Elite Journal of Laboratory Medicine. Volume 2 Issue 6(2024), Pp. 33-48
https://epjournals.com/journals/EJLM

Cytokine Responses in HIV-Infected Pediatric Patients: A Review

Emmanuel Ifeanyi Obeagu

Department of Medical Laboratory Science, Kampala International University, Uganda, emmanuelobeagu@yahoo.com

Abstract

Cytokine responses are central to the immunopathogenesis of HIV infection, particularly in pediatric patients whose immune systems are still developing. This review examines the unique cytokine profiles observed in HIV-infected children, highlighting how elevated levels of pro-inflammatory cytokines such as IL-6, TNF-α, and IL-1β contribute to chronic immune activation and inflammation. These responses differ significantly from those in adults, with younger children displaying a more pronounced Th2 response and a shift towards a Th1 response as the disease progresses. Such variations underline the importance of age and disease stage in understanding pediatric HIV pathogenesis. The chronic immune activation driven by cytokine responses in HIV-infected pediatric patients leads to immune cell exhaustion and apoptosis, resulting in the depletion of CD4+ T cells and systemic inflammation. This inflammation is linked to various comorbidities, including cardiovascular and neurocognitive disorders, as well as developmental delays in children. Antiretroviral therapy (ART) effectively reduces viral load and pro-inflammatory cytokine levels, though some cytokines, like IL-6, may remain elevated, indicating persistent immune activation. Monitoring cytokine levels is crucial for assessing treatment efficacy and identifying potential ART resistance. Emerging therapeutic strategies aim to modulate cytokine responses to mitigate immune activation and inflammation. These approaches include the use of cytokine inhibitors, anti-inflammatory agents, and immune modulators.

Keywords: *HIV**,** Pediatric**,** Cytokine**,** Immune Response**,** Immunopathogenesis**,** Antiretroviral Therapy (ART)**,** Inflammation*

Introduction

Human Immunodeficiency Virus (HIV) remains one of the most significant global health challenges, particularly affecting vulnerable populations, including children. Pediatric HIV infection, predominantly transmitted vertically from mother to child, accounts for a substantial proportion of the global HIV burden. The immune system of children, still undergoing

development, exhibits distinct responses to HIV infection compared to adults, necessitating a comprehensive understanding of these differences to optimize therapeutic strategies.[1-5] Cytokines, small signaling proteins secreted by immune cells, play a pivotal role in orchestrating the body's immune response. They are involved in various functions, including cell communication, inflammation regulation, and immune cell recruitment. In the context of HIV infection, cytokine responses are critical in determining disease progression, immune system activation, and the overall pathogenesis of the infection. The study of cytokine responses in HIV-infected pediatric patients reveals unique patterns that differ significantly from those observed in adults.[6-10] The baseline cytokine levels in HIV-infected children are often elevated, reflecting a state of chronic immune activation. This heightened immune response, characterized by increased levels of pro-inflammatory cytokines such as IL-6, TNF-α, and IL-1β, plays a central role in the disease's progression. These cytokines are markers of inflammation and immune activation, which are central features of HIV pathogenesis. Understanding the baseline cytokine profiles in pediatric patients provides crucial insights into how the immune system initially responds to HIV infection.[11-15] The age of the child and the stage of HIV infection significantly influence cytokine responses. Younger children tend to exhibit a more pronounced Th2 cytokine profile, marked by higher levels of IL-10 and IL-12. This response reflects the immune system's attempt to regulate inflammation and prevent tissue damage. However, as the disease progresses, there is a shift towards a Th1 cytokine profile, with increased levels of IFN-γ and IL-17, indicating a more inflammatory response. This shift is associated with the deterioration of the immune system and progression towards AIDS.[16-20]

Chronic immune activation, driven by persistent HIV replication and cytokine dysregulation, is a hallmark of HIV pathogenesis. This continuous state of activation leads to the exhaustion and apoptosis of immune cells, particularly CD4+ T cells, which are crucial for maintaining immune function. The depletion of these cells results in the weakening of the immune system, making the body more susceptible to opportunistic infections and diseases. This cycle of activation and cell death underscores the importance of understanding and managing cytokine responses in HIV-infected pediatric patients.[21-25] Prolonged elevation of pro-inflammatory cytokines contributes to systemic inflammation, which can have far-reaching effects beyond the immune system. In pediatric HIV patients, this inflammation can lead to various comorbidities, including cardiovascular diseases, neurocognitive disorders, and developmental delays. The impact of systemic inflammation on growth and development in children underscores the need for early intervention and effective management of cytokine responses to mitigate these adverse outcomes.[26-30] Antiretroviral therapy (ART) has transformed the prognosis of HIV infection, significantly reduced viral load and improved immune function. ART's impact on cytokine profiles in pediatric patients is profound, as it reduces the levels of pro-inflammatory cytokines and mitigates immune activation. However, despite effective viral suppression, some cytokines, such as IL-6 and D-dimer, may remain elevated, indicating residual immune activation. Understanding the nuances of ART's impact on cytokine responses is essential for optimizing treatment regimens and improving long-term outcomes for pediatric patients.[31-35]

In cases where ART fails or resistance develops, there is often a resurgence of viremia and immune activation. This resurgence is accompanied by increased levels of pro-inflammatory cytokines and

a further deterioration of immune function. Monitoring cytokine levels in ART-resistant pediatric patients provides valuable insights into their immune system status and can guide therapeutic adjustments. It highlights the need for vigilant monitoring and potential alternative therapeutic strategies to manage these challenging cases.[36-40] Emerging therapeutic strategies aim to modulate cytokine responses to reduce immune activation and inflammation in HIV-infected pediatric patients. These strategies include the use of cytokine inhibitors, anti-inflammatory agents, and immune modulators. Research into the long-term effects of these therapies on pediatric patients is ongoing, with the potential to significantly improve outcomes by reducing chronic inflammation and immune activation. The development of such targeted therapies represents a promising avenue for enhancing the quality of life for HIV-infected children.[41-45] Cytokines also hold potential as biomarkers for monitoring disease progression and treatment efficacy in pediatric HIV patients. Regular monitoring of cytokine profiles can aid in the early identification of treatment failure or disease progression, enabling timely and targeted interventions. The use of cytokines as biomarkers can improve personalized treatment approaches and optimize therapeutic outcomes for pediatric patients.[46-50]

Cytokine Profiles in Pediatric HIV Infection

In HIV-infected pediatric patients, baseline cytokine levels are often significantly elevated compared to their uninfected counterparts. These elevated levels reflect a state of chronic immune activation, a hallmark of HIV infection. Key pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β) are typically found at higher concentrations in the plasma of HIV-infected children. These cytokines are critical mediators of inflammation and play a significant role in the immune system's response to infection.[51-55] The cytokine profiles in HIV-infected pediatric patients are influenced by the child's age and the stage of the disease. Younger children tend to exhibit higher levels of Th2 cytokines, including interleukin-10 (IL-10) and interleukin-12 (IL-12). IL-10 is an anti-inflammatory cytokine that helps regulate immune responses, while IL-12 promotes the development of Th1 cells, which are essential for fighting viral infections. As HIV infection progresses, there is often a shift towards a Th1 cytokine profile characterized by increased levels of interferon-gamma (IFN-γ) and interleukin-17 (IL-17). This shift is associated with a more inflammatory response and correlates with the advancement of the disease and immune system deterioration.[56-60] Chronic immune activation is a significant driver of HIV pathogenesis in pediatric patients. Elevated cytokine levels contribute to the persistent activation of immune cells, leading to their exhaustion and apoptosis. This continuous cycle of immune activation and cell death depletes critical immune cells, particularly CD4+ T cells, which are vital for maintaining immune function. The depletion of CD4+ T cells result in a weakened immune system, making the body more susceptible to opportunistic infections and other diseases. Understanding the mechanisms of immune activation and its effects on cytokine profiles is crucial for developing effective therapeutic strategies.[61-65] Prolonged elevation of pro-inflammatory cytokines leads to systemic inflammation, which can cause significant damage to various organs. In pediatric HIV patients, chronic inflammation is associated with the development of comorbidities such as cardiovascular diseases, neurocognitive

disorders, and growth and developmental delays. The impact of systemic inflammation on the overall health and development of HIV-infected children highlights the need for early and effective management of cytokine responses. Reducing chronic inflammation through targeted therapies can help mitigate these adverse outcomes and improve the quality of life for these patients.[66-67]

Antiretroviral Therapy (ART) and Cytokine Modulation

Antiretroviral therapy (ART) has dramatically improved the prognosis of HIV infection by reducing viral load and improving immune function. ART significantly impacts cytokine profiles in pediatric patients by lowering the levels of pro-inflammatory cytokines and reducing immune activation. However, despite effective viral suppression, some cytokines, such as IL-6 and D-dimer, may remain elevated, indicating persistent immune activation. Understanding the nuances of ART's impact on cytokine responses is essential for optimizing treatment regimens and ensuring the best possible outcomes for pediatric patients.[68-70] In cases of ART resistance or failure, there is often a resurgence of viremia and immune activation. This resurgence is accompanied by increased levels of pro-inflammatory cytokines and a further decline in immune function. Monitoring cytokine levels in ART-resistant pediatric patients provides valuable insights into their immune system status and can guide therapeutic adjustments. Identifying and addressing ART resistance promptly is critical to maintaining effective viral control and preventing disease progression.[71-72] Cytokines serve as valuable biomarkers for monitoring disease progression and treatment efficacy in pediatric HIV patients. Regular monitoring of cytokine profiles can aid in the early identification of treatment failure or disease progression, enabling timely and targeted interventions. The use of cytokines as biomarkers can improve personalized treatment approaches and optimize therapeutic outcomes for pediatric patients. By tracking changes in cytokine levels, healthcare providers can better understand the patient's immune status and make more informed decisions about their care.[73-75] Emerging therapeutic strategies aim to modulate cytokine responses to reduce immune activation and inflammation in HIV-infected pediatric patients. These strategies include the use of cytokine inhibitors, anti-inflammatory agents, and immune modulators. Research into the long-term effects of these therapies on pediatric patients is ongoing and holds promise for improving outcomes. By targeting specific cytokines and pathways involved in chronic inflammation and immune activation, these therapies have the potential to enhance the quality of life for HIV-infected children and reduce the burden of comorbidities associated with the infection.[76-77]

Cytokine-Mediated Immune Activation and Pathogenesis

Chronic immune activation is a cornerstone of HIV pathogenesis and is particularly pronounced in pediatric patients. HIV infection triggers the immune system to produce a wide array of cytokines, which are signaling molecules that mediate and regulate immunity, inflammation, and hematopoiesis. Among the critical cytokines involved are interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β). These cytokines contribute to a persistent state of immune activation by continuously stimulating immune cells. This ongoing activation leads to the exhaustion and eventual apoptosis of these cells, particularly CD4+ T cells, which are crucial for immune function. The depletion of CD4+ T cells is a hallmark of HIV progression and a direct consequence of chronic immune activation.[78-80] The mechanisms underlying immune

activation in HIV-infected pediatric patients are multifaceted. One significant factor is the direct effect of the HIV virus on immune cells. HIV primarily targets CD4+ T cells, integrating its genetic material into the host cell's DNA and hijacking the cell's machinery to produce more virus particles. This viral replication cycle induces the production of pro-inflammatory cytokines, further stimulating immune activation.[81] Another contributing factor is microbial translocation, which occurs when the integrity of the gastrointestinal mucosal barrier is compromised. This allows microbial products from the gut lumen, such as lipopolysaccharides (LPS), to enter the systemic circulation. These microbial products act as potent stimulators of the immune system, driving the production of pro-inflammatory cytokines and perpetuating immune activation. Pediatric patients, with their still-developing immune systems and potentially less robust mucosal barriers, may be particularly susceptible to microbial translocation and its effects.[82]

The consequences of chronic immune activation are profound and far-reaching. One of the most direct outcomes is the accelerated loss of CD4+ T cells. These cells are not only directly targeted and destroyed by the virus but also suffer from bystander apoptosis due to the inflammatory milieu created by constant cytokine signaling. The depletion of CD4+ T cells lead to a compromised immune system, which in turn heightens the risk of opportunistic infections and other diseases.[83] Additionally, chronic immune activation and the associated elevated levels of cytokines such as IL-6 and TNF-α can cause systemic inflammation. This systemic inflammation is a significant contributor to the development of non-AIDS-related comorbidities. For instance, high levels of TNF-α have been linked to cardiovascular diseases, as this cytokine can promote the formation of atherosclerotic plaques and vascular inflammation. Similarly, IL-6 is associated with a variety of inflammatory conditions and has been implicated in the development of neurocognitive disorders.[84] In pediatric patients, the systemic inflammation resulting from chronic immune activation can also impact growth and development. Inflammation can interfere with normal metabolic processes and nutrient absorption, leading to growth delays and failure to thrive. Moreover, neuroinflammation, driven by cytokines such as IL-1β and IL-6, can adversely affect brain development and cognitive function. Children with HIV are at an increased risk for neurocognitive impairments, which can manifest as learning difficulties, behavioral issues, and delayed developmental milestones.[85]

Antiretroviral therapy (ART) has been highly effective in reducing HIV viral load and improving immune function. However, ART's impact on cytokine-mediated immune activation is complex. While ART reduces the levels of some pro-inflammatory cytokines and helps reconstitute the immune system, certain cytokines like IL-6 and D-dimer may remain elevated even in patients with well-controlled HIV. This residual immune activation can continue to drive systemic inflammation and contribute to the development of comorbidities.[86] When ART fails or resistance develops, there is typically a resurgence of viremia and immune activation. This is accompanied by an increase in pro-inflammatory cytokines and a further decline in immune function. Monitoring cytokine levels in ART-resistant pediatric patients is crucial for understanding their immune status and guiding treatment decisions. High levels of cytokines can indicate ongoing viral replication and immune system stress, necessitating adjustments in therapy or the introduction of new treatment strategies.[87]

Therapeutic Approaches to Modulate Cytokine Responses

Given the significant role of cytokines in immune activation and pathogenesis, modulating these responses presents a promising therapeutic avenue. Various strategies are being explored to reduce immune activation and inflammation in HIV-infected pediatric patients. These include the use of cytokine inhibitors, which can specifically block the action of pro-inflammatory cytokines like IL-6 and TNF-α. Anti-inflammatory agents, such as corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs), can also help mitigate the effects of systemic inflammation. Additionally, immune modulators that enhance regulatory T cell function or block pathways involved in immune activation are under investigation.[88] The long-term implications of cytokine-mediated immune activation in pediatric HIV patients are significant. Chronic inflammation and immune activation not only affect immediate health outcomes but also have lasting effects on growth, development, and overall quality of life. Continued research into the mechanisms driving immune activation and the development of targeted therapies is essential for improving the prognosis and management of HIV in children. Understanding the interplay between HIV, the immune system, and cytokine responses will facilitate the development of more effective treatments and interventions.[89]

Impact of ART on Cytokine Levels

Antiretroviral therapy (ART) has been a game-changer in the management of HIV infection, significantly reducing viral loads and improving immune function. ART's ability to suppress HIV replication has a profound effect on cytokine profiles in pediatric patients. By lowering viral loads, ART reduces the chronic immune activation and inflammation that are hallmarks of untreated HIV infection. Studies have shown that effective ART can lead to a significant reduction in the levels of pro-inflammatory cytokines such as IL-6, TNF-α, and IL-1β. This reduction in cytokine levels is associated with decreased immune activation and improved clinical outcomes.[90] Despite the effectiveness of ART in controlling HIV replication, some studies have reported that certain cytokines remain elevated in some patients, even when viral loads are undetectable. For instance, IL-6 and D-dimer levels often stay high, suggesting ongoing immune activation and inflammation. This residual immune activation can be attributed to several factors, including persistent viral reservoirs, microbial translocation, and immune system dysregulation. The persistence of elevated cytokine levels, despite viral suppression, underscores the complexity of HIV pathogenesis and the need for additional therapeutic strategies to address immune activation.[91] When ART fails or resistance develops, there is often a resurgence in viral load and a concomitant increase in immune activation. This is typically marked by a spike in pro-inflammatory cytokines, indicating a renewed state of immune system stress and inflammation. Monitoring cytokine levels in ART-resistant pediatric patients is crucial for understanding the extent of immune activation and guiding treatment adjustments. Increased cytokine levels can signal the need for a change in therapy, such as switching to a different ART regimen or adding additional treatments to address immune activation.[92] Cytokines serve as valuable biomarkers for monitoring the efficacy of ART in pediatric HIV patients. Regular measurement of cytokine levels can help clinicians assess the degree of immune activation and inflammation, providing insights into the effectiveness of the current treatment regimen. For instance, a decrease in IL-6 and TNF-α levels can indicate

successful suppression of immune activation, while persistently high levels might suggest ongoing issues such as drug resistance or inadequate viral suppression. Utilizing cytokines as biomarkers can enhance personalized treatment approaches, ensuring that each patient receives the most effective therapy based on their specific immune response.

Therapeutic Strategies for Cytokine Modulation

Given the role of cytokines in driving immune activation and inflammation, various therapeutic strategies are being explored to modulate cytokine responses in HIV-infected pediatric patients. These strategies include the use of cytokine inhibitors, anti-inflammatory agents, and immune modulators.

1. **Cytokine Inhibitors**: These drugs specifically target and block the activity of pro-inflammatory cytokines. For example, IL-6 inhibitors can reduce inflammation and potentially improve outcomes in HIV patients with high levels of this cytokine. Clinical trials are ongoing to evaluate the safety and efficacy of these inhibitors in pediatric populations.[93]
2. **Anti-Inflammatory Agents**: Medications such as corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) can help reduce systemic inflammation. While these agents are not specific to cytokines, they can provide symptomatic relief and reduce the overall inflammatory burden in HIV-infected patients.[94]
3. **Immune Modulators**: These therapies aim to restore balance to the immune system by enhancing the function of regulatory T cells or inhibiting pathways that contribute to immune activation. Immune modulators can help mitigate the chronic immune activation that persists despite ART, potentially reducing the risk of comorbidities and improving long-term health outcomes.[95]

Therapeutic Implications and Future Directions

Despite the significant advancements in antiretroviral therapy (ART), residual immune activation remains a critical challenge in the management of HIV-infected pediatric patients. Persistent elevations in pro-inflammatory cytokines, such as IL-6 and D-dimer, even in the context of effective ART, indicate ongoing immune dysregulation. Addressing this residual immune activation is crucial for reducing the risk of comorbidities and improving long-term health outcomes. Developing therapeutic strategies that specifically target these cytokines and their pathways could mitigate chronic inflammation and enhance the efficacy of ART.[96-97] One promising approach involves the use of cytokine inhibitors. These drugs can selectively block the activity of pro-inflammatory cytokines that contribute to chronic immune activation. For instance, IL-6 inhibitors, which have been studied in other inflammatory diseases, may offer benefits in managing persistent inflammation in HIV-infected pediatric patients. Ongoing clinical trials are exploring the safety and effectiveness of these inhibitors in this population. If successful, cytokine inhibitors could become a valuable adjunct to ART, helping to control immune activation and reduce related complications.[98-99] Anti-inflammatory agents, such as corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs), have long been used to manage inflammation in

various conditions. While these agents are not specific to the cytokine pathways involved in HIV, they can provide symptomatic relief and reduce overall inflammation. However, their long-term use in pediatric HIV patients must be carefully managed to avoid potential side effects. Research into more targeted anti-inflammatory therapies that can be safely used alongside ART is ongoing and holds promise for improving patient outcomes.[100]

Immune modulators represent another therapeutic avenue for addressing immune activation in HIV-infected pediatric patients. These therapies aim to restore immune balance by enhancing the function of regulatory T cells or inhibiting specific pathways involved in immune activation. For example, drugs that modulate the activity of the PD-1/PD-L1 pathway, which is involved in T cell exhaustion, may help rejuvenate immune responses and reduce chronic activation. Further research is needed to identify the most effective and safe immune modulators for use in children with HIV.[101] The variability in cytokine responses among HIV-infected pediatric patients underscores the need for personalized medicine approaches. Tailoring treatment strategies based on individual cytokine profiles can optimize therapy and improve outcomes. Regular monitoring of cytokine levels can help identify patients who may benefit from additional interventions, such as cytokine inhibitors or immune modulators. Personalized treatment plans that consider the unique immune status of each patient can enhance the effectiveness of ART and reduce the risk of long-term complications.[102] Combining ART with cytokine modulators and anti-inflammatory agents may offer a more comprehensive approach to managing HIV in pediatric patients. Combination therapies can target multiple aspects of the disease, addressing both viral replication and immune activation. For instance, pairing ART with an IL-6 inhibitor could help control viral load while simultaneously reducing inflammation. Clinical trials investigating various combination therapies are essential to determine the most effective and safe regimens for children.[103] Long-term studies are critical for understanding the impact of cytokine modulation on the growth, development, and overall health of HIV-infected pediatric patients. These studies can provide valuable insights into the benefits and potential risks of new therapies, guiding clinical practice. Regular follow-up and monitoring are essential to ensure that treatments are effective and to adjust strategies as needed. Long-term data will also help identify any delayed effects of cytokine modulation and inform future therapeutic developments. Chronic immune activation and systemic inflammation in HIV-infected pediatric patients contribute to the development of non-AIDS-related comorbidities, such as cardiovascular disease, neurocognitive impairments, and growth delays. Addressing cytokine dysregulation through targeted therapies could reduce the incidence of these comorbidities and improve quality of life. For example, reducing IL-6 levels might lower the risk of cardiovascular diseases, while modulating neuroinflammatory pathways could protect cognitive function and brain development.

Future Research Directions

Discovering reliable biomarkers for immune activation and inflammation will improve the ability to monitor disease progression and treatment efficacy. This can lead to more precise and timely interventions. Continued development of therapies that specifically target the pathways involved in chronic immune activation is essential. These therapies should be safe and effective for long-term use in children. Investigating the mechanisms underlying ART resistance and their

relationship with cytokine responses can inform strategies to overcome resistance and maintain viral suppression. Utilizing multi-omics approaches (e.g., genomics, proteomics, metabolomics) can provide a comprehensive understanding of the immune dysregulation in pediatric HIV patients and identify novel therapeutic targets.[103]

Conclusion

Cytokine responses play a critical role in the pathogenesis and progression of HIV infection in pediatric patients. Despite significant advancements in antiretroviral therapy (ART) that have transformed the prognosis for these patients, challenges such as chronic immune activation and residual inflammation persist. Elevated levels of pro-inflammatory cytokines, even in the context of effective ART, highlight the complex interplay between the virus and the immune system. Addressing these challenges is essential for improving long-term outcomes and the quality of life for HIV-infected children. The persistent activation of the immune system and the resultant cytokine dysregulation contribute to immune cell exhaustion, systemic inflammation, and increased susceptibility to opportunistic infections and non-AIDS-related comorbidities. Targeted therapies that modulate cytokine responses, such as cytokine inhibitors, anti-inflammatory agents, and immune modulators, hold promise for mitigating these adverse effects and enhancing the overall efficacy of ART. The incorporation of cytokine monitoring as biomarkers for disease progression and treatment efficacy can significantly enhance personalized medicine approaches. By tailoring treatment strategies based on individual cytokine profiles, healthcare providers can optimize therapy and achieve better patient outcomes. Combination therapies that address both viral replication and immune activation represent a comprehensive approach to managing HIV in pediatric patients.

References

1. Yu JC, Khodadadi H, Malik A, Davidson B, Salles ÉD, Bhatia J, Hale VL, Baban B. Innate immunity of neonates and infants. Frontiers in immunology. 2018; 9:1759.
2. Goenka A, Kollmann TR. Development of immunity in early life. Journal of Infection. 2015;71: S112-120.
3. 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.
4. 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
5. Obeagu EI, Obeagu, GU. Counting Cells, Shaping Fates: CD4/CD8 Ratios in HIV. Elite Journal of Scientific Research and Review, 2024; 2(1): 37-50
6. 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.
7. 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).

8. 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
9. 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.
10. Obeagu EI, Anyiam AF, Obeagu GU. Erythropoietin Therapy in HIV-Infected Individuals: A Critical Review. Elite Journal of HIV, 2024; 2(1): 51-64
11. Obeagu EI, Obeagu GU. Strength in Unity: Building Support Networks for HIV Patients in Uganda. Elite Journal of Medicine, 2024; 2(1): 1-16
12. 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
13. Kuhn L, Meddows-Taylor S, Gray G, Tiemessen C. Human immunodeficiency virus (HIV)–specific cellular immune responses in newborns exposed to HIV in utero. Clinical infectious diseases. 2002;34(2):267-276.
14. Muenchhoff M, Prendergast AJ, Goulder PJ. Immunity to HIV in early life. Frontiers in immunology. 2014; 5:391.
15. Geng ST, Zhang ZY, Wang YX, Lu D, Yu J, Zhang JB, Kuang YQ, Wang KH. Regulation of gut microbiota on immune reconstitution in patients with acquired immunodeficiency syndrome. Frontiers in microbiology. 2020; 11:594820.
16. 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
17. 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.
18. Obeagu EI, Obeagu GU. Assessing Platelet Functionality in HIV Patients Receiving Antiretroviral Therapy: Implications for Risk Assessment. Elite Journal of HIV, 2024; 2(3): 14-26
19. 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
20. Obeagu EI, Obeagu GU. Understanding ART and Platelet Functionality: Implications for HIV Patients. Elite Journal of HIV, 2024; 2(2): 60-73
21. 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
22. Obeagu EI. Erythropoietin and the Immune System: Relevance in HIV Management. Elite Journal of Health Science, 2024; 2(3): 23-35
23. 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
24. Obeagu EI, Obeagu GU. Anemia and Erythropoietin: Key Players in HIV Disease Progression. Elite Journal of Haematology, 2024; 2(3): 42-57

25. 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
26. 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
27. Ifeanyi OE, Obeagu GU. The values of prothrombin time among HIV positive patients in FMC owerri. International Journal of Current Microbiology and Applied Sciences. 2015;4(4):911-916. https://www.academia.edu/download/38320140/Obeagu_Emmanuel_Ifeanyi_and_Obeagu__Getrude_Uzoma2.EMMA1.pdf.
28. Yu JC, Khodadadi H, Malik A, Davidson B, Salles ÉD, Bhatia J, Hale VL, Baban B. Innate immunity of neonates and infants. Frontiers in immunology. 2018; 9:1759.
29. Levy O. Innate immunity of the newborn: basic mechanisms and clinical correlates. Nature Reviews Immunology. 2007;7(5):379-390.
30. Pieren DK, Boer MC, de Wit J. The adaptive immune system in early life: The shift makes it count. Frontiers in immunology. 2022; 13:1031924.
31. Rackaityte E, Halkias J. Mechanisms of fetal T cell tolerance and immune regulation. Frontiers in immunology. 2020; 11:588.
32. Sereme Y, Toumi E, Saifi E, Faury H, Skurnik D. Maternal immune factors involved in the prevention or facilitation of neonatal bacterial infections. Cellular Immunology. 2024; 395:104796.
33. Zhang X, Zhivaki D, Lo-Man R. Unique aspects of the perinatal immune system. Nature Reviews Immunology. 2017;17(8):495-507.
34. Sanidad KZ, Zeng MY. Neonatal gut microbiome and immunity. Current opinion in microbiology. 2020; 56:30-37.
35. Izuchukwu IF, Ozims SJ, Agu GC, Obeagu EI, Onu I, Amah H, Nwosu DC, Nwanjo HU, Edward A, Arunsi MO. Knowledge of preventive measures and management of HIV/AIDS victims among parents in Umuna Orlu community of Imo state Nigeria. Int. J. Adv. Res. Biol. Sci. 2016;3(10): 55-65.DOI; 10.22192/ijarbs.2016.03.10.009
36. Chinedu K, Takim AE, Obeagu EI, Chinazor UD, Eloghosa O, Ojong OE, Odunze U. HIV and TB co-infection among patients who used Directly Observed Treatment Short-course centres in Yenagoa, Nigeria. IOSR J Pharm Biol Sci. 2017;12(4):70-75. links/5988ab6d0f7e9b6c8539f73d/HIV-and-TB-co-infection-among-patients-who-used-Directly-Observed-Treatment-Short-course-centres-in-Yenagoa-Nigeria.pdf
37. Oloro OH, Oke TO, Obeagu EI. Evaluation of Coagulation Profile Patients with Pulmonary Tuberculosis and Human Immunodeficiency Virus in Owo, Ondo State, Nigeria. Madonna University journal of Medicine and Health Sciences. 2022;2(3):110-119.
38. Nwosu DC, Obeagu EI, Nkwocha BC, Nwanna CA, Nwanjo HU, Amadike JN, Elendu HN, Ofoedeme CN, Ozims SJ, Nwankpa P. Change in Lipid Peroxidation Marker (MDA) and Non enzymatic Antioxidants (VIT C & E) in HIV Seropositive Children in an Urban Community of Abia State. Nigeria. J. Bio. Innov. 2016;5(1):24-30. links/5ae735e9a6fdcc5b33eb8d6a/CHANGE-IN-LIPID-PEROXIDATION-MARKER-

MDAAND-NON-ENZYMATIC-ANTIOXIDANTS-VIT-C-E-IN-HIV-SEROPOSITIVE-CHILDREN-IN-AN-URBAN-COMMUNITY-OF-ABIA-STATE-NIGERIA.pdf.

39. Ifeanyi OE, Obeagu GU, Ijeoma FO, Chioma UI. The values of activated partial thromboplastin time (APTT) among HIV positive patients in FMC Owerri. Int J Curr Res Aca Rev. 2015; 3:139-144. https://www.academia.edu/download/38320159/Obeagu_Emmanuel_Ifeanyi3__et_al.IJCRAR.pdf.
40. Obiomah CF, Obeagu EI, Ochei KC, Swem CA, Amachukwu BO. Hematological indices o HIV seropositive subjects in Nnamdi Azikiwe University teaching hospital (NAUTH), Nnewi. Ann Clin Lab Res. 2018;6(1):1-4. links/5aa2bb17a6fdccd544b7526e/Haematological-Indices-of-HIV-Seropositive-Subjects-at-Nnamdi-Azikiwe.pdf
41. Omo-Emmanuel UK, Ochei KC, Osuala EO, Obeagu EI, Onwuasoanya UF. Impact of prevention of mother to child transmission (PMTCT) of HIV on positivity rate in Kafanchan, Nigeria. Int. J. Curr. Res. Med. Sci. 2017;3(2): 28-34.DOI: 10.22192/ijcrms.2017.03.02.005
42. Aizaz M, Abbas FA, Abbas A, Tabassum S, Obeagu EI. Alarming rise in HIV cases in Pakistan: Challenges and future recommendations at hand. Health Science Reports. 2023;6(8):e1450.
43. Obeagu EI, Amekpor F, Scott GY. An update of human immunodeficiency virus infection: Bleeding disorders. J Pub Health Nutri. 2023; 6 (1). 2023;139. links/645b4a6c2edb8e5f094d9bd9/An-update-of-human-immunodeficiency-virus-infection-Bleeding.pdf.
44. Obeagu EI, Scott GY, Amekpor F, Ofodile AC, Edoho SH, Ahamefula C. Prevention of New Cases of Human Immunodeficiency Virus: Pragmatic Approaches of Saving Life in Developing Countries. Madonna University journal of Medicine and Health Sciences. 2022;2(3):128-134. https://madonnauniversity.edu.ng/journals/index.php/medicine/article/view/86.
45. Walter O, Anaebo QB, Obeagu EI, Okoroiwu IL. Evaluation of Activated Partial Thromboplastin Time and Prothrombin Time in HIV and TB Patients in Owerri Metropolis. Journal of Pharmaceutical Research International. 2022:29-34.
46. Odo M, Ochei KC, Obeagu EI, Barinaadaa A, Eteng EU, Ikpeme M, Bassey JO, Paul AO. Cascade variabilities in TB case finding among people living with HIV and the use of IPT: assessment in three levels of care in cross River State, Nigeria. Journal of Pharmaceutical Research International. 2020;32(24):9-18.
47. Obeagu EI, Obeagu GU. A Review of knowledge, attitudes and socio-demographic factors associated with non-adherence to antiretroviral therapy among people living with HIV/AIDS. Int. J. Adv. Res. Biol. Sci. 2023;10(9):135-142.DOI: 10.22192/ijarbs.2023.10.09.015 links/6516faa61e2386049de5e828/A-Review-of-knowledge-attitudes-and-socio-demographic-factors-associated-with-non-adherence-to-antiretroviral-therapy-among-people-living-with-HIV-AIDS.pdf
48. Obeagu EI, Onuoha EC. Tuberculosis among HIV Patients: A review of Prevalence and Associated Factors. Int. J. Adv. Res. Biol. Sci. 2023;10(9):128-134.DOI:

10.22192/ijarbs.2023.10.09.014 links/6516f938b0df2f20a2f8b0e0/Tuberculosis-among-HIV-Patients-A-review-of-Prevalence-and-Associated-Factors.pdf.

49. Obeagu EI, Ibeh NC, Nwobodo HA, Ochei KC, Iwegbulam CP. Haematological indices of malaria patients coinfected with HIV in Umuahia. Int. J. Curr. Res. Med. Sci. 2017;3(5):100-104.DOI: 10.22192/ijcrms.2017.03.05.014 https://www.academia.edu/download/54317126/Haematological_indices_of_malaria_patients_coinfected_with_HIV.pdf
50. Okorie HM, Obeagu Emmanuel I, Okpoli Henry CH, Chukwu Stella N. Comparative study of enzyme linked immunosorbent assay (Elisa) and rapid test screening methods on HIV, Hbsag, Hcv and Syphilis among voluntary donors in. Owerri, Nigeria. J Clin Commun Med. 2020;2(3):180-183.DOI: **DOI:** 10.32474/JCCM.2020.02.000137 links/5f344530458515b7291bd95f/Comparative-Study-of-Enzyme-Linked-Immunosorbent-Assay-ElISA-and-Rapid-Test-Screening-Methods-on-HIV-HBsAg-HCV-and-Syphilis-among-Voluntary-Donors-in-Owerri-Nigeria.pdf.
51. Emannuel G, Martin O, Peter OS, Obeagu EI, Daniel K. Factors Influencing Early Neonatal Adverse Outcomes among Women with HIV with Post Dated Pregnancies Delivering at Kampala International University Teaching Hospital, Uganda. Asian Journal of Pregnancy and Childbirth. 2023 Jul 29;6(1):203-211. http://research.sdpublishers.net/id/eprint/2819/.
52. Vincent CC, Obeagu EI, Agu IS, Ukeagu NC, Onyekachi-Chigbu AC. Adherence to Antiretroviral Therapy among HIV/AIDS in Federal Medical Centre, Owerri. Journal of Pharmaceutical Research International. 2021;33(57A):360-368.
53. Madekwe CC, Madekwe CC, Obeagu EI. Inequality of monitoring in Human Immunodeficiency Virus, Tuberculosis and Malaria: A Review. Madonna University journal of Medicine and Health Sciences. 2022;2(3):6-15. https://madonnauniversity.edu.ng/journals/index.php/medicine/article/view/69
54. Echendu GE, Vincent CC, Ibebuike J, Asodike M, Naze N, Chinedu EP, Ohale B, Obeagu EI. WEIGHTS OF INFANTS BORN TO HIV INFECTED MOTHERS: A PROSPECTIVE COHORT STUDY IN FEDERAL MEDICAL CENTRE, OWERRI, IMO STATE. European Journal of Pharmaceutical and Medical Research, 2023; 10(8): 564-568
55. Wilson EM, Sereti I. Immune restoration after antiretroviral therapy: the pitfalls of hasty or incomplete repairs. Immunological reviews. 2013;254(1):343-354.
56. Misgena DK. The pattern of immunologic and virologic responses to Highly Active Antiretroviral Treatment (HAART): Does success bring further challenges? Ethiopian Journal of Health Development. 2011;25(1):61-70.
57. Davenport MP, Khoury DS, Cromer D, Lewin SR, Kelleher AD, Kent SJ. Functional cure of HIV: the scale of the challenge. Nature Reviews Immunology. 2019;19(1):45-54.
58. Geretti AM, Brook G, Cameron C, Chadwick D, French N, Heyderman R, Ho A, Hunter M, Ladhani S, Lawton M, MacMahon E. British HIV Association guidelines on the use of vaccines in HIV-positive adults 2015. HIV medicine. 2016;17(53):S2-81.
59. Laupèze B, Del Giudice G, Doherty MT, Van der Most R. Vaccination as a preventative measure contributing to immune fitness. npj Vaccines. 2021;6(1):93.
60. Obeagu EI, Obeagu GU. Immunological Aspects of HIV Control in Perinatally Infected Infants: A Review**.** Elite Journal of Immunology, 2024; 2(6): 1-14

61. Obeagu EI, Obeagu GU. An update on Early Immunological Markers in HIV-Exposed Infants**.** Elite Journal of Immunology, 2024; 2(6): 15-25
62. Obeagu EI. Markers of Immune Activation in HIV-Exposed Infants. Elite Journal of Health Science, 2024; 2(6): 1-14
63. Obeagu EI, Obarezi TN, Omeh YN, Okoro NK, Eze OB. Assessment of some haematological and biochemical parametrs in HIV patients before receiving treatment in Aba, Abia State, Nigeria. Res J Pharma Biol Chem Sci. 2014; 5:825-830.
64. Obeagu EI, Obarezi TN, Ogbuabor BN, Anaebo QB, Eze GC. Pattern of total white blood cell and differential count values in HIV positive patients receiving treatment in Federal Teaching Hospital Abakaliki, Ebonyi State, Nigeria. International Journal of Life Science, Biotechnology and Pharama Research. 2014; 391:186-189.
65. Obeagu EI. A Review of Challenges and Coping Strategies Faced by HIV/AIDS Discordant Couples. Madonna University journal of Medicine and Health Sciences. 2023; 3 (1): 7-12.
66. Oloro OH, Obeagu EI. A Systematic Review on Some Coagulation Profile in HIV Infection. International Journal of Innovative and Applied Research. 2022;10(5):1-11.
67. Nwosu DC, Obeagu EI, Nkwuocha BC, Nwanna CA, Nwanjo HU, Amadike JN, Ezemma MC, Okpomeshine EA, Ozims SJ, Agu GC. Alterations in superoxide dismutiase, vitamins C and E in HIV infected children in Umuahia, Abia state. International Journal of Advanced Research in Biological Sciences. 2015;2(11):268-271.
68. Nwovu AI, Ifeanyi OE, Uzoma OG, Nwebonyi NS. Occurrence of Some Blood Borne Viral Infection and Adherence to Universal Precautions among Laboratory Staff in Federal Teaching Hospital Abakaliki Ebonyi State. Arch Blood Transfus Disord. 2018;1(2).
69. Obeagu EI, Obeagu GU, Ede MO, Odo EO, Buhari HA. Translation of HIV/AIDS knowledge into behavior change among secondary school adolescents in Uganda: A review. Medicine (Baltimore). 2023;102(49): e36599. doi: 10.1097/MD.0000000000036599. PMID: 38065920; PMCID: PMC10713174.
70. Anyiam AF, Arinze-Anyiam OC, Irondi EA, Obeagu EI. Distribution of ABO and rhesus blood grouping with HIV infection among blood donors in Ekiti State Nigeria. Medicine (Baltimore). 2023;102(47): e36342. doi: 10.1097/MD.0000000000036342. PMID: 38013335; PMCID: PMC10681551.
71. Echefu SN, Udosen JE, Akwiwu EC, Akpotuzor JO, Obeagu EI. Effect of Dolutegravir regimen against other regimens on some hematological parameters, CD4 count and viral load of people living with HIV infection in South Eastern Nigeria. Medicine (Baltimore). 2023;102(47): e35910. doi: 10.1097/MD.0000000000035910. PMID: 38013350; PMCID: PMC10681510.
72. Opeyemi AA, Obeagu EI. Regulations of malaria in children with human immunodeficiency virus infection: A review. Medicine (Baltimore). 2023;102(46): e36166. doi: 10.1097/MD.0000000000036166. PMID: 37986340; PMCID: PMC10659731.
73. Obeagu EI, Ubosi NI, Uzoma G. Storms and Struggles: Managing HIV Amid Natural Disasters. Int. J. Curr. Res. Chem. Pharm. Sci. 2023;10(11):14-25.
74. Obeagu EI, Obeagu GU. Human Immunodeficiency Virus and tuberculosis infection: A review of prevalence of associated factors. Int. J. Adv. Multidiscip. Res. 2023;10(10):56-62.

75. Obeagu EI, Obeagu GU. Unmasking the Truth: Addressing Stigma in the Fight Against HIV. Elite Journal of Public Health. 2024;2(1):8-22.
76. Obeagu EI, Obeagu GU, Okwuanaso CB. Optimizing Immune Health in HIV Patients through Nutrition: A Review. Elite Journal of Immunology. 2024;2(1):14-33.
77. Obeagu EI, Obeagu GU. Utilization of immunological ratios in HIV: Implications for monitoring and therapeutic strategies. Medicine. 2024;103(9): e37354.
78. Obeagu EI, Obeagu GU. CD8 Dynamics in HIV Infection: A Synoptic Review. Elite Journal of Immunology. 2024;2(1):1-3.
79. Obeagu EI, Obeagu GU. Implications of B Lymphocyte Dysfunction in HIV/AIDS. Elite Journal of Immunology. 2024;2(1):34-46.
80. Obeagu EI, Obeagu GU. Maternal Influence on Infant Immunological Responses to HIV: A Review. Elite Journal of Laboratory Medicine. 2024;2(1):46-58.
81. Obeagu EI, Obeagu GU. Understanding B Lymphocyte Functions in HIV Infection: Implications for Immune Dysfunction and Therapeutic Strategies. Elite Journal of Medicine. 2024;2(1):35-46.
82. Obeagu EI, Obeagu GU. Platelet-Driven Modulation of HIV: Unraveling Interactions and Implications. Journal home page: http://www. journalijiar. com.;12(01).
83. Obeagu EI, Anyiam AF, Obeagu GU. Managing Hematological Complications in HIV: Erythropoietin Considerations. Elite Journal of HIV. 2024;2(1):65-78.
84. Obeagu EI, Obeagu GU, Hauwa BA, Umar AI. Hematocrit Variations in HIV Patients Co-infected with Malaria: A Comprehensive Review. Journal home page: http://www. journalijiar. com.;12(01).
85. Obeagu EI, Obeagu GU. Synergistic Effects of Blood Transfusion and HIV in Children Under 5 Years with Severe Malaria: A Review. Elite Journal of HIV. 2024;2(1):31-50.
86. Obeagu EI, Anyiam AF, Obeagu GU. Unveiling B Cell Mediated Immunity in HIV Infection: Insights, Challenges, and Potential Therapeutic Avenues. Elite Journal of HIV. 2024;2(1):1-5.
87. Obeagu EI, Obeagu GU. Hematocrit Fluctuations in HIV Patients Co-infected with Malaria Parasites: A Comprehensive Review. Int. J. Curr. Res. Med. Sci. 2024;10(1):25-36.
88. Obeagu EI, Obeagu GU. Transfusion Therapy in HIV: Risk Mitigation and Benefits for Improved Patient Outcomes. Sciences. 2024;4(1):32-37.
89. Obeagu EI, Obeagu GU. Mental Health and Psychosocial Effects of natural disaster on HIV Patients. Sciences. 2024;4(1):38-44.
90. Obeagu EI, Obeagu GU. Eosinophil-Associated Changes in Neonatal Thymic T Regulatory Cell Populations in HIV-Infected Pregnancies. Elite Journal of Health Science. 2024;2(1):33-42.
91. Obeagu EI, Obeagu GU. Advances in Understanding the Impact of Blood Transfusion on Anemia Resolution in HIV-Positive Children with Severe Malaria: A Comprehensive Review. Elite Journal of Haematology. 2024;2(1):26-41.
92. Obeagu EI, Ayogu EE, Obeagu GU. Interactions between Blood Transfusion and Antiretroviral Medications: Implications for Patient Care. Elite Journal of Medicine. 2024;2(2):104-115.

93. 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.
94. Obeagu EI, Anyanwu CN, Obeagu GU. Challenges and Considerations in Managing Blood Transfusion for Individuals with HIV. Elite Journal of HIV. 2024;2(2):1-7.
95. Obeagu EI, Ubosi NI, Obeagu GU, Akram M. Early Infant Diagnosis: Key to Breaking the Chain of HIV Transmission. Elite Journal of Public Health. 2024;2(1):52-61.
96. Obeagu EI, Obeagu GU. Understanding Hematocrit Fluctuations in HIV-Malaria Coinfection for Improved Management. Elite Journal of Public Health. 2024;2(1):22-34.
97. Obeagu EI, Obeagu GU. The Impact of Erythropoietin on Preeclampsia in HIV-Positive Women: A Review. Elite Journal of Nursing and Health Science. 2024;2(1):21-31.
98. Obeagu EI, Obeagu GU. Platelet Distribution Width (PDW) as a Prognostic Marker for Anemia Severity in HIV Patients: A Comprehensive Review. Journal home page: http://www. journalijiar. com.;12(01).
99. 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.
100. Obeagu EI, Obeagu GU. Assessing Platelet Functionality in HIV Patients Receiving Antiretroviral Therapy: Implications for Risk Assessment. Elite Journal of HIV. 2024;2(3):14-26.
101. Obeagu EI, Obeagu GU. Advancements in HIV Prevention: Africa's Trailblazing Initiatives and Breakthroughs. Elite Journal of Public Health. 2024;2(1):52-63.
102. Obeagu EI, Obeagu GU. Maternal Influence on Infant Immunological Responses to HIV: A Review. Elite Journal of Laboratory Medicine. 2024;2(1):46-58.
103. Obeagu EI, Obeagu GU. Counting Cells, Shaping Fates: CD4/CD8 Ratios in HIV. Elite Journal of Scientific Research and Review. 2024;2(1):37-50.