Review Article

Pharmacological Prevention of Severe Respiratory Syncytial Virus Infection in High-Risk Infants and Children: Maternal Immunization and Long-Acting Monoclonal Antibodies

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ABSTRACT

Respiratory syncytial virus (RSV) remains a major cause of lower respiratory tract disease and hospitalization in infants, with the highest burden concentrated during the first months of life and among children with limited cardiopulmonary reserve. Prevention has shifted from selective, repeatedly administered palivizumab to maternal RSV prefusion F protein vaccination and extended-half-life monoclonal antibodies. This review examines the pharmacological basis, clinical efficacy, safety, pharmacokinetics, real-world effectiveness, resistance, and implementation of these approaches, with emphasis on infants at increased risk of severe disease. Maternal RSVpreF vaccination raises maternal neutralizing antibodies and transfers IgG across the placenta, protecting the newborn. Nirsevimab and clesrovimab provide direct passive immunity and maintain antibody exposure through much of the first RSV season after a single dose. Randomized trials show substantial reductions in medically attended RSV lower respiratory tract disease and hospitalization with nirsevimab, while clesrovimab has demonstrated similar short-term clinical protection. Real-world studies from Europe, North America, and South America broadly confirm these benefits, although effectiveness varies with timing, coverage, endpoint definition, and epidemiology. Maternal vaccination has also shown protection against infant hospitalization in post-licensure studies. Important uncertainties include waning protection, breakthrough infection, resistance, second-season prevention, access, and the optimal use of maternal vaccination and infant monoclonal antibodies within the same health system. Prevention programs should therefore integrate product characteristics with gestational age, maternal vaccination status, infant age, medical risk, seasonality, and local delivery capacity.

Key words: Respiratory Syncytial Virus, RSV, Maternal Immunization, Rsvpref, Nirsevimab, Clesrovimab, Palivizumab, Infants, Monoclonal Antibodies, Prophylaxis, Pharmacokinetics.

Respiratory syncytial virus (RSV) is an enveloped, negative-sense, single-stranded RNA virus and one of the principal causes of acute lower respiratory tract infection in young children. Prospective and population-based studies have shown that infection is common during infancy, while the clinical burden is concentrated in the youngest children. In a prospective US study, RSV accounted for a substantial proportion of acute respiratory disease encounters and hospitalizations in the first two years of life, with the highest hospitalization risk during early infancy [1]. A Finnish birth cohort similarly demonstrated a high incidence of RSV illness during the first year of life, including outpatient disease and hospitalization [2].

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Community surveillance has also shown that infection occurs across the first two years, with age, household exposure, and seasonal circulation influencing risk [3]. The burden remains clinically important despite changes in RSV seasonality during and after the COVID-19 pandemic. Multicentre US surveillance found increased hospitalization rates during the atypical 2021 and 2022 seasons, while a population-based Canadian study documented a marked increase in pediatric hospitalization and intensive care use during the 2022-2023 season [4,5]. RSV is also a major cause of infant hospitalization in routine health-system data, and disease is not confined to infants with recognized comorbidities [6,7]. Hospitalized children younger than six months have particularly high rates of oxygen requirement and intensive care admission, while prematurity is associated with prolonged hospitalization [8].

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Data from southern India further illustrate that RSV contributes substantially to hospitalized acute lower respiratory infection and circulates beyond the temperate seasonal pattern commonly used in high-income-country prevention programs [9]. These observations explain the pharmacological rationale for prevention during the period of greatest vulnerability. Palivizumab established that antibody-mediated prophylaxis could reduce severe RSV disease in selected high-risk infants, but its monthly dosing and restricted eligibility limited its population reach [10,11]. This review examines the pharmacological prevention of severe RSV disease from maternal immunization to direct infant passive immunization, with particular emphasis on infants and children at increased risk of severe disease. It discusses the biological rationale, clinical efficacy, pharmacokinetics, real-world effectiveness, safety, resistance, second-season prevention, and implementation considerations relevant to the current RSV prevention landscape.

2. BIOLOGICAL AND PHARMACOLOGICAL BASIS OF PREVENTION

The RSV fusion (F) glycoprotein is the principal target of neutralizing antibodies used in contemporary prevention strategies. Maternal vaccination and monoclonal antibodies exploit the same broad principle, neutralization of the virus before extensive replication, but differ in how antibodies are generated or delivered. Maternal RSVpreF vaccination stimulates active immunity in the pregnant person, followed by placental transfer of IgG to the fetus. In the phase 2b maternal vaccine study, vaccination generated substantially higher RSV-A and RSV-B neutralizing titers in maternal and cord blood, with efficient transplacental transfer [12]. The phase 3 MATISSE trial subsequently demonstrated clinically meaningful protection against severe medically attended RSV lower respiratory tract disease in infants through the first six months of life [13].

Direct passive immunization avoids dependence on maternal vaccination, maternal immune response, and placental transfer. Nirsevimab is a recombinant human IgG1 monoclonal antibody directed against a conserved prefusion F-protein epitope and engineered for prolonged systemic exposure. Its dosing strategy is weight-based and designed to maintain protective concentrations during the RSV season. Population pharmacokinetic analyses indicate that body weight and postmenstrual age are the principal determinants of clearance, while congenital heart disease and chronic lung disease do not materially alter exposure [14]. This supports weight-banded dosing across healthy, preterm, and medically vulnerable infants. Clesrovimab is another extended-half-life monoclonal antibody directed against RSV F. In a phase 3 trial of healthy preterm and full-term infants, a single 105-mg dose reduced medically attended RSV lower respiratory tract infection and hospitalization over 150 days, with serious adverse events occurring at similar frequencies in the clesrovimab and placebo groups [15]. A subsequent randomized trial in infants at increased risk for severe disease evaluated clesrovimab against palivizumab during the first season and a higher dose during the second season, extending the clinical evidence into medically vulnerable populations [16].

The pharmacological distinction between maternal vaccination and monoclonal antibodies is therefore one of exposure control. Maternal vaccination creates variable infant antibody exposure determined by maternal response, gestational age at vaccination and delivery, and placental transfer. Monoclonal antibodies deliver a defined dose directly to the infant, but protection depends on timely administration and subsequent antibody decay. These differences become particularly relevant for preterm infants, infants born shortly before or during an RSV season, and children whose mothers were not vaccinated.

3. MATERNAL RSV VACCINATION

Maternal immunization is designed to place neutralizing antibodies in the infant circulation before the first exposure to RSV. The RSVpreF vaccine evaluated in MATISSE is a bivalent prefusion F protein vaccine administered during late pregnancy. In the phase 3 trial, severe medically attended RSV-associated lower respiratory tract disease occurred substantially less often among infants born to vaccinated mothers than among those born to mothers who received placebo, with protection evident during the first 180 days [13].

The trial therefore established proof that maternal vaccination can prevent clinically important infant disease without direct infant administration. Safety interpretation requires attention to product-specific evidence. A separate phase 3 trial of an investigational RSVPreF3-Mat vaccine was stopped early after an imbalance in preterm birth was observed; the trial reported lower infant RSV disease but a higher rate of preterm birth in the vaccine group [17]. This finding concerns the investigational product and does not establish the same risk for RSVpreF used in MATISSE. In the MATISSE program, subsequent analyses continued to evaluate pregnancy outcomes and infant safety, and the clinical interpretation of maternal vaccination must remain product-specific rather than treating all prefusion-F vaccines as interchangeable. Real-world evidence has begun to complement trial data. In Argentina, where RSVpreF was introduced nationally as a maternal immunization strategy in 2024, a multicentre test-negative study found substantial effectiveness against RSV-associated lower respiratory tract disease requiring infant hospitalization and against severe hospitalized disease [18]. A large French population-based study subsequently found lower infant RSV-LRTI hospitalization risk after maternal RSVpreF vaccination, with the greatest protection during the first two weeks after

birth and attenuation over subsequent weeks [19]. US surveillance during the 2024-2025 season also estimated protection from maternal vaccination against RSV-associated hospitalization, although estimates differed from those for emergency department outcomes [20]. The principal limitations of maternal vaccination are programmatic rather than purely biological. Protection depends on vaccination before delivery and sufficient time for antibody generation and transfer. Very preterm birth may occur before adequate transplacental transfer, and antibody concentrations decline after birth. Maternal vaccine uptake may also be lower in populations with limited antenatal-care access, uncertainty about vaccination, or supply constraints. Consequently, maternal vaccination can provide strong birth-to-infancy protection but cannot guarantee equivalent exposure in every newborn.

4. NIRSEVIMAB

Nirsevimab has the most extensive clinical evidence among currently available long-acting infant monoclonal antibodies. In preterm infants born at 29 to 34 weeks' gestation, a single dose reduced medically attended RSV lower respiratory tract infection compared with placebo [21]. In the MELODY phase 3 trial among healthy late-preterm and term infants, efficacy against medically attended RSV lower respiratory tract infection through 150 days was 74.5%, while hospitalization was less frequent, but the trial was not powered to establish a statistically significant hospitalization effect [22]. A pooled analysis of randomized trials subsequently demonstrated reductions in medically attended RSV lower respiratory tract infection, hospitalization, and very severe disease, and pharmacokinetic data supported extrapolation to infants with chronic lung disease, congenital heart disease, and extreme prematurity [23].

The pragmatic HARMONIE trial strengthened the hospitalization evidence. Among more than 8000 infants entering their first RSV season, hospitalization for RSV-associated lower respiratory tract infection occurred in 0.3% of nirsevimab recipients compared with 1.5% receiving standard care, corresponding to 83.2% efficacy [24]. The 180-day analysis showed similar protection through six months, with 0.3% versus 1.7% hospitalized and an efficacy of 82.7% [25]. These data are important because prevention programs must cover variable RSV-season duration, and infants may be exposed late in the season. Real-world studies broadly reproduce the trial signal. A population-based study from Galicia reported 82.0% effectiveness against RSV-LRTI hospitalization and 86.9% against severe RSV-LRTI requiring oxygen support after high program coverage [26]. In Catalonia and Andorra, a test-negative study estimated 81.0% effectiveness against RSV-LRTI hospitalization and 85.6% against severe disease requiring noninvasive or invasive ventilation [27]. A US multicentre surveillance study estimated 89% effectiveness against medically attended RSV-associated acute respiratory illness and 93% against RSV-associated hospitalization in the 2023-2024 season [28]. Kaiser Permanente Northern California data from the same season estimated 87.2% effectiveness against RSV-LRTD and 98.0% against hospitalized RSV-LRTD [29].

More recent data indicate that effectiveness persists under broader implementation but varies with endpoint and setting. A US study using five health systems estimated 77% effectiveness against hospitalization during the 2024-2025 season [20]. Italian regional studies have reported substantial reductions in hospitalization after universal implementation, including approximately 82% fewer RSV hospitalizations in Tuscany and a 78.3% adjusted effectiveness estimate in a multicentre Italian test-negative study [30,31]. A southern Italian surveillance study using screening, test-negative, and cohort methods produced effectiveness estimates ranging from 72.5% to 84.4%, illustrating the methodological sensitivity of real-world estimates [32]. The evidence therefore supports nirsevimab as a highly effective means of reducing severe RSV disease during the first season. However, real-world effectiveness should not be interpreted as a fixed biological percentage. Estimates are influenced by time from dosing to exposure, circulating RSV subtype, coverage, healthcare-seeking behavior, outcome definition, and residual confounding in observational designs.

5. CLESROVIMAB AND THE EVOLUTION OF LONG-ACTING ANTIBODY PREVENTION

Clesrovimab expands the long-acting monoclonal-antibody platform rather than representing a fundamentally different biological strategy. In the CLEVER phase 3 trial, 2412 infants received a single 105-mg dose and 1202 received placebo. Over 150 days, medically attended RSV lower respiratory tract infection occurred in 2.6% versus 6.5%, giving 60.4% efficacy, while RSV-associated hospitalization was reduced by 84.2% [15]. Serious adverse events were similar between groups. These results establish clinically important protection while also showing that efficacy estimates depend strongly on the endpoint used. The SMART trial extended evaluation to infants at increased risk for severe disease and included a second-season dosing component [16].

This is particularly relevant to children with chronic lung disease of prematurity, congenital heart disease, and other conditions associated with persistent vulnerability. The existence of two extended-half-life antibodies also creates practical flexibility in settings where product supply, eligibility, cost, or national policy differs. At present, the evidence base does not justify assuming interchangeability for every clinical situation. Trial populations, dosing regimens, age ranges, and second-season data differ between products. Product-specific pharmacokinetics, regulatory indications, and local

recommendations should therefore guide use. The clinically important advance is the ability to provide prolonged direct passive immunity with a single administration during the first season, rather than the specific identity of one antibody.

6. PALIVIZUMAB: HISTORICAL FOUNDATION

Palivizumab remains the historical benchmark for antibody prophylaxis. In the IMpact-RSV randomized trial, five monthly doses reduced RSV hospitalization among high-risk infants born prematurely or with bronchopulmonary dysplasia [33]. A subsequent randomized trial in children with hemodynamically significant congenital heart disease also demonstrated a reduction in RSV hospitalization [34].

These studies established the causal relationship between passive antibody administration and prevention of severe RSV outcomes. Palivizumab's main limitation is pharmacological exposure over time. Monthly administration is necessary to maintain adequate concentrations throughout a season, creating repeated opportunities for missed doses and increasing administration burden. Real-world outcome studies also documented the logistical consequences of a multidose strategy [35]. Long-acting antibodies retain the core mechanism while reducing the number of administrations. Thus, palivizumab is best understood as the foundation from which current extended-half-life approaches developed rather than as a direct competitor with equivalent dosing convenience.

7. MATERNAL VACCINATION AND INFANT MONOCLONAL ANTIBODIES: COMPLEMENTARY PATHWAYS

Maternal vaccination and infant monoclonal antibodies address different points in the prevention pathway. Maternal vaccination can protect from birth and uses established antenatal-care contacts, whereas monoclonal antibodies provide predictable direct exposure after birth. The practical choice depends on whether the mother was vaccinated during the recommended gestational window, whether sufficient time elapsed before delivery, the infant's age and weight, medical risk, RSV seasonality, and local product availability. Routine administration of both interventions to every infant is not supported by current evidence. A 2026 randomized phase 4 study comparing maternal RSVpreF vaccination, infant nirsevimab, and sequential combinations found that the approaches were safe and generated high neutralizing-antibody concentrations through interim follow-up, but the study was designed primarily around safety, tolerability, and immunogenicity rather than definitive comparative clinical efficacy [36].

Thus, combined administration may be relevant to specific circumstances but should not automatically be equated with additional clinical benefit. For infants born soon after maternal vaccination, individualized assessment is particularly important. The relevant questions are the interval between vaccination and delivery, expected antibody transfer, infant age at exposure, and whether the infant has conditions that increase the consequences of breakthrough infection. Direct antibody prophylaxis is pharmacologically attractive when the maternal pathway is incomplete or uncertain.

8. HIGH-RISK INFANTS, SECOND-SEASON PREVENTION, AND SPECIAL POPULATIONS

The rationale for high-risk prevention is based on both probability of severe disease and consequences of infection. Prematurity is associated with immature lung development and reduced opportunity for transplacental antibody acquisition. Chronic lung disease of prematurity and hemodynamically significant congenital heart disease further reduce respiratory reserve. The original palivizumab trials established the relevance of these conditions, while nirsevimab pharmacokinetic and clinical data support extended-half-life antibody use in medically vulnerable infants [23,33,34].

Second-season prevention is more selective because age-related maturation reduces the absolute risk for most children. Nevertheless, selected children remain vulnerable, particularly those with severe chronic lung disease or immunocompromise. The MEDLEY program and subsequent studies have provided evidence regarding repeated exposure and pharmacokinetics, including sustained neutralizing antibody levels after nirsevimab administration [37]. Importantly, follow-up of children previously receiving nirsevimab has not demonstrated evidence of enhanced disease during the subsequent RSV season [38]. Children with cystic fibrosis and complex congenital heart disease require individualized consideration because risk depends on disease severity, treatment, and residual cardiopulmonary reserve. Evidence for these groups is less extensive than evidence in healthy term infants. This limitation should be explicit when translating population-level efficacy estimates to individual high-risk children.

9. RESISTANCE, BREAKTHROUGH INFECTION, AND DURABILITY

No prophylactic antibody completely prevents infection. Breakthrough RSV infection can occur when antibody concentrations decline, when exposure is intense, or when viral variants show reduced susceptibility. The prefusion F epitope targeted by nirsevimab creates a defined genetic region in which amino-acid substitutions can affect neutralization. Early surveillance did not identify a major population-wide resistance problem, but continued genomic monitoring is necessary. Recent French multicentre data provide direct evidence that resistance can emerge under real-world selective pressure. Among sequenced breakthrough infections during the 2024-2025 season, resistance-associated substitutions were identified more frequently in RSV-B than RSV-A, and phenotypic

resistance was documented in a subset of RSV-B breakthrough viruses [39]. Importantly, these findings do not establish that resistance is common among all infections or that clinical effectiveness has been lost. A separate French effectiveness study during high RSV-B circulation continued to demonstrate protection against hospitalization, although effectiveness estimates differed across analyses [40].

Durability is similarly context-dependent. Nirsevimab trials demonstrate protection for approximately five to six months after administration, but antibody concentrations and clinical protection decline over time. Maternal vaccine-derived antibody also declines after birth. These kinetics matter in regions with prolonged or year-round circulation. Programs in tropical or subtropical settings therefore require local surveillance rather than direct transfer of dosing calendars developed for highly seasonal temperate regions.

10. IMPLEMENTATION, EQUITY, AND THE INDIAN CONTEXT

The clinical efficacy of an RSV prevention product can translate into population benefit only when administration occurs before exposure. Program design therefore has to address supply, cold-chain management, documentation, timing of birth, maternal vaccine uptake, newborn discharge pathways, and follow-up of high-risk children. Maternal vaccination depends on antenatal-care contact, whereas monoclonal antibodies depend on reliable delivery through maternity, neonatal, pediatric, or primary-care services.

The economic and operational comparison is also context-specific. A single-dose infant antibody can reduce the burden of repeated administration, but biologic acquisition costs may be substantial. Maternal vaccination can reach the infant through an established antenatal platform but depends on uptake and adequate timing before delivery. In health systems with limited resources, the relevant evidence gap is therefore not only efficacy but the relationship between product price, coverage, hospitalization burden, and delivery feasibility. India illustrates this problem. RSV has been detected throughout the year in hospitalized children in southern India, with seasonal peaks that do not necessarily conform to a single nationwide winter pattern [9].

A national prevention strategy would therefore require regional epidemiological surveillance, characterization of birth-season risk, assessment of maternal vaccination coverage, and health-economic evaluation. Evidence from high-income countries can inform pharmacology and efficacy but cannot simply determine an optimal Indian implementation model. Studies evaluating locally feasible delivery pathways and the effect of prevention on hospitalization, oxygen use, and intensive care would be particularly valuable.

11. CURRENT EVIDENCE GAPS AND FUTURE DIRECTIONS

Several evidence gaps remain. First, direct head-to-head clinical comparisons of maternal RSV vaccination, nirsevimab, and clesrovimab are limited. Differences in trial populations and endpoints make indirect comparison uncertain. Second, most first-season studies provide approximately five to six months of follow-up, whereas the ideal duration of protection varies with local seasonality and birth timing. Third, evidence in extremely preterm infants, children with severe immunocompromise, and complex cardiopulmonary disease remains smaller than the evidence base in healthy infants. Fourth, observational studies increasingly demonstrate high effectiveness, but estimates vary between test-negative, cohort, screening, and ecological designs. The range of reported estimates should therefore be interpreted as evidence of consistent direction and substantial benefit rather than as a single universal effectiveness value. Fifth, resistance surveillance needs to be integrated with clinical surveillance. Sequencing breakthrough infections and linking viral genotype to phenotypic neutralization can identify emerging escape before it materially affects program performance.

Future pharmacological development is likely to focus on longer duration, broader epitope coverage, improved manufacturability, and lower dosing complexity. The possibility of combining maternal vaccination with infant antibodies requires clinical studies that evaluate meaningful outcomes rather than antibody concentrations alone. Research should also address delivery in low- and middle-income settings, where the absolute burden of RSV disease may be high but access to novel biologics is constrained. For individual infants, prevention decisions will increasingly depend on integrating gestational age, maternal vaccination history, infant weight and age, medical risk, seasonality, and availability of a suitable product.

CONCLUSION

Pharmacological prevention of severe RSV disease has moved from selective monthly antibody prophylaxis toward two complementary long-acting approaches: maternal vaccination and direct infant monoclonal-antibody administration. Maternal RSVpreF vaccination can establish passive antibody protection before birth, whereas nirsevimab and clesrovimab provide predictable direct passive immunity after birth. Randomized trials and early post-licensure studies consistently show substantial reductions in RSV-associated lower respiratory tract disease and hospitalization, although the magnitude of effect varies by product, endpoint, population, and timing. For high-risk infants and children, the practical objective is to ensure adequate antibody protection during the period when severe disease is most consequential. This requires more than selecting a product on the basis of trial efficacy.

Maternal vaccination status, timing of delivery, infant age, prematurity, cardiopulmonary disease, local RSV circulation, product availability, and the feasibility of timely administration all influence the expected benefit. Continued surveillance is essential for safety, durability, breakthrough infection, resistance, and equity. The next phase of RSV prevention should therefore be built around coordinated, evidence-based prevention pathways rather than reliance on a single technology.

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