Received: 04.11.2025. *Revised:* 03.02.2026. *Accepted:* 25.02.2026.

JOURNAL OF HUMAN BIOLOGY AND CLINICAL MEDICINE

Journal homepage: https://humanbiomed.com/en Vol. 2. No. 1. 2026

UDC 616-018+616-008.1+616-092.19+618.36 DOI: 10.63621/jhbcm/1.2026.28

Transplacental antibody perfusion in the development of the newborn passive immunity: A literature review

Ol'ga Zhilyaeva
PhD in Medical Sciences, Associate Professor
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0009-0005-9896-8443

Viktoria Shishkina*
PhD in Medical Sciences, Associate Professor
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0000-0001-9185-4578

Svetlana Zolotareva
PhD in Biological Associate Professor
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0000-0002-2136-8272

Elena Ivanova
PhD in Medical Sciences, Associate Professor
Federal State Budgetary Educational Institution of Higher Education "Voronezh State Medical University named after N.N. Burdenko"
394036, 10 Studencheskaya Str., Voronezh, Russian Federation
https://orcid.org/0000-0001-8920-8059

Nazgul Tashmatova
PhD in Biological Associate Professor
Osh International Medical University
723500, 428 Alymbek Datka Str., Osh, Kyrgyz Republic
https://orcid.org/0000-0002-3068-1922

Kyialbek Sakibaev
PhD in Medical Sciences, Associate Professor
Osh International Medical University
723500, 428 Alymbek Datka Str., Osh, Kyrgyz Republic
https://orcid.org/0000-0001-7303-1252

Akmaral Argynbaeva
PhD in Biological Sciences, Associate Professor
Osh International Medical University
723500, 428 Alymbek Datka Str., Osh, Kyrgyz Republic
https://orcid.org/0000-0003-3835-2282

Abstract. Reducing infant mortality resulted from infections, particularly among premature infants, is a major public health concern. This fact emphasises the significance of transplacental transfer of maternal antibodies, including immunoglobulin G (IgG), to develop passive immunity and protect newborns from infections. The aim

Suggested Citation:
Zhilyaeva O, Shishkina V, Zolotareva S, Ivanova E, Tashmatova N, Sakibaev K, et al. Transplacental antibody perfusion in the development of the newborn passive immunity: A literature review. J Hum Biol Clin Med, 2026;2(1):28–39.

**Corresponding author*

of this studywas to summarise current findings on the mechanisms and factors affecting transplacental transfer of IgG and their role in the developing passive immunity in newborns. This review provided a systematic analysis of the scientific literature, including studies on the morphofunctional features of the placenta, the mechanisms of transplacental IgG perfusion, and their impact on fetal and neonatal immunological protection. The study supported the fact that IgG served a crucial function in the development of passive immunity in newborns, crossing the placental barrier and providing protection against infections in the early stages of life. As evidenced, the efficiency of transplacental IgG transfer varied depending on gestational age, with lower transfer rates in premature compared to full-term infants. The results demonstrated that different IgG subclasses (IgG1, IgG3, IgG4, and IgG2) were transferred across the placenta with varying efficiency, the fact associating with their affinity for the Fc receptor on the placenta. Infections and hypergammaglobulinemia in mothers were proved to affect transplacental IgG transfer, highlighting the need to monitor the infectious status of pregnant women to optimise neonatal protection. Maternal vaccination at specific stages of pregnancy was found to significantly increase IgG levels in the newborn's blood, contributing to improved passive immunity and reducing the risk of infectious diseases in infants. The study results can be introduced into clinical practice to optimise vaccination strategies for pregnant women and develop personalised approaches for preventing infectious diseases in newborns, especially among premature neonates

Keywords: placenta; transplacental transfer; immunoglobulin G; neonatal infections; immunological protection; placental barrier; gestational age

INTRODUCTION

Currently, reducing infant mortality appears to be one of the foremost challenges in healthcare, particularly among premature infants, for whom infections are a major cause of death. In this regard, the development of passive immunity, including through transplacental transfer of maternal antibodies, is an essential mechanism to protect the foetus and newborn. Immunoglobulin G (IgG) serves a crucial function in this process, being the only class of an antibody with the potential to cross the placental barrier and provide protection against infections early in life. However, despite the significance of this mechanism, existing data on transplacental IgG perfusion, as well as the influence of various factors on the effectiveness of this process, require further investigations to optimise infectious disease prevention in newborns.

The placenta, as a unique organ, has a complex functional morphology, combining immunomodulatory properties with a barrier function. As stated by L. Pomar & D. Baud [1], the placenta ensures immunological tolerance between the organisms of the mother and foetus, while simultaneously performing selective transport of biologically active substances. This emphasises the significance of its role in protecting the foetus from potentially dangerous agents, including viruses and bacteria, that can penetrate this barrier. E.N. Kravchenko *et al*. [2] specified the main risk factors for intrauterine infection of the foetus with the decreased placental barrier function, with viral infection being pivotal. It was also noted that disturbances in immune defence could contribute to a higher susceptibility of the foetus to diseases. In their study, W. Wang *et al*. [3] highlighted the importance of the cytokine profile and T-cell response in the favourable pregnancy completion and pathogenesis of its complications. This immune response also affected the interaction between the mother and foetus, and, consequently, pregnancy outcomes. M.P. Piccinni *et al*. [4] supported the primary role of the relationship between hormonal and immune mechanisms of the female reproductive function. These mechanisms maintained homeostasis and normal development of the foetus, and their disruption could result in complications such as preeclampsia. L.J. Yockey *et al*. [5] clearly demonstrated the effect of maternal antibodies on the developing passive immunity and increased resistance in the foetus and newborn. Maternal antibodies served as a crucial component of fetal protection from infections prior to birth. In addition, A.G. Rumyantsev [6], analysing maternal-fetal interactions in the developing immune system of the newborn, concluded that disruption of these interactions could cause immunodeficiencies in newborns. Of particular importance in terms of immune protection was the transplacental transfer of IgG, which provided the basis of passive immunity of the newborn. According to A.M. Ciobanu *et al*. [7], immunoglobulins G were the only class of antibodies that could penetrate the placental barrier and provided protection against neonatal infections, but simultaneously, the transfer of autoantibodies could contribute to the development of neonatal autoimmune diseases. Currently, research has significantly expanded our understanding of the mechanisms of transplacental immunoglobulins transport. Thus, A.N. Shuid *et al*. [8] reviewed the relationship between viral infections and the risk of developing autism disorders; as emphasised, it is crucial to immunologically protect the foetus from viral agents, including the effect of antibody transplacental transfer. These studies focused on viral infections as risk factors in the context of the developing immunity in newborns. Furthermore, L.L. Shook *et al*. [9] specified a relationship between the activity of the

maternal immune system exposed to SARS-cov-2 infection during pregnancy and impaired brain development in the foetus. Despite significant progress in understanding the mechanisms of transplacental transfer of antibodies, the issues of the dynamics of transplacental IgG transfer at different gestational stages are not fully understood. Identifying factors contributing to the effectiveness of transplacental transfer and changes in the qualitative composition of IgG depending on infection with various pathogens, as well as assessing the effectiveness of IgG perfusion after vaccination, also requires further research. The relevance of these issues necessitates a personalised approach to assessing and correcting passive immunity in newborns.

The aim of this study was to analyse current findings on the mechanisms of transplacental transfer of IgG and their role in the developing passive immunity in newborns, to identify factors contributing to the effectiveness of this process. This study provided a systematic review of the scientific literature, analysed data on the morphofunctional features of the placenta, the mechanisms of transplacental IgG perfusion, and their impact on fetal immunological protection. Studies examining the impact of gestational age, maternal immune status, infections, and vaccination on the transfer of antibodies across the placental barrier were included in the review. The dynamics and effectiveness of transplacental transfer of various IgG subclasses, such as IgG1, IgG2, IgG3, and IgG4, and their relationship with neonatal immune protection was also analysed.

The role of IgG subclasses in the development of maternal immunity

The phenomenon of the placenta, as an organ, lies in its unique functional morphology; normal pregnancy is characterised by a balance between immune tolerance to the foetus and protection from infections; the disruption of this balance can result in complications such as preeclampsia, intrauterine growth retardation, and premature birth. These conclusions were made in the review by D.E. Galkina *et al.* [10] on current knowledge of the immunological mechanisms of regular and pathological pregnancy. According to J. Ding *et al*. [11], while directly involved in the formation of the maternal immune response, the placenta simultaneously functioned as a selective barrier, thereby promoting immunological tolerance between two foreign organisms. One of the pathways of the mother's immunomodulatory effect on the developing immune responses in the offspring was the transplacental transfer of IgG and immunocompetent cells. C. Napodano *et al*. [12] presented current views on the functions of IgG subclasses and their role in immune defence, systematising the knowledge on the functions of IgG subclasses and their clinical significance, including transplacental transfer. IgG in the maternal body accounted for a significant proportion of the total number of antibodies and were the main factor in the humoral link of immune defence. V.A. Kozlov *et al*. [13] demonstrated that the presence of specific immunoglobulins of class G after an illness or vaccination indicated the formation of sterile immunity to the corresponding bacterial or viral infection. A decreased IgG level of any subclass might result from a violated synthesis of antibodies of a certain specificity. Pregnant women with a reduced level of IgG2 synthesis had weakened immune protection against polysaccharide antigens of the wall of some bacteria and viruses, such as pneumococci, meningococci, group A streptococci and Haemophilis influenzae. As reported in the basic research by A.K. Abbas *et al*. [14], if the production of IgG1 was more intense than the synthesis of immunoglobulins of other subclasses, then a decreased amount of IgG1 subclass antibodies might be due to general hypogammaglobulinemia, or associated with chronic, recurrent lower respiratory tract infections, or resulted from chronic fatigue syndrome. The IgG3 subclass was the most powerful complement activator, had a high affinity for protein antigens; its deficiency, often accompanied by a decrease in IgG1 antibodies, was observed in women with recurrent chronic infectious lung diseases. IgG4 antibodies developed an immune response to allergens and blocked IgE-dependent reactions, their concentration was normally lower than that of other subclasses, and, therefore, they were quite difficult to detect. Nevertheless, the data obtained by B.A Chernyak & I.I. Vorzheva [15] indicated that their deficiency might be associated with chronic antigenic stimulation combined with recurrent respiratory tract infections; however, this statement was ambiguous, since a low concentration of immunoglobulins of this subclass was observed quite often in healthy individuals.

The role of the neonatal Fc receptor (FcRn) in the selective transport of IgG across the placental barrier

R.W. Pitcher-Wilmott *et al.* [16] conducted one of the first studies related to the transplacental transfer of IgG subclasses and came to the following conclusions: IgG subclasses were transferred across the placenta with different efficiency: IgG1 > IgG3 > IgG4 > IgG2, the fact associating with different affinities of IgG subclasses to FcRn (neonatal Fc receptor). They also assumed that IgG transfer might be restricted by competition between subclasses for binding to FcRn. M.F. Jennewein *et al.* [17] specified the key role of the neonatal Fc receptor (FcRn) in the selective transport of IgG across the placental barrier. It was found that the efficiency of transfer of different IgG subclasses (IgG1-IgG4) varied significantly depending on the affinity to FcRn.

N. Twisselmann *et al.* [18] studied glycosylating Fc fragments of IgG in premature and full-term neonates. These studies were the first to link the features of IgG glycosylation with the efficiency of transplacental transfer and opened up new potentials to study the mechanisms of passive immunity development, emphasising the complexity and multifactorial nature of the process of IgG transplacental transfer. The major conclusions reached in the study indicated that premature infants had differences in IgG glycosylation compared to full-term infants, and these differences could affect IgG binding to FcRn and, as a result, the efficiency of transplacental transfer. IgG glycosylation in premature infants depended on the gestational age: the earlier the term of delivery, the less mature the glycosylation profile. S. Borghi *et al.* [19] also supported that the efficiency of IgG transfer depended on its subclass and glycosylation of the Fc fragment. K.M. Knudsen Sand *et al.* [20] applied experimental models to study the role of FcRn and FcγR (Fc-gamma receptors) in the IgG transplacental transfer, and also came to the conclusion that FcRn was the main receptor mediating the transplacental transfer of IgG; FcγR did not have a pivotal role in this process. Competition between IgG for binding to FcRn explained the inverse correlation between the level of maternal antibodies and their transfer. This was supported by N.A. Lozano *et al.* [21], who focused on the expression of the FcRn receptor, the main molecular carrier of immunoglobulins in the placenta. It was found that the efficiency of this transport directly correlated with IgG levels in full-term and premature infants, which highlighted the placental receptor apparatus as a critical control point of neonatal immunity.

Dynamics of IgG transplacental transfer depending on the gestational age

As reported by S.A. Ozdemir *et al.* [22] in their early retrospective study on IgG and IgM levels in premature and full-term neonates, the level of IgG in the blood serum of premature infants was significantly lower than that of full-term infants, and the IgG concentration correlated with gestational age: the earlier the term of delivery, the lower the IgG level. The level of IgM, on the contrary, did not depend on the gestational age, which confirmed its synthesis by the foetus itself, rather than its transfer from the mother. R.A. Pereira *et al.* [23] analysing the transplacental transfer of IgG and its subclasses in foetuses at different gestational stages also supported the universality of the "accumulative transfer" mechanism, regardless of the gestational age. Active transplacental transfer of maternal immunoglobulins began in the second trimester of pregnancy, approximately at the thirteenth week of gestation. The level of total IgG in the foetus increased with increasing gestational age. A relatively low concentration was observed between 17 and 22 weeks and constituted approximately 5-10% of the volume of maternal antibodies. According to R.A. Pereira *et al.,* passive diffusion of antibodies predominated during this period; at 32 weeks, approximately 1/2 of antibodies was determined; at birth, the amount of antibodies in the fetal blood exceeded the maternal level. This fact proved the concept of "accumulative transfer" and stressed the significance of the third trimester for the development of the fetal passive immunity. Further studies by S.D. Bokonbaeva *et al*. [24] provided detailed issues of immunity development in the most vulnerable groups of children – premature infants. The authors identified specific changes in the immune status that determined the severity of respiratory disorders in premature infants. van I.C. Duuren *et al.* [25] in their review considered the developing immune system of premature infants not only as a risk factor but also as a potential resource for new therapeutic solutions, demonstrating a link between immune immaturity and the subsequent development of respiratory tract infections and wheezing, providing immunological correction strategies.

A. Palmeira *et al.* [26] demonstrated that concentrations of total IgG and IgG subclasses in maternal plasma were positively associated with concentrations in fetal cord blood and negatively associated with transplacental transfer coefficients. Higher levels of total IgG and the IgG1 subtype were found in the cord blood of full-term newborns compared to the maternal blood. The efficiency of transplacental perfusion of immunoglobulin subclasses at full-term pregnancy (37-42 weeks) was expressed as IgG1 > IgG3 > IgG4 = IgG2, which demonstrated a relatively constant ratio of maternal/umbilical cord IgG subclasses and did not depend on the term of delivery after 37 weeks of gestation. However, it should be stressed that, as of 2026, there is no consensus regarding the hierarchy of transplacental transfer of different IgG subclasses.

Factors affecting the efficiency of transplacental IgG transfer

Study results by H. Akbulut *et al.* [27] complemented understanding of the variability of transplacental IgG transfer, focusing on ethnic and geographic features. Using data from a prospective cohort study of 120 mother-newborn pairs, they analysed the correlation between gestational age, maternal parity, and the efficiency of antibody transfer, measuring the levels of total IgG and subclasses (IgG1-IgG4) in maternal and cord blood using enzyme-linked immunosorbent assay (ELISA). As evidenced, the transfer coefficient of total IgG (cord/maternal concentration) was 1.2-1.5, supporting active transport. In full-term newborns, the IgG level exceeded the maternal level by 15-30%, the fact being consistent with the data obtained by R.A. Pereira *et al.* [23]. The transfer hierarchy was as follows: IgG1 (1.4) > IgG3 (1.1) > IgG4 (0.9) > IgG2 (0.7). Low IgG2 transfer was explained by its weak binding to FcRn. Women from Eastern Anatolia had elevated

levels of total IgG and IgG1 compared to Western populations. In children born before 34 weeks, IgG levels were 40% lower than in full-term infants, as reported by S.A. Ozdemir *et al.* [22]. Multiparous women (>3 births) had higher IgG1 levels but reduced efficiency of its transfer (coefficient 1.1 vs. 1.4 in primiparous women). Previously, S.S. Omowale *et al.* [28] evidenced a direct relationship between maternal hypergammaglobulinemia and a decreased IgG1 and IgG2 transfer without affecting IgG3 and IgG4, compared to maternal IgG in malaria; this being the proof-of-concept for the decreased efficiency of transplacental IgG perfusion in hypergammaglobulinemia in pregnant women. A similar effect was observed in Black women, whose total IgG levels were initially higher than in light-skinned women. Based on the results obtained, A. Ohlsson & J.B. Lacy [29] concluded that transplacental transfer correlated with maternal race. Older pregnant women had lower total plasma IgG levels, but this did not affect the efficiency of transplacental perfusion and antibody levels in the cord blood. Women, who had previously had live births, manifested higher levels of total IgG and IgG1 at birth. However, this did not affect the blood of newborns, and the transfer coefficient was reduced. This provided further evidence that, despite the positive correlation between IgG levels in the maternal and umbilical cord blood, transplacental capacity might be limited by potential saturation of Fc transport receptors at higher IgG concentrations. No effect of parity of pregnant women on the transfer of antigen-specific IgG or changes in their concentration in the blood of the umbilical cord and placental vessels was revealed. L.N. Balanovskaya & Ya.A. Loginova [30] also analysed the features of newborn immunity in different countries, thus, supporting an essential effect of external factors and regional epidemiological conditions on the "programming" of the immune system from birth.

Mechanisms of competition between IgG subclasses and their clinical significance

As demonstrated, there was observed a negative correlation between maternal total IgG levels and the transfer coefficients of the IgG1, IgG2, and IgG3 subclasses during normal pregnancy. This suggested that higher levels of IgG1, IgG2, and IgG3 competed with other subclasses for placental transfer. It should be noted that maternal blood concentrations of IgG2, IgG3, and IgG4 correlated with IgG1. This effect might not always be indicative of a specific antibody subclass but was due to a higher concentration of IgG1, which was more efficiently transferred. Furthermore, elevated maternal IgG4 levels had limited correlation with total IgG transfer, and IgG4 perfusion did not correlate with maternal antibodies, despite the positive correlation between maternal IgG4 and IgG1, as suggested by T. Clements *et al.* [31].

The highest correlation was observed between the cord blood IgG4 concentrations and total IgG concentrations in both the cord blood and maternal blood, suggesting no relationship between the transfer of antibodies of this subclass and maternal antibody titre variability. In this case, there was a certain probability to infer the mechanism underlying the development of passive immunity in newborns against specific pathogens. The clinical significance of the IgG4 immunoglobulins transfer was pivotal and required further research. Conclusions supported the current views that higher maternal IgG levels contributed to increased antibody concentrations in the offspring, but the relative efficiency of placental immunoglobulin transfer might be reduced by maternal hyperglobulinemia. B.J. Okoko *et al*. [32] in their study demonstrated that infectious diseases (in particular malaria) and hypergammaglobulinemia in the mother could significantly reduce the transport of IgG and its subclasses, making the newborn vulnerable.

Studies of the immune status of children born at later stages of uncomplicated pregnancy demonstrated significantly higher levels of IgG1 and IgG4. The increase in IgG4 concentration was likely due to the high level of IgG4 in the mother's body with an unchanged rate of transplacental transfer. The IgG1 level was not associated with the transfer coefficient and increased transplacental activity in late weeks but reflected the cumulative effect in the fetal circulation. In other words, in full-term pregnancy, each additional week of delay in delivery increased neonatal levels of IgG1 and IgG4. N.A. Lozano *et al*. [21] detected a positive relationship between the total IgG level and antigen-specific IgG in premature infants depending on the term of premature delivery. The clinical block of publications clearly demonstrated the assessment of the immune status of a premature infant as a dynamic system with predictable risks. E.N. Volkova & L.I. Ippolitova [33] in their study related to TREC and KREC markers allowed for an objective assessment of the "naivety" of the immune system and the potential for the development of immune components, the fact providing physicians with a tool for the early identification of children with critically low adaptive resources and the risk of developing infectious complications. The study by H. Lu *et al*. [34] introduced the concept of ferroptosis – programmed cell death – into neonatal practice and opened new horizons in understanding the mechanisms of hypoxic-ischemic encephalopathy. This might result in developing targeted therapy with the potential to minimise brain damage in critically ill newborns.

Effect of vaccination and infections on the transplacental IgG transfer

A key aspect of research is the study of the impact of maternal pathological conditions on the efficiency of transplacental antibody transfer. S. Atyeo *et al*. [35] greatly contributed to addressing issues related to the

impact of maternal infections, including COVID-19, on the profile of transplacentally transferred antibodies. It was proven that viral infections altered the qualitative composition of transferred IgG; there were identified significant differences in the transfer of antibodies to various pathogens, which in turn affected the efficiency of neonatal protection. Multiple pregnancy deserves special attention as a specific risk factor. S.C.L. Stach *et al.* [36] in their study demonstrated the distribution patterns of total IgG and specific antibodies (to Klebsiella, Pseudomonas, and group B Streptococcus) in twins. These data indicated that the placental transport resource might be limited or distributed unevenly, which required a differentiated approach to assessing infectious risks in each twin.

Publications related to the clinical segment discuss the issues of therapeutic correction and prevention. R.K. Satlikov & I.Yu. Masharipova [37] in their study supported the effectiveness of using anti-Rhesus immunoglobulin as the gold standard for the prevention of hemolytic disease; this was a successful example of passive immunity management. Immunological examination of children born to Rh-negative mothers demonstrated decreased IgG2 levels and lower IgG2 transfer coefficients after the anti-D IgG introduction at the 28th week of pregnancy. The parameters of other IgG subclasses were comparable with those in the control group. This suggested that the administered IgG could compete with the transfer of endogenous IgG2. O.V. Kozlyakov *et al*. [38] addressed the urgent issues of expanding the screening of pregnant women. The emphasis the authors had taken on the Rh factor (D antigen) following the conventional approach was complemented by their investigations of other anti-erythrocyte antibodies (Kell, Duffy, Kidd systems etc.). The authors analysed the need for specific prophylaxis in cases of combined sensitisation, pointing out that the presence of other antibodies could modulate the immune response and complicate the course of pregnancy, even if Rh prophylaxis was performed in a timely manner. This validated the transition from routine examination to extended immunological typing of couples. The results obtained by V.E. Potapova *et al.* [39] indirectly supported the above, as they focused on the fundamental aspects of hemolytic disease of the newborn, considering this condition not only as a process of hemolysis of fetal erythrocytes under the influence of maternal IgG, but also as a complex reaction affecting erythropoiesis in the fetal bone marrow. The authors emphasised that the severity of the damage depended on the expressivity of antigens, the subclass of maternal antibodies (mainly IgG1 and IgG3) and the functional state of placental Fc receptors, thus identifying the prognosis for the newborn.

Z. Zhong *et al.* [40] in their study provided essential data on the immunization timing. In particular, using influenza vaccination as an example, they demonstrated that the timing of maternal vaccine administration directly determined the titre of protective antibodies in the infant at birth. As reported, there was a "golden window" for vaccination, which allowed for the most effective use of the mechanism of active transport across the placenta providing passive immunity to the child in the first months of life. The highest concentrations of antibodies in newborns were detected if mothers were vaccinated 4-24 weeks before delivery, while the timing of vaccination of the pregnant woman did not affect the efficiency of transplacental transfer of IgG subclasses. A critical contribution to the evidenced safety of immunisation during pregnancy was the study conducted by E.B. Efimkova *et al.* [41]. Based on a retrospective analysis of the pandemic period (2020-2023), the effect of vaccination on obstetric outcomes was provided. The researchers argued that vaccination of pregnant women against COVID-19 did not only protect the mother from severe infection but also promoted a favourable gestational process. In the studies performed by M.P. Kostinov *et al.* [42], it was highlighted that vaccination should be considered as part of pre-pregnancy preparation and management of pregnancy, thus inherently developing a modern paradigm for vaccination in obstetrics. V.I. Krasnopolsky *et al*. [43] convincingly demonstrated based on the extensive clinical material that vaccination against the human papillomavirus (even performed in periods close to conception or in the early stages before pregnancy was detected) had no teratogenic effect and did not worsen gestational outcomes.

Based on the data obtained, it can be concluded that transplacental antibody transfer is crucial for developing passive immunity in newborns. The presence of specific antibodies in the mother significantly enhances the foetus's immune defence, especially in cases of vaccination or previous infections. However, the effectiveness of this process varies depending on diverse factors, such as gestational age, maternal age, and the impact of infectious diseases.

CONCLUSIONS

Immunoglobulin G (IgG) are pivotal in fetal protection, forming the basis of maternal humoral immunity. IgG1, the most common subclass, protects against protein antigens and activates complement. Decreased IgG1 levels are associated with hypogammaglobulinemia and chronic respiratory infections. IgG2 deficiency increases the risk of bacterial infections, and decreased IgG3 is observed in chronic respiratory diseases. IgG4 involved in allergic reactions has the lowest concentration among the subclasses, and its deficiency is associated with chronic antigen stimulation.

Transplacental transfer of IgG begins in the second trimester and peaks by 32 weeks, when fetal IgG levels exceed maternal levels providing passive immunity. Premature infants have lower IgG concentrations, this fact increasing their vulnerability to infections. Full-term infants exhibit higher levels of IgG1

and IgG4, and the ratio of IgG1 > IgG3 > IgG4 = IgG2 subclasses remains constant. Transfer efficiency depends on maternal antibody levels, age, race, and hypergammaglobulinemia, which reduces the transfer of IgG1 and IgG2.

Vaccination during pregnancy strengthens the passive immunity of newborns. Influenza vaccination increases IgG4 levels in both mother and foetus. However, infections such as malaria and COVID-19 can disrupt transplacental antibody transfer. Hypergammaglobulinemia associated with malaria reduces the transfer of IgG1 and IgG2 highlighting the need to control infections in pregnant women. It is essential to study the molecular mechanisms of placental FcRn receptors and develop diagnostic models for assessing newborn immune defences. This will enable the development of personalised prenatal immunocorrection protocols and vacciWWnation programs to improve neonatal outcomes.

ACKNOWLEDGEMENTS

None.

FUNDING

None.

CONFLICT OF INTEREST

None.

REFERENCES

[1] Pomar L, Baud D. Special issue “Emerging virus infections in adverse pregnancy outcomes”. Viruses. 2022;14(2):285. DOI: 10.3390/v14020285
[2] Kravchenko EN, Kuklina LV, Shevlyukova TP. Intrauterine infection: Risk factors and diagnostic challenges during pregnancy. Obst Gynecol: News Opin Train. 2023;11(4):35–44. DOI: 10.33029/2303-9698-2023-11-4-35-44
[3] Wang W, Sung N, Gilman-Sachs A, Kwak-Kim J. T helper (Th) cell profiles in pregnancy and recurrent pregnancy losses: Th1/Th2/Th9/Th17/Th22/Tfh cells. Front Immunol. 2020;11:2025. DOI: 10.3389/fimmu.2020.02025
[4] Piccinni MP, Raghupathy R, Saito S, Szekeres-Bartho J. Cytokines, hormones and cellular regulatory mechanisms favoring successful reproduction. Front Immunol. 2021;12:717808. DOI: 10.3389/fimmu.2021.717808
[5] Yockey LJ, Lucas C, Iwasaki A. Contributions of maternal and fetal antiviral immunity in congenital disease. Science. 2020;368(6491):eaz1960. DOI: 10.1126/science.aaz1960
[6] Rumyantsev AG. The role of innate fetal immunity factors, microbiome, and maternal infections in the formation of perinatal pathology in newborns. Pediatria. 2025;104(1):8–14. DOI: 10.24110/0031-403X-2025-104-1-8-14
[7] Ciobanu AM, Dumitru AE, Gica N, Botezatu R, Peltecu G, Panaitescu AM. Benefits and risks of IgG transplacental transfer. Diagnostics. 2020;10(8):583. DOI: 10.3390/diagnostics10080583
[8] Shuid AN, Jayusman PA, Shuid N, Ismail J, Kamal Nor N, Mohamed IN. Association between viral infections and risk of autistic disorder: an overview. Int J Environ Res Public Health. 2021;18(6):2817. DOI: 10.3390/ijerph18062817
[9] Shook LL, Batorsky RE, De Guzman RM, McCrea LT, Brigida SM, Horng JE, et al. Maternal SARS-CoV-2 impacts fetal placental macrophage programs and placenta-derived microglial models of neurodevelopment. J Neuroinflammation. 2024;21(1):163. DOI: 10.1186/s12974-024-03157-w
[10] Galkina DE, Makarenko TA, Okladnikov DV. Immunological aspects of normal and pathological pregnancy. Bull Russ Acad Med Sci. 2022;77(1):13–24. DOI: 10.15690/vramn1507
[11] Ding J, Maxwell A, Adzibolosu N, Hu A, You Y, Liao AH, et al. Mechanisms of immune regulation by the placenta: Role of type I interferon and interferon-stimulated genes signaling during pregnancy. Immunol Rev. 2022;308(1):9–24. DOI: 10.1111/imr.13077
[12] Napodano C, Marino MP, Stefanile A, Pocino K, Scatena R, Gulli F, et al. Immunological role of IgG subclasses. Immunol Investig. 2020;50:427–44. DOI: 10.1080/08820139.2020.1775643
[13] Kozlov VA, Savchenko AA, Simbirtsev AS, Kudlai DA, Anisimova EN, Borisov AG. Immunopathological syndromes associated with adaptive immunity. Cytokine Inflamm. 2024;21(4):186–95. DOI: 10.17816/CI679042
[14] Abbas AK, Lichtman AH, Pillai S. Basic immunology: Functions and disorders of the immune system. 7th ed. Philadelphia, PA: Elsevier Health Sciences; 2023. 352 P.
[15] Chernyak BA, Vorzheva II. Pathology of the respiratory system in primary immunodeficiencies. In: Respiratory medicine: A guide in 5 volumes. 3rd ed. Moscow: Pulmomedia LLC; 2024. P. 386–421. DOI: 10.18093/978-5-6052509-1-3-2024-4-386-421
[16] Pitcher-Wilmott RW, Hindocha P, Wood CB. The placental transfer of IgG subclasses in human pregnancy. Clin Exp Immunol. 1980;41(2):303–8.
[17] Jennewein MF, Goldfarb I, Dolatshahi S, Cosgrove C, Noelette FJ, Krykbaeva M, et al. Fc glycan-mediated regulation of placental antibody transfer. Cell. 2019;178(1):202–15. DOI: 10.1016/j.cell.2019.05.044
[18] Twisselmann N, Bartsch YC, Pagel J, Wieg C, Hartz A, Ehlers M, et al. IgG Fc glycosylation patterns of preterm infants differ with gestational age. Front Immunol. 2019;9:3166. DOI: 10.3389/fimmu.2018.03166

[19] Borghi S, Bournazos S, Thulin NK, Li C, Gajewski A, Sherwood RW, et al. FcRn, but not FcγRs, drives maternal-fetal transplacental transport of human IgG antibodies. Proc Natl Acad Sci U S A. 2020;117(23):12943–51. DOI: 10.1073/pnas.2004325117
[20] Knudsen Sand KM, Gruber MM, Sandlie I, Mathiesen L, Andersen JT, Wadsack C. Contribution of the *ex vivo* placental perfusion model in understanding transplacental immunoglobulin G transfer. Placenta. 2022;127:77–87. DOI: 10.1016/j.placenta.2022.07.019
[21] Lozano NA, Lozano A, Marini V, Saranz RJ, Blumberg RS, Baker K, et al. Expression of FcRn receptor in placental tissue and its relationship with IgG levels in term and preterm newborns. Am J Reprod Immunol. 2018;80(3):e12972. DOI: 10.1111/aji.12972
[22] Ozdemir SA, Ozer EA, Kose S, Ilhan O, Ozturk C, Sutcuoglu S. Reference values of serum IgG and IgM levels in preterm and term newborns. J Matern Fetal Neonatal Med. 2016;29(6):472–6. DOI: 10.3109/14767058.2015.1027680
[23] Pereira RA, Oliveira de Almeida V, Vidori L, Colvero MO, Amantéa SL. Immunoglobulin G and subclasses placental transfer in fetuses and preterm newborns: A systematic review. J Perinatol. 2023;43(1):3–9. DOI: 10.1038/s41372-022-01528-w
[24] Bokonbaeva SD, Urmatova BK, Zeivald SV, Asanbekova ZhA. Immune status and inflammatory mediators in premature newborns with respiratory distress syndrome. Sci Rev Med Sci. 2025;(2):16–21. DOI: 10.17513/srms.1436
[25] van Duuren IC, van Hengel ORJ, Penders J, Duijts L, Smits HH, Tramper-Stranders GA. The developing immune system in preterm born infants: From contributor to potential solution for respiratory tract infections and wheezing. Allergy. 2024;79(11):2924–42. DOI: 10.1111/all.16342
[26] Palmeira A, Sousa E, Köseler A, Sabirli R, Gören T, Türkçüer İ, et al. Correction: Palmeira et al. Preliminary virtual screening studies to identify GRP78 inhibitors which may interfere with SARS-CoV-2 infection. Pharmaceuticals (Basel). 2024;17(4):411. DOI: 10.3390/ph17040411
[27] Akbulut H, Çelik İ, Çelik A, Akbulut A, Ayar A. Placental transfer of total IgG and IgG subclasses in a Turkish population living in eastern Anatolia. Nobel Med. 2012;8(2):59–64.
[28] Omowale SS, Gary-Webb TL, Wallace ML, Wallace JM, Rauktis ME, Eack SM, et al. Stress during pregnancy: An ecological momentary assessment of stressors among black and white women with implications for maternal health. Womens Health (Lond). 2022;18:17455057221126808. DOI: 10.1177/17455057221126808
[29] Ohlsson A, Lacy JB. Intravenous immunoglobulin for suspected or proven infection in neonates. Cochrane Database Syst Rev. 2020;2020(1):CD001239. DOI: 10.1002/14651858.CD001239.pub6
[30] Balanovskaya LN, Loginova YA. Features of newborns' immunity in different countries. In: Interdisciplinary immunology: Prospects for development: Materials of the II all-Russian scientific and practical conference of students and young scientists. Kirov: Kirov State Medical University; 2025. P. 20–1.
[31] Clements T, Rice TF, Vamvakas G, Barnett S, Barnes M, Donaldson B, et al. Update on transplacental transfer of IgG subclasses: Impact of maternal and fetal factors. Front Immunol. 2020;11:1920. DOI: 10.3389/fimmu.2020.01920
[32] Okoko BJ, Wesumperuma LH, Ota MOC, Pinder M, Banya W, Gomez SF, et al. The influence of placental malaria infection and maternal hypergammaglobulinemia on transplacental transfer of antibodies and IgG subclasses in a rural West African population. J Infect Dis. 2001;184(5):627–33. DOI: 10.1086/322808
[33] Volkova EN, Ippolitova LI. Markers of the cellular immune response (TREC and KREC) in newborns of different gestational age. Verkhnevolzhsky Med J. 2025;24(1):35–8.
[34] Lu H, Shen H, Mao L, Mussap M, Song L. A ferroptosis-related ceRNA network for investigating the molecular mechanisms and the treatment of neonatal hypoxic-ischemic encephalopathy. Transl Pediatr. 2024;13(1):119–36. DOI: 10.21037/tp-23-596
[35] Atyeo C, DeRiso EA, Davis C, Bordt EA, De Guzman RM, Shook LL, et al. COVID-19 mRNA vaccines drive differential antibody Fc-functional profiles in pregnant, lactating, and nonpregnant women. Sci Transl Med. 2021;13(617):eabi8631. DOI: 10.1126/scitranslmed.abi8631
[36] Stach SCL, Brizot ML, Liao AW, Palmeira P, Francisco RPV, Carneiro-Sampaio MMS, et al. Placental transfer of IgG antibodies specific to *Klebsiella* and *Pseudomonas* LPS and to group B *Streptococcus* in twin pregnancies. Scand J Immunol. 2015;81(2):13541. DOI: 10.1111/sji.12258
[37] Satlikov RK, Masharipova IYu. Management tactics of pregnancy and treatment with anti-Rh immunoglobulin of pregnant women with negative Rh factor. Internauka. 2021;(5–1):46–8.
[38] Kozlyakova OV, Klimovich OV, Goldinberg BM, Kamalova OV, Rak MA, Avtukhova TE, et al. The first results of immunological screening of pregnant women. Is there a need for specific prevention of Rh-immunization in the presence of another type of anti-erythrocyte antibodies? Reprod Health East Eur. 2021;11(2):177–84. DOI: 10.34883/PI.2021.11.2.004
[39] Potapova VE, Prishchepenko OA, Lysenko IM. Modern concepts of the pathogenesis of hemolytic disease of newborns. Prot Mother Child. 2020;(2):95–101.

[40] Zhong Z, Haltalli M, Holder B, Rice T, Donaldson B, O'Driscoll M, et al. The impact of timing of maternal influenza immunization on infant antibody levels at birth. Clin Exp Immunol. 2019;195(2):139–52. DOI: 10.1111/cei.13234
[41] Efimkova EB, Dulaeva EV, Kravtsova ON. SARS-CoV-2 vaccination of pregnant women during the 2020-2023 pandemic: Implications for obstetric and perinatal outcomes. Doctor Ru. 2025;24(5):68–73. DOI: 10.31550/1727-2378-2025-24-5-68-73
[42] Kostinov MP, Markelova EV, Svitich OA, Polishchuk VB. Immune mechanisms of SARS-CoV-2 and potential drugs in the prevention and treatment of COVID-19. Pulmonologiya. 2020;30(5):700–8. DOI: 10.18093/0869-0189-2020-30-5-700-708
[43] Krasnopolsky VI, Petrukhin VA, Zarochentseva NV, Belaya YM, Bocharova II, Vodovatova VA, et al. The features of the course of pregnancy and its outcomes in women vaccinated against papillomavirus infection. Obstet Gynecol. 2020;1:146–54. DOI: 10.18565/aig.2020.1.146-15

Жаңы төрөлгөн баладагы пассивдүү иммунитеттин калыптанышына антителолордун трансплацентардык перфузиясынын ролу: адабияттарга сереп

Ольга Жиляева
Медицина илимдеринин кандидаты, доцент
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси «Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0009-0005-9896-8443

Виктория Шишкина
Медицина илимдеринин кандидаты, доцент
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси «Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0000-0001-9185-4578

Светлана Золотарева
Биология илимдеринин кандидаты, доцент
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси «Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0000-0002-2136-8272

Елена Иванова
Медицина илимдеринин кандидаты, доцент
Федералдык мамлекеттик бюджеттик жогорку билим берүү мекемеси «Н.Н. Бурденко атындагы Воронеж мамлекеттик медициналык университети»
394036, Студенческая көч., 10, Воронеж ш., Россия Федерациясы
https://orcid.org/0000-0001-8920-8059

Назгул Ташматова
Биология илимдеринин кандидаты, доцент
Ош эл аралык медициналык университети
723500, Алымбек Датка көч., 428, Ош ш., Кыргыз Республикасы
https://orcid.org/0000-0002-3068-1922

Кыялбек Сакибаев
Кандидат медицинских наук, доцент
Ош эл аралык медициналык университети
723500, Алымбек Датка көч., 428, Ош ш., Кыргыз Республикасы
https://orcid.org/0000-0001-7303-1252

Акмарал Аргынбаева
Биология илимдеринин кандидаты, доцент
Ош эл аралык медициналык университети
723500, Алымбек Датка көч., 428, Ош ш., Кыргыз Республикасы
https://orcid.org/0000-0003-3835-2282

Аннотация. Коомдук саламаттыкты сактоонун негизги көйгөйлөрүнүн бири – жугуштуу оорулардан улам ымыркайлардагы, айрыкча ара төрөлгөндөрүнүн арасындагы өлүмдү азайтуу болуп саналат. Энедеги иммуноглобулин G (IgG) сыяктуу антителолордун трансплацентардык жол менен өткөрүлүп берилүүсү ымыркайда пассивдүү иммунитеттин калыптанышына жана ага жугуштуу оорулардан коргонуусуна чон мааниси бар. Изилдөөнүн максаты IgGнин трансплацентардык жол менен өткөрүлүшүнө таасир этүүчү механизмдерди жана факторлорду аныктоо, алардын жаңы төрөлгөн балдарда пассивдүү иммунитеттин калыптанышындагы ролу боюнча учурдагы маалыматтарды жалпылоо болуп саналат. Бул адабий серепте плацентанын морфофункционалдык өзгөчөлүктөрүнө, трансплацентардык IgG перфузиясынын механизмдерине жана алардын түйүлдүктүн жана жаңы төрөлгөн баланын иммунологиялык корголушуна тийгизген таасирине арналган эмгектерди камтыган илимий адабияттардын системалуу анализи колдонулган. IgG жаңы төрөлгөн балдарда пассивдүү иммунитеттин калыптанышында, плаценталык тосмодон өтүп, жашоонун алгачкы этаптарында инфекциялардан коргоонуну камсыз кылууда маанилүү ролду ойной турганын изилдөө тастыктады. Трансплацентардык IgG өткөрүп берүүнүн натыйжалуулугу кош бойлуулуктун курагына жараша өзгөрүп тураары, толук мөөнөттүү ымыркайларга салыштырмалуу ара төрөлгөн ымыркайларда өткөрүп берүү көрсөткүчү төмөн экени аныкталган. Кош бойлуулуктун айрым этаптарында эненин эмдөөсү жаңы төрөлгөн баланын канындагы IgG деңгээлин бир топ жогорулатары аныкталды, бул пассивдүү иммунитетти жакшыртууга жана ымыркайларда жугуштуу оорулардын коркунучун азайтууга жардам берет. Изилдөөнүн жыйынтыктарын практикада оптималдаштыруу үчүн колдонсо болот. Изилдөөнүн жыйынтыктары кош бойлуу аялдарды эмдөө стратегияларын оптималдаштыруу жана жаңы төрөлгөн балдарда, айрыкча ара төрөлгөн балдарда жугуштуу оорулардын алдын алуунун жекелештирилген ыкмаларын иштеп чыгуу үчүн практикада колдонулушу мүмкүн

Негизги сөздөр: плацента; трансплацентардык өткөрүү; G-иммуноглобулиндер; жаңы төрөлгөн балдардын инфекциялары; иммунологиялык коргоо; плацентардык тосмо; гестациялык курагы

Роль трансплацентарной перфузии антител в формировании пассивного иммунитета новорожденного: обзор литературы

Ольга Жиляева
Кандидат медицинских наук, доцент
Федеральное государственное бюджетное образовательное учреждение высшего образования «Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0009-0005-9896-8443

Виктория Шишкина
Кандидат медицинских наук, доцент
Федеральное государственное бюджетное образовательное учреждение высшего образования «Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0000-0001-9185-4578

Светлана Золотарева
Кандидат биологических наук, доцент
Федеральное государственное бюджетное образовательное учреждение высшего образования «Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0000-0002-2136-8272

Елена Иванова
Кандидат медицинских наук, доцент
Федеральное государственное бюджетное образовательное учреждение высшего образования «Воронежский государственный медицинский университет имени Н.Н. Бурденко»
394036, ул. Студенческая, 10, г. Воронеж, Российская Федерация
https://orcid.org/0000-0001-8920-8059

Назгул Ташматова
Кандидат биологических наук, доцент
Ошский международный медицинский университет
723500, ул. Алымбек Датка, 428, г. Ош, Кыргызская Республика
https://orcid.org/0000-0002-3068-1922

Кыялбек Сакибаев
Кандидат медицинских наук, доцент
Ошский международный медицинский университет
723500, ул. Алымбек Датка, 428, г. Ош, Кыргызская Республика
https://orcid.org/0000-0001-7303-1252

Акмарал Аргынбаева
Кандидат биологических наук, доцент
Ошский международный медицинский университет
723500, ул. Алымбек Датка, 428, г. Ош, Кыргызская Республика
https://orcid.org/0000-0003-3835-2282

Аннотация. Одной из ключевых проблем здравоохранения является снижение младенческой смертности, особенно среди недоношенных детей, вызванной инфекциями, что подчеркивает важность трансплацентарного переноса материнских антител, в том числе иммуноглобулинов G (IgG), для формирования пассивного иммунитета и защиты новорожденных от инфекций. Целью исследования являлось обобщение современных данных о механизмах и факторах, влияющих на трансплацентарный перенос IgG, и их роли в формировании пассивного иммунитета новорожденных. В обзоре использован систематический анализ научной литературы, включая работы, посвященные морфофункциональным особенностям плаценты, механизмам трансплацентарной перфузии IgG и их влиянию на иммунологическую защиту плода и новорожденного. Исследование подтвердило, что IgG играют ключевую роль в формировании пассивного иммунитета у новорожденных, преодолевая плацентарный барьер и обеспечивая защиту от инфекций на ранних этапах жизни. Установлено, что эффективность

трансплацентарного переноса IgG варьирует в зависимости от срока гестации, с более низким уровнем переноса у недоношенных детей по сравнению с доношенными. Результаты показали, что различные подклассы IgG (IgG1, IgG3, IgG4 и IgG2) переносятся через плаценту с разной эффективностью, что связано с их аффинностью к Fc-рецептору на плаценте. Влияние инфекций и гипергаммаглобулинемии у матерей на трансплацентарный перенос IgG было подтверждено, что подчеркивает необходимость мониторинга инфекционного статуса беременных для оптимизации защиты новорожденных. Выявлено, что вакцинация матери в определенные сроки беременности существенно повышает уровни IgG в крови новорожденного, что способствует улучшению пассивного иммунитета и снижению риска инфекционных заболеваний у младенцев. Результаты исследования могут быть использованы на практике для оптимизации стратегии вакцинации беременных и разработки персонализированных методов профилактики инфекционных заболеваний у новорожденных, особенно среди недоношенных детей

Ключевые слова: плацента; трансплацентарный перенос; иммуноглобулины G; неонатальные инфекции; иммунологическая защита; плацентарный барьер; гестационный возраст