandemic emphasizes the need for proactive maternal vaccination, ensuring maximally boosted titers regardless of natural community circulation. Future research must, therefore, pivot from measuring total antibody quantity to defining the functional correlates of protection through neutralizing antibody assays, providing precise biomarkers for individualized risk assessment and management

in this profoundly vulnerable population.

Figure. 1

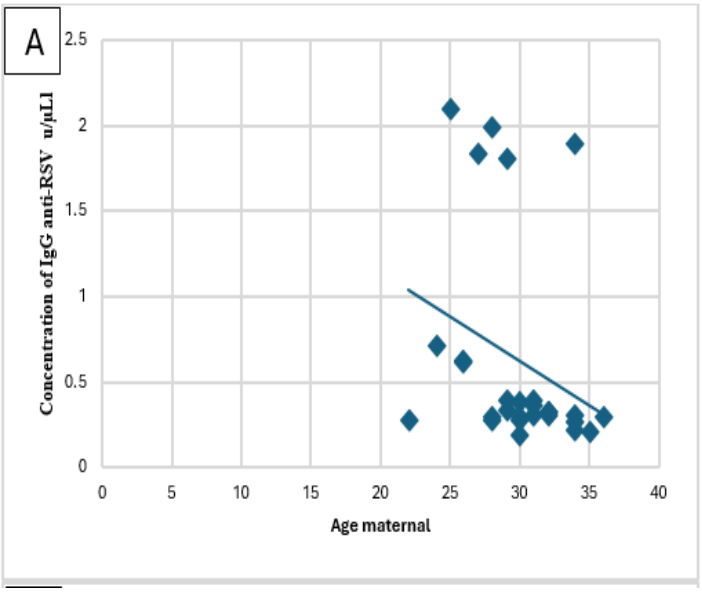


Figure. 2A

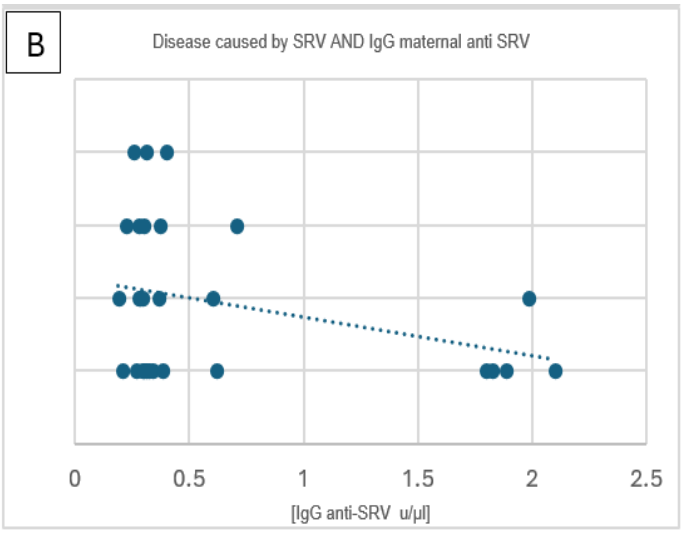
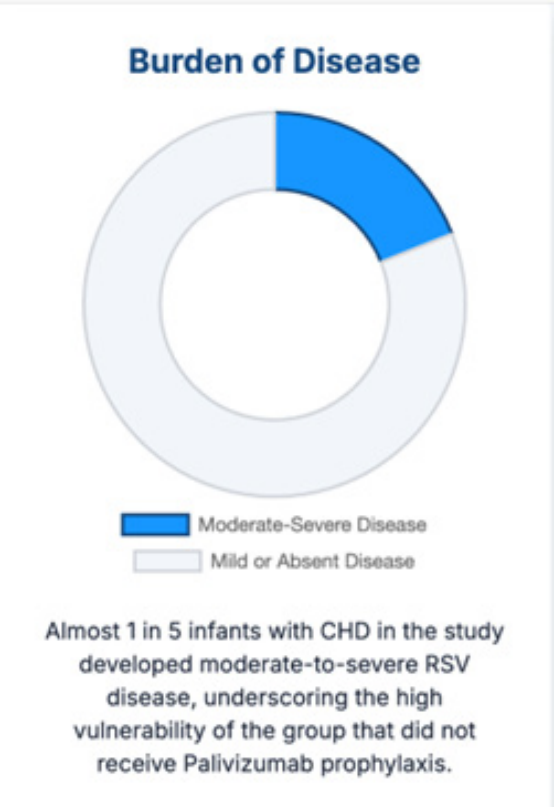


Figure. 2B

A Significant negative correlation (r = -0.42, p = 0.026) was found. As maternal age increases, the concentration of anti-RSV IgG antibodies tends to decrease, suggesting that natural immunity may wane over time.

B No significant correlation (r = -0.25, p = 0.205) was found. Maternal antibody levels did not reliably predict whether the infant would develop severe disease, potentially due to altered viral circulation dynamics during the COVID-19 pandemic.



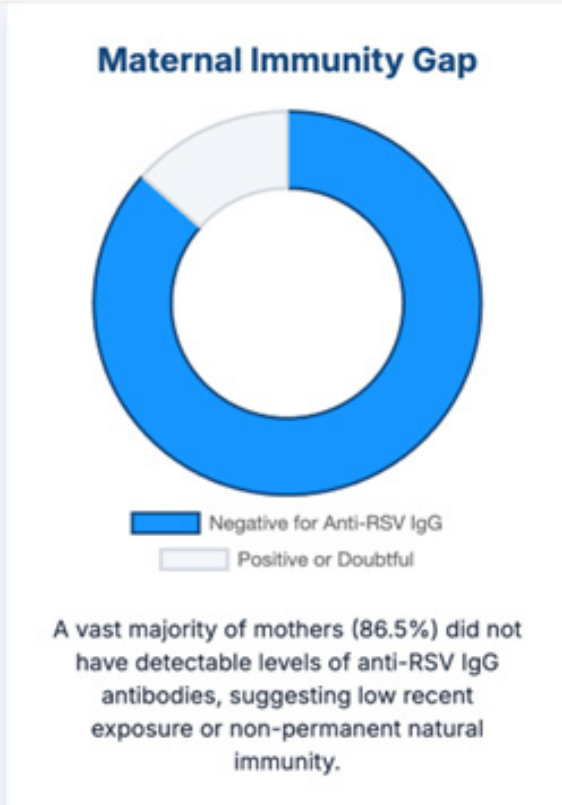


Figure. 3

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