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HIV-Induced Immune Activation in Pediatric Populations

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Abstract

HIV-induced immune activation is a critical aspect of the pathogenesis of the disease, particularly in pediatric populations. This review explores the mechanisms underlying immune activation in HIV-infected children, highlighting the consequences of chronic inflammation and immune dysregulation. The infection directly impacts the immune system, leading to increased levels of pro-inflammatory cytokines, dysregulated T-cell responses, and heightened susceptibility to opportunistic infections and co-morbidities. The role of antiretroviral therapy (ART) in managing immune activation is also discussed, emphasizing that while ART significantly reduces viral load and improves immune function, it may not fully restore immune homeostasis in affected children. Persistent immune activation can occur even in those receiving effective treatment, necessitating comprehensive monitoring and management strategies to address this ongoing challenge.

Keywords*: HIV, immune activation, pediatric populations, immune response, inflammation*

Introduction

HIV infection poses a significant public health challenge globally, with an estimated 1.7 million children living with the virus as of 2021. Pediatric populations are particularly vulnerable to the effects of HIV due to their developing immune systems, which are still maturing in terms of both function and complexity. One of the most critical consequences of HIV infection in children is immune activation, a state characterized by chronic inflammation and dysregulated immune responses. Immune activation in HIV-infected individuals is primarily driven by the virus itself, which directly infects and depletes CD4+ T-cells, the key orchestrators of the adaptive immune response. The loss of these cells leads to a compensatory increase in the activation of CD8+ T-cells and other immune cells, resulting in a chronic inflammatory state. In children, this immune activation can have profound consequences, as their immune systems may not be equipped to handle the sustained inflammatory response, leading to increased susceptibility to infections and complications. Additionally, HIV-induced immune activation is compounded by the presence of co-infections, which are common in pediatric populations. Co-infections with pathogens such as tuberculosis, hepatitis viruses, and respiratory viruses can exacerbate the inflammatory response, further impairing immune function. The interplay between HIV and these co-infecting agents underscores the complexity of managing immune activation in children, necessitating a nuanced understanding of how these interactions influence health outcomes.[1-5]

Antiretroviral therapy (ART) has dramatically transformed the prognosis for children living with HIV, allowing many to achieve viral suppression and improve immune function. However, while ART is effective at reducing viral loads, it does not entirely eliminate immune activation. Some

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level of immune dysregulation may persist even in those receiving effective treatment, leading to ongoing health challenges. Research has shown that chronic immune activation can lead to a range of complications in HIV-infected children, including increased rates of opportunistic infections, autoimmune disorders, and non-infectious comorbidities such as cardiovascular disease. The implications of sustained inflammation and immune dysregulation are profound, impacting not only the immediate health of pediatric patients but also their long-term quality of life. Consequently, addressing immune activation is an essential component of comprehensive HIV care for children. The management of HIV-induced immune activation involves several key strategies, including early initiation of ART, regular monitoring of immune function, and proactive management of co-infections. Clinicians must be vigilant in assessing immune status and adjusting treatment plans to address ongoing immune dysregulation. Additionally, vaccination plays a crucial role in protecting HIV-infected children from preventable infections, further highlighting the need for tailored immunization strategies in this population.[6-10] Despite advances in treatment and understanding, gaps remain in the knowledge of how to best manage immune activation in pediatric populations. Ongoing research into the mechanisms of HIV-induced immune activation and the effects of ART on immune health is critical for informing clinical practice. Innovations in treatment strategies, including adjunctive therapies aimed at reducing inflammation, may provide additional benefits for managing immune activation in HIV-infected children.

Mechanisms of HIV-Induced Immune Activation

HIV-induced immune activation is a complex process that arises from various interactions between the virus and the host immune system. Understanding the mechanisms behind this immune activation is crucial for developing effective strategies to mitigate its effects, particularly in pediatric populations. Several key mechanisms contribute to HIV-induced immune activation, including direct viral effects, chronic antigen stimulation, the role of innate immune responses, and the impact of co-infections. HIV primarily targets CD4+ T-cells, which are essential for orchestrating adaptive immune responses. Upon infection, HIV hijacks the cellular machinery of these immune cells, leading to their depletion and dysfunction. The loss of CD4+ T-cells triggers compensatory mechanisms in the immune system, resulting in increased activation of CD8+ T-cells and other immune cells. This hyperactivation leads to a chronic state of immune activation, characterized by the production of pro-inflammatory cytokines and the upregulation of activation markers. In individuals living with HIV, the continuous presence of viral antigens leads to sustained immune activation. Even in individuals on effective antiretroviral therapy (ART), residual viral replication can maintain antigenic stimulation. This chronic exposure to HIV antigens causes T-cells to become functionally exhausted, characterized by reduced proliferative capacity and diminished effector functions. The persistent antigen load drives the immune system to remain in an activated state, contributing to the chronic inflammation often observed in HIV-infected individuals.[11-15]

The innate immune system plays a pivotal role in the early response to HIV infection. Dendritic cells, macrophages, and natural killer (NK) cells are crucial in recognizing and responding to HIV. Upon recognition of the virus, these innate immune cells become activated and secrete pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). While these cytokines help to initiate an immune response, their prolonged production can contribute to systemic inflammation and further exacerbate immune activation. Chronic HIV

infection is associated with elevated levels of various cytokines and chemokines that promote inflammation. Key cytokines, including TNF-α, IL-1, IL-6, and interferon-gamma (IFN-γ), are often found at increased levels in HIV-infected individuals. This cytokine milieu not only facilitates immune activation but also contributes to the dysregulation of immune cell functions. For instance, increased levels of IL-6 are linked to the development of B-cell hyperactivation, leading to abnormal antibody production and immune dysregulation. HIV infection induces significant changes in T-cell dynamics, leading to an imbalance between effector and regulatory T-cells. The chronic immune activation can cause a depletion of regulatory T-cells (Tregs), which normally function to maintain immune homeostasis and prevent excessive inflammation. The reduced numbers of Tregs contribute to an unchecked immune response, further perpetuating immune activation and increasing susceptibility to autoimmune conditions and co-infections.[16-20] Prolonged exposure to HIV antigens and chronic immune activation can lead to T-cell exhaustion, a state characterized by the expression of inhibitory receptors such as PD-1, CTLA-4, and Tim-3. Exhausted T-cells exhibit reduced proliferative capacity, diminished cytokine production, and impaired ability to control viral replication. This state of exhaustion not only hinders the effectiveness of the immune response against HIV but also contributes to the overall state of immune dysregulation and inflammation. Emerging research highlights the role of the gut microbiome in influencing immune responses in HIV-infected individuals. The integrity of the gut barrier is often compromised in HIV infection, leading to increased translocation of microbial products into the bloodstream. This translocation can trigger systemic inflammation and further drive immune activation. The gut microbiome's composition can also impact immune responses, with dysbiosis potentially exacerbating chronic inflammation. Co-infections with other pathogens, such as tuberculosis, hepatitis viruses, or respiratory viruses, can amplify HIV-induced immune activation. The presence of these co-infections can exacerbate the inflammatory response, leading to higher levels of immune activation and further complicating the management of HIV. The interplay between HIV and co-infecting pathogens underscores the importance of comprehensive clinical management to address the multifaceted nature of immune activation.[21-25] Genetic predisposition may also play a role in influencing the degree of immune activation in individuals living with HIV. Variations in genes involved in immune regulation and response can affect how an individual's immune system reacts to HIV infection.

Consequences of Immune Activation

HIV-induced immune activation has profound consequences for the health and well-being of infected individuals, particularly in pediatric populations. The persistent state of immune activation can lead to a variety of short-term and long-term effects, impacting both the immune system's functionality and overall health outcomes. Chronic immune activation is associated with an increased risk of opportunistic infections, which are infections that take advantage of a weakened immune system. In HIV-infected children, immune dysregulation can compromise the body's ability to effectively respond to pathogens, leading to higher rates of infections such as pneumonia, tuberculosis, and candidiasis. The combination of reduced CD4+ T-cell counts and heightened immune activation creates an environment where the immune system is less capable of mounting adequate responses to combat opportunistic pathogens. Sustained immune activation can lead to the development of autoimmune disorders, where the immune system mistakenly targets the body's own tissues. In HIV-infected individuals, the chronic inflammatory state can

contribute to the dysregulation of immune tolerance mechanisms, increasing the risk of conditions such as autoimmune hemolytic anemia, systemic lupus erythematosus, and rheumatoid arthritis. The interplay between HIV infection, immune activation, and autoimmune responses necessitates careful monitoring and management of potential autoimmune complications.[26-29]

Chronic immune activation in HIV-infected individuals is linked to the development of non-infectious comorbidities, including cardiovascular disease, metabolic syndrome, and neurocognitive disorders. The persistent inflammatory state can contribute to endothelial dysfunction, increasing the risk of cardiovascular complications. Additionally, immune activation has been associated with insulin resistance and obesity, leading to metabolic syndrome. The implications of these comorbidities can significantly affect the long-term health of pediatric patients living with HIV. Effective antiretroviral therapy (ART) aims to suppress viral replication and promote immune reconstitution. However, chronic immune activation can hinder the ability of the immune system to recover fully. In HIV-infected children, the presence of persistent immune activation can result in incomplete restoration of CD4+ T-cell counts, leaving them more vulnerable to infections and other complications. This incomplete immune reconstitution emphasizes the need for ongoing monitoring and tailored interventions to address residual immune dysregulation. The effects of HIV-induced immune activation extend beyond physical health, influencing the psychosocial well-being of affected children and their families. The burden of living with a chronic infection and its associated complications can lead to anxiety, depression, and social stigma. Children may face challenges in school, peer relationships, and family dynamics due to their health status, which can further exacerbate mental health issues. Addressing these psychosocial factors is crucial for promoting holistic health and improving the quality of life for HIV-infected children.[30-32]

Immune activation can impact the effectiveness of vaccinations in HIV-infected individuals. Chronic inflammation and immune dysregulation may lead to suboptimal responses to vaccines, putting children at risk for vaccine-preventable diseases. This underscores the importance of ensuring that HIV-infected children receive appropriate vaccinations and monitoring their immune responses to vaccination to optimize protection against infectious diseases. Chronic immune activation in HIV-infected individuals is associated with accelerated immune aging, a phenomenon characterized by the decline in immune function typically observed in older adults. This accelerated aging can result in diminished responsiveness to new infections and vaccines, increasing vulnerability to both infectious and non-infectious diseases. In pediatric populations, this can have long-term implications for health and longevity, necessitating careful monitoring and management of immune health over time. In children, chronic immune activation can adversely affect growth and development. The metabolic demands of an activated immune system can interfere with nutritional status, leading to growth delays and malnutrition. Additionally, immune dysregulation may impact cognitive development and learning, contributing to challenges in educational settings. Addressing these growth and developmental concerns is essential for ensuring that HIV-infected children achieve their full potential. Chronic immune activation is increasingly recognized as a factor that may contribute to mental health issues in HIV-infected individuals. Inflammation has been implicated in the development of mood disorders and cognitive impairment, potentially affecting the psychological well-being of pediatric patients.[33-37]

The Role of Antiretroviral Therapy (ART)

Antiretroviral therapy (ART) has revolutionized the management of HIV infection, transforming it from a life-threatening condition to a manageable chronic disease. In pediatric populations, ART plays a critical role in reducing viral loads, improving immune function, and mitigating the consequences of HIV-induced immune activation. The primary goal of ART is to suppress HIV replication, thereby reducing the viral load in the body. By inhibiting various stages of the viral life cycle—such as entry into host cells, reverse transcription, integration, and viral assembly—ART effectively lowers the amount of circulating virus. This reduction in viral load leads to decreased antigenic stimulation of the immune system, which is crucial for alleviating the chronic immune activation associated with HIV infection. Lower levels of viral antigens reduce the overall inflammatory burden on the immune system, contributing to improved immune health. ART significantly decreases the levels of pro-inflammatory cytokines and markers of immune activation in HIV-infected individuals. Studies have shown that effective viral suppression through ART leads to reductions in markers such as CD38 and HLA-DR on T-cells, which are indicative of immune activation. By mitigating immune activation, ART helps restore a more balanced immune response, reducing the risk of opportunistic infections and other complications associated with chronic inflammation. One of the most significant benefits of ART is its ability to promote immune reconstitution, particularly the recovery of CD4+ T-cells. In children, the early initiation of ART has been shown to facilitate rapid recovery of CD4+ T-cell counts, which is essential for restoring immune function. Improved immune reconstitution enhances the body's ability to respond to infections and reduces the likelihood of developing non-infectious comorbidities associated with immune dysregulation.[38-41]

Initiating ART as early as possible in pediatric patients has been associated with long-term benefits, including better immune recovery and reduced rates of morbidity and mortality. Early ART can minimize the extent of immune activation and prevent the detrimental effects of chronic inflammation on growth and development. Additionally, children who start ART early are more likely to achieve and maintain viral suppression, leading to improved overall health outcomes. While ART has demonstrated significant benefits, adherence to treatment remains a challenge, particularly in pediatric populations. Factors such as complex dosing regimens, the need for lifelong treatment, and potential side effects can affect adherence rates. Additionally, psychosocial factors, including stigma, family dynamics, and access to healthcare, can further complicate treatment adherence in children. Improving adherence is essential for maximizing the effectiveness of ART and minimizing the risk of treatment failure and drug resistance. Regular monitoring of immune function and viral load is crucial for assessing the effectiveness of ART in pediatric populations. Healthcare providers must evaluate CD4+ T-cell counts and HIV viral loads to determine the appropriate ART regimen and make necessary adjustments. In some cases, children may develop drug resistance, requiring a switch to alternative medications. Personalized treatment approaches that consider the unique needs of each child are essential for optimizing ART effectiveness and managing immune activation. ART not only targets HIV but also has implications for the management of co-infections that commonly affect HIV-infected children. Effective viral suppression can improve immune responses to co-infecting pathogens, reducing the risk of opportunistic infections. Moreover, addressing co-infections through appropriate treatment can further alleviate immune activation and inflammation, contributing to better overall health outcomes.[42-46]

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The initiation of ART can have positive effects on the psychosocial well-being of children living with HIV. Achieving viral suppression can reduce anxiety and stigma associated with HIV, allowing children to lead more normal lives. Improved health and well-being can enhance the quality of life for both the child and their family, fostering a supportive environment for optimal development and social integration. While ART is effective in managing HIV infection, there are concerns regarding potential long-term effects on immune function and overall health. Some studies suggest that certain ART regimens may be associated with metabolic complications, cardiovascular risks, or effects on bone health. Monitoring for these potential complications is important to ensure that children receiving ART maintain optimal health as they grow older.[47]

Strategies for Mitigating Immune Activation

Mitigating HIV-induced immune activation is essential for improving the health and quality of life of pediatric populations living with HIV. Chronic immune activation is linked to various adverse health outcomes, including increased susceptibility to infections, development of non-infectious comorbidities, and impaired immune function. This section outlines several strategies aimed at reducing immune activation in HIV-infected children, including early initiation of antiretroviral therapy (ART), management of co-infections, nutritional interventions, vaccination strategies, and psychosocial support. Starting ART as early as possible in HIV-infected children is one of the most effective strategies for reducing immune activation. Early treatment can minimize the duration and extent of viral replication, leading to decreased antigenic stimulation and inflammation. Studies have shown that children who begin ART early experience better immune recovery, lower levels of immune activation markers, and improved overall health outcomes. This approach emphasizes the importance of early diagnosis and prompt initiation of treatment. Ongoing monitoring of immune function and viral load is essential for assessing the effectiveness of ART and managing immune activation. Healthcare providers should routinely evaluate CD4+ T-cell counts and inflammatory markers to tailor treatment plans for individual patients. Personalized treatment approaches that account for the unique needs of each child can help optimize ART regimens, enhance adherence, and reduce immune activation. This may include switching to alternative ART regimens that are more effective in suppressing viral replication or reducing inflammation.[48-49]

Co-infections are common in HIV-infected children and can exacerbate immune activation. Proactively managing co-infections, such as tuberculosis, hepatitis viruses, and other opportunistic pathogens, is crucial for reducing inflammation and promoting immune health. Effective treatment of co-infections can improve overall immune function and decrease the inflammatory burden on the body. Comprehensive care that includes screening and appropriate treatment for co-infections is essential for optimizing health outcomes. Nutrition plays a vital role in supporting immune function and reducing inflammation. Ensuring that HIV-infected children receive adequate nutrition can help bolster their immune systems and mitigate the effects of immune activation. Nutritional interventions may include providing a balanced diet rich in essential vitamins and minerals, addressing micronutrient deficiencies, and promoting healthy eating habits. Collaborating with nutritionists and healthcare providers to create tailored dietary plans can

enhance immune health and overall well-being. Vaccination is a critical component of preventing infections in HIV-infected children. However, immune activation can impact vaccine responses. Strategies to enhance vaccine efficacy in this population may include administering vaccinations at optimal times when immune function is better, using adjuvants to boost immune responses, and providing catch-up vaccination schedules for those who may have missed immunizations. Ensuring that HIV-infected children receive timely vaccinations against preventable diseases can significantly reduce the risk of infections and associated immune activation. In addition to ART, adjunctive therapies that target inflammation and immune activation may be beneficial. Research into the use of anti-inflammatory agents, such as statins or corticosteroids, is ongoing, with some studies suggesting that these treatments may help reduce markers of immune activation in HIV-infected individuals. While more research is needed to establish the safety and efficacy of these adjunctive therapies in pediatric populations, they may represent a promising avenue for mitigating chronic inflammation.[50]

The psychosocial aspects of living with HIV can significantly impact immune health. Providing psychosocial support to children and their families is essential for addressing the emotional and social challenges associated with HIV. Mental health interventions, counseling, and support groups can help alleviate stress and anxiety, which are known contributors to immune dysregulation. A supportive environment that fosters emotional well-being can enhance adherence to treatment and improve overall health outcomes. Educating children and their families about HIV, its management, and the importance of adherence to ART is vital for promoting long-term health. Empowering families with knowledge about immune activation and its consequences can encourage proactive engagement in care. Providing resources, workshops, and support networks can help families navigate the challenges of living with HIV and facilitate better health outcomes for children.[51-55]

Conclusion

HIV-induced immune activation presents significant challenges for pediatric populations, impacting their immune health and overall well-being. The persistent state of immune activation is associated with increased susceptibility to infections, the development of non-infectious comorbidities, and psychosocial difficulties. However, through a combination of effective strategies, including early initiation of antiretroviral therapy (ART), regular monitoring of immune function, management of co-infections, nutritional support, and vaccination, healthcare providers can significantly mitigate the adverse effects of immune activation in HIV-infected children. Early ART initiation is paramount, as it not only suppresses viral replication but also reduces chronic inflammation and promotes immune reconstitution. Personalized treatment approaches tailored to the unique needs of each child, alongside effective management of co-infections, play crucial roles in enhancing immune responses and preventing complications. Additionally, addressing the nutritional needs of HIV-infected children and ensuring timely vaccinations against preventable diseases can further bolster their immune health. Psychosocial support and education are equally important in promoting adherence to treatment and fostering a supportive environment for children and their families. Empowering families with knowledge about HIV and its management, as well

as providing mental health resources, can help alleviate stress and improve the quality of life for those affected.

References

1. Obeagu EI, Anyiam AF, Obeagu GU. Managing Anemia in HIV through Blood Transfusions: Clinical Considerations and Innovations. Elite Journal of HIV, 2024; 2(1): 16-30
2. Obeagu EI, Obeagu, GU. Counting Cells, Shaping Fates: CD4/CD8 Ratios in HIV. EliteJournal of Scientific Research and Review, 2024; 2(1): 37-50
3. Obeagu EI, Obeagu GU. Hematological Changes Following Blood Transfusion in Young Children with Severe Malaria and HIV: A Critical Review. Elite Journal of Laboratory Medicine, 2024; 2(1): 33-45
4. Obeagu EI, Obeagu GU. The Role of Blood Transfusion Strategies in HIV Management: Current Insights and Future Directions. Elite Journal of Medicine, 2024; 2(1):10-22
5. Lewis DB, Weitkamp JH, Levy O. Developmental immunology and role of host defenses in fetal and neonatal susceptibility to infection. In Remington and Klein's Infectious Diseases of the Fetus and Newborn Infant 2025: 73-159. Elsevier.
6. Netea MG. Training innate immunity: the changing concept of immunological memory in innate host defence. European journal of clinical investigation. 2013;43(8):881-884.
7. Obeagu EI, Obeagu GU. Eosinophil Dynamics in Pregnancy among Women Living with HIV: A Comprehensive Review. Int. J. Curr. Res. Med. Sci. 2024;10(1):11-24.
8. Viola N, Kimono E, Nuruh N, Obeagu EI. Factors Hindering Elimination of Mother to Child Transmission of HIV Service Uptake among HIV Positive Women at Comboni Hospital Kyamuhunga Bushenyi District. Asian Journal of Dental and Health Sciences. 2023;3(2):7-14.
9. Obeagu EI, Obeagu GU. Transfusion-Related Complications in Children Under 5 with Coexisting HIV and Severe Malaria: A Review. Int. J. Curr. Res. Chem. Pharm. Sci. 2024;11(2):9-19.
10. Obeagu EI, Obeagu GU, Hauwa BA, Umar AI. Neutrophil Dynamics: Unveiling Their Role in HIV Progression within Malaria Patients. Journal home page: http://www. journalijiar. com.;12(01).
11. Obeagu EI, Obeagu, GU. P-Selectin and Platelet Activation in HIV: Implications for Antiviral Therapy. Elite Journal of Scientific Research and Review, 2024; 2(1): 17-41
12. Obeagu EI, Obeagu GU. The Intricate Relationship Between Erythropoietin and HIV-Induced Anemia: Unraveling Pathways for Therapeutic Insights. Int. J. Curr. Res. Chem. Pharm. Sci. 2024;11(2):30-40.
13. Arikawa S, Rollins N, Newell ML, Becquet R. Mortality risk and associated factors in HIV-exposed, uninfected children. Tropical Medicine & International Health. 2016;21(6):720-734.
14. Brennan AT, Bonawitz R, Gill CJ, Thea DM, Kleinman M, Useem J, Garrison L, Ceccarelli R, Udokwu C, Long L, Fox MP. A meta-analysis assessing all-cause mortality in HIV-exposed uninfected compared with HIV-unexposed uninfected infants and children. Aids. 2016;30(15):2351-2360.

Citation: Obeagu EI. HIV-Induced Immune Activation in Pediatric Populations. Elite Journal of Immunology, 2024; 2(7): 28-38

15. Obeagu EI, Anyiam AF, Obeagu GU. Erythropoietin Therapy in HIV-Infected Individuals: A Critical Review. Elite Journal of HIV, 2024; 2(1): 51-64
16. Obeagu EI, Obeagu GU. Strength in Unity: Building Support Networks for HIV Patients in Uganda . Elite Journal of Medicine, 2024; 2(1): 1-16
17. Obeagu EI, Obeagu GU. Eosinophilic Changes in Placental Tissues of HIV-Positive Pregnant Women: A Review. Elite Journal of Laboratory Medicine, 2024; 2(1): 14-32
18. Obeagu EI, Obeagu, GU. The Crucial Role of Erythropoietin in Managing Anemia in HIV: A Review. Elite Journal of Scientific Research and Review, 2024; 2(1): 24-36
19. Obeagu EI, Ubosi NI, Obeagu GU, Obeagu AA. Nutritional Strategies for Enhancing Immune Resilience in HIV: A Review. Int. J. Curr. Res. Chem. Pharm. Sci. 2024;11(2):41-51.
20. Obeagu EI, Nweke JO. Neonatal Immune Development in the Context of HIV Infection: A Review. Elite Journal of Immunology. 2024;2(5):29-38.
21. Obeagu EI. Immune Dysregulation in HIV-Positive Neonates: A Review. Elite Journal of Laboratory Medicine. 2024;2(6):49-66.
22. Obeagu EI, Obeagu GU. Maternal Influence on Infant Immunological Responses to HIV: A Review. Elite Journal of Laboratory Medicine. 2024;2(1):46-58.
23. Obeagu EI, Obeagu GU. An update on Early Immunological Markers in HIV-Exposed Infants. Elite Journal of Immunology. 2024;2(6):15-25.
24. Kampmann B, Jones CE. Factors influencing innate immunity and vaccine responses in infancy. Philosophical Transactions of the Royal Society B: Biological Sciences. 2015 Jun 19;370(1671):20140148.
25. Obeagu EI. HIV-Specific T-Cell Responses in Infants: A Review. Elite Journal of Medical Sciences. 2024;2(6):10-23.
26. Diamond MS, Kanneganti TD. Innate immunity: the first line of defense against SARS-CoV-2. Nature immunology. 2022;23(2):165-176.
27. Amarante-Mendes GP, Adjemian S, Branco LM, Zanetti LC, Weinlich R, Bortoluci KR. Pattern recognition receptors and the host cell death molecular machinery. Frontiers in immunology. 2018; 9:2379.
28. Andoniou CE, Andrews DM, Degli-Esposti MA. Natural killer cells in viral infection: more than just killers. Immunological reviews. 2006;214(1):239-250.
29. Mayer LS, Uciechowski P, Meyer S, Schwerdtle T, Rink L, Haase H. Differential impact of zinc deficiency on phagocytosis, oxidative burst, and production of pro-inflammatory cytokines by human monocytes. Metallomics. 2014;6(7):1288-1295.
30. Basha S, Surendran N, Pichichero M. Immune responses in neonates. Expert review of clinical immunology. 2014;10(9):1171-1184.
31. Maródi L. Neonatal innate immunity to infectious agents. Infection and immunity. 2006;74(4):1999-2006.
32. Obeagu EI, Elamin EAI Obeagu GU. Understanding the Intersection of Highly Active Antiretroviral Therapy and Platelets in HIV Patients: A Review. Elite Journal of Haematology, 2024; 2(3): 111-117

Citation: Obeagu EI. HIV-Induced Immune Activation in Pediatric Populations. Elite Journal of Immunology, 2024; 2(7): 28-38

33. Obeagu EI, Obeagu GU. Neonatal Outcomes in Children Born to Mothers with Severe Malaria, HIV, and Transfusion History: A Review. Elite Journal of Nursing and Health Science, 2024; 2(3): 38-58
34. Obeagu EI. Erythropoietin and the Immune System: Relevance in HIV Management. Elite Journal of Health Science, 2024; 2(3): 23-35
35. Obeagu EI, Obeagu GU. Understanding Immune Cell Trafficking in Tuberculosis-HIV Coinfection: The Role of L-selectin Pathways. Elite Journal of Immunology, 2024; 2(2): 43-59
36. Obeagu EI, Obeagu GU. Anemia and Erythropoietin: Key Players in HIV Disease Progression. Elite Journal of Haematology, 2024; 2(3): 42-57
37. Obeagu EI, Ayogu EE, Obeagu GU. Impact on Viral Load Dynamics: Understanding the Interplay between Blood Transfusion and Antiretroviral Therapy in HIV Management. Elite Journal of Nursing and Health Science, 2024; 2(2): 5-15
38. Obeagu EI, Obeagu GU. Immune Modulation in HIV-Positive Neonates: Insights and Implications for Clinical Management. Elite Journal of Nursing and Health Science, 2024; 2(3): 59-72
39. Blanco JR, Negredo E, Bernal E, Blanco J. Impact of HIV infection on aging and immune status. Expert Review of Anti-infective Therapy. 2021;19(6):719-731.
40. Olbrich L, Stockdale L, Basu Roy R, Song R, Cicin-Sain L, Whittaker E, Prendergast AJ, Fletcher H, Seddon JA. Understanding the interaction between cytomegalovirus and tuberculosis in children: the way forward. PLoS Pathogens. 2021;17(12): e1010061.
41. Fok ET, Davignon L, Fanucchi S, Mhlanga MM. The lncRNA connection between cellular metabolism and epigenetics in trained immunity. Frontiers in Immunology. 2019; 9:3184.
42. Cuenca AG, Wynn JL, Moldawer LL, Levy O. Role of innate immunity in neonatal infection. American journal of perinatology. 2013;30(02):105-112.
43. Obeagu EI, Obeagu GU. Maternal Influence on Infant Immunological Responses to HIV: A Review. Elite Journal of Laboratory Medicine. 2024;2(1):46-58.
44. Obeagu EI, Obeagu GU. Impact of Maternal Eosinophils on Neonatal Immunity in HIV-Exposed Infants: A Review. Elite Journal of Immunology. 2024;2(3):1-8.
45. Lowenthal ED, Bakeera-Kitaka S, Marukutira T, Chapman J, Goldrath K, Ferrand RA. Perinatally acquired HIV infection in adolescents from sub-Saharan Africa: a review of emerging challenges. The Lancet infectious diseases. 2014;14(7):627-639.
46. Obeagu EI, Chukwu PH. HIV and Natural Killer (NK) Cell Responses in Neonates: A Review. Elite Journal of Immunology. 2024;2(5):39-49.
47. Ruck C, Reikie BA, Marchant A, Kollmann TR, Kakkar F. Linking susceptibility to infectious diseases to immune system abnormalities among HIV-exposed uninfected infants. Frontiers in immunology. 2016; 7:310.
48. Langel SN, Blasi M, Permar SR. Maternal immune protection against infectious diseases. Cell Host & Microbe. 2022 May 11;30(5):660-674.
49. Obeagu EI. Markers of Immune Activation in HIV-Exposed Infants. Elite Journal of Health Science. 2024;2(6):1-4.

Citation: Obeagu EI. HIV-Induced Immune Activation in Pediatric Populations. Elite Journal of Immunology, 2024; 2(7): 28-38

50. Obeagu EI, Obeagu GU. Maternal Eosinophilic Responses in HIV-Positive Pregnant Women: Unraveling Immunological Dynamics for Improved Maternal-Fetal Health. Elite Journal of Immunology. 2024;2(1):47-64.
51. Obeagu EI, Obeagu GU. Impact of Breastfeeding on Infant Immune Responses in the Context of HIV. Elite Journal of Nursing and Health Science. 2024;2(4):23-39.
52. Obeagu EI. HIV and Innate Immune Memory in Neonates. Elite Journal of Immunology, 2024; 2(6): 44-52
53. Obeagu EI. HIV and T-Cell Exhaustion in Pediatric Populations. Elite Journal of Immunology, 2024; 2(6): 53-62
54. Obeagu EI. Immunological Memory Development in HIV-Exposed Children. Elite Journal of Immunology, 2024; 2(7): 1-14
55. Obeagu EI. Adaptive Immune Responses in HIV-Infected Infants. Elite Journal of Immunology, 2024; 2(7): 15-27