International Journal of Current Research and Review
DOI: doi.org/10.31782/IJCRR.2025.172402

Research Article

IJCRR
Section: Healthcare

Immunohaematological effects of *Giardia lamblia* and *Plasmodium spp.* co-infection in school-aged children, in Owerri West, Imo State, Nigeria

Nwachukwu, J.C.[1], Anyalogbu, E. A. A.[1], Nwachukwu, A. A.[1]

Department of Biotechnology, School of Biological Sciences, Federal University of Technology, Owerri, Nigeria.

ABSTRACT

Introduction: *Giardia lamblia* and *Plasmodium* species are the pathogenic organisms for Giardiasis and Malaria, respectively, both human protozoan infections. Parasitic infections have continued to pose significant health challenges to children, especially in developing countries.

Aim/Objective: This study investigated the immunohaematological effects of single and co-infections with *Giardia lamblia* and *Plasmodium* species among school-aged children (5–15 years) in Owerri West Local Government Area, Imo State, Nigeria.

Methods: A total of 108 pupils were sampled from 20 randomly selected primary schools across rural, semi-urban, and urban settings. Blood and stool samples were collected for parasitological and immunohaematological analysis. Parasite identification was performed using direct microscopy: Stool samples for *Giardia lamblia* and thick/thin blood smears for *Plasmodium* spp. White blood cell (WBC) counts and differentials were measured using standard haematological techniques.

Results: The total parasite load was $272.36\times10^5\pm1.32$ parasites/µm, with *Giardia* and *Plasmodium* accounting for $76.2\times10^5\pm1.24$ µm and $104.4\times10^5\pm0.88$ µm, respectively. Co-infection was recorded at $91.76\times10^5\pm1.08$ µm. The total WBC count in co-infected pupils ($15.4\times10^9\pm0.03$/L) was higher than in Giardia-only ($6.95\times10^9\pm0.02$/L), *Plasmodium*-only ($7.3\times10^9\pm0.02$/L), and control ($7.05\times10^9\pm0.01$/L) groups. Differential counts revealed marked elevations in total WBCs, neutrophils, eosinophils, basophils, monocytes, and lymphocytes in co-infection cases, with percentage values of 118.44, 34.38, 361.54, 566.67, 169.23, and 26.67%, respectively.

Conclusion: The findings highlight the intensified immune response in co-infections and underscore the importance of integrated surveillance for parasitic diseases.

Key Words: *Giardia lamblia, Plasmodium spp*, co-infection, Haematology, White blood cells.

INTRODUCTION

Parasitic infections remain a leading cause of morbidity in sub-Saharan Africa, particularly among children. *Giardia lamblia*, a flagellated protozoan, and *Plasmodium* spp., the causative agent of malaria, are among the most common protozoan parasites affecting school-aged children. These infections contribute to malnutrition, anaemia, and immunosuppression, particularly in resource-limited settings.[1] While the individual impacts of these parasites are well-documented, the immunohaematological effects of co-infection have received limited attention.

Infection with *G. lamblia* typically leads to malabsorption and gastrointestinal disturbances, while *Plasmodium* targets erythrocytes, resulting in anaemia and systemic inflammation.[2,3] The overlapping endemicity of both parasites raises concern over their synergistic impact on children's health, particularly their immunohaematological profiles, which are crucial for diagnosing disease severity and guiding treatment. This study seeks to elucidate the immunohaematological alterations, especially WBC responses, among children with single and co-infections of *G. lamblia* and *Plasmodium* spp in Owerri West

METHODOLOGY

Sample Population/Area: This study was conducted among 108 pupils aged 5–15 years, drawn from 20 government-approved primary schools in Owerri West LGA, Imo State,

Corresponding Author:
Nwachukwu, J. C., Department of Biotechnology, School of Biological Sciences, Federal University of Technology, Owerri, Nigeria.
Email: iheomasinachi@gmail.com

ISSN: 2231-2196 (Print) **ISSN:** 0975-5241 (Online)
Received: 02.11.2025 **Revised:** 27.11.2025 **Accepted:** 16.12.2025 **Published:** 29.12.2025

Nigeria. Schools were selected to reflect rural, semi-urban, and urban settings. Following screening of the pupils for *Plasmodium species* (blood) and *Giardia lamblia* (stool), those with parasitic infection were considered the test group. In contrast, those without parasitic infection who are healthy were considered the control group. Ethical clearance and informed consent were obtained from relevant authorities and parents/guardians.

Sample Collection and Analysis: Stool samples were collected using a random sampling technique from each participant using clean, wide-mouthed, screw-capped sterile universal bottles. The pupils (and their parents/guardians) were instructed to collect the sample carefully to avoid contamination with urine or soil. The samples were labelled with unique identification numbers and transported to the laboratory for immediate parasitological analysis.

Stool samples were examined microscopically using saline and iodine wet mounts to detect *G. lamblia* cysts and trophozoites. Blood samples were collected via finger prick and stained with Giemsa for thick and thin smear analysis to identify *Plasmodium* species. Parasite counts were recorded as parasites per micrometre (µm).

Haematological analysis involved the use of an automated haematology analyser to measure total and differential white blood cell counts, including neutrophils, eosinophils, basophils, monocytes, and lymphocytes.

Data Analysis: Statistical comparisons were performed using one-way ANOVA with post hoc tests to determine significance (at a 5% confidence level) between infection groups.

RESULTS

Parasite Count

The total parasite count observed was $272.36\times10^5\pm1.32$/µm. *G. lamblia* accounted for $76.2\times10^5\pm1.24$ µm, *Plasmodium* spp for $104.4\times10^5\pm0.88$ µm, and co-infection for $91.76\times10^5\pm1.08$ µm. No parasites were observed in the 30 control samples (Table 1)

White Blood Cell Parameters

Table 2 summarizes the mean total and differential white blood cell counts for the test groups and the control group. The total WBC count in the control group ($7.05 \times 10^9\pm0.01$/L) was similar to that in the *Giardia* and *Plasmodium* groups, but lower than in the *Plasmodium* + *Giardia* group ($15.4 \times 10^9\pm0.03$/L). Neutrophil counts were also highest in the *Plasmodium* + *Giardia* group ($4.3\pm0.04 \times 10^9$/L) compared to the control ($3.2 \times 10^9\pm0.01$/L). The eosinophil count was significantly elevated in the *Plasmodium* + Giardia group ($2.4 \times 10^9\pm0.08$/L) compared to the control ($0.52 \times 10^9\pm0.02$/L). Basophils, monocytes, and lymphocytes followed similar patterns, with elevated levels observed in the *Plasmodium* + *Giardia* group.

Percentage Changes Relative to Control

Figure 4.3 presents the effect of parasitic infections on total and differential white blood cell (WBC) counts. Analysis of the data shows that the total WBC count was minimally decreased by Giardia (0.71%) but elevated under co-infection (*Plasmodium* + *Giardia*) at 118.44%, compared to a moderate increase in *Plasmodium* alone at 3.55%. Neutrophil levels also showed increase under co-infection at 34.38%, compared to an increase of 18.75% in *Plasmodium* spp and a decrease of 6.25% in *Giardia* infection.

Eosinophils were drastically reduced in *Plasmodium* alone (80.77%), while co-infection led to a substantial increase (361.54%), and a minor decrease was observed in *Giardia* (1.92%). Basophil counts followed a similar trend, with a significant reduction in *Plasmodium* (14.29%), a sharp increase in co-infection (566.67%), and a small decrease in Giardia (4.76%).

Monocyte counts were elevated across all infections, with co-infection showing the highest increase at 169.23%, followed by *Plasmodium* (111.54%) and *Giardia* (40.39%). Lymphocyte levels were reduced in *Plasmodium* (30.0%), but showed increases in *Giardia* (10.0%) and co-infection (26.67%).

DISCUSSION

Effective *Plasmodium* control measures, including vector control, prompt diagnosis, and anti-Plasmodium treatment, are critical in reducing the disease burden.

The co-infected group's mean parasite count suggests potential additive effects or interactions between parasites that enhance survival and proliferation. Co-infections complicate disease management, necessitating integrated approaches, including enhanced diagnostic tools like multiplex PCR assays and tailored treatment strategies.[4]

White blood cells (WBCs) play a critical role in the immune system, defending the body against infections. The minimal reduction may suggest that *Giardia* elicits an immune response. According to Farthing[5], Giardia infections primarily recruit lymphocytes and macrophages to the site of infection, resulting in moderate immune activation.

The WBC count elevation in plasmodium-infected children reflects the immune response to *Plasmodium*, which triggers the activation and recruitment of neutrophils, macrophages, and lymphocytes.[6] Co-infections are known to exacerbate immune responses, leading to pronounced viral infections

often damage the respiratory epithelium and alter immune signaling. When a secondary bacterial infection occurs, the immune system mounts a "super-response." While viral infections often cause leukopenia (low WBC) or lymphopenia, the addition of a bacterial pathogen typically triggers a sharp rise in neutrophils (neutrophilia), resulting in overall leukocytosis.[7] Both *Plasmodium* and *Giardia* infections trigger inflammatory responses, and their co-occurrence likely amplifies this effect, creating a synergistic immune reaction This heightened immune activity may also signal systemic inflammation, which, if prolonged, could lead to immune dysregulation and increased susceptibility to other infections.[8]

Neutrophils are the most abundant type of WBC and are responsible for responding to bacterial infections and inflammation. The slightly lower neutrophil count in children with *Giardia* infection might reflect a subdued inflammatory response since Giardia is typically more associated with gastrointestinal disturbances than systemic inflammation.[9]

In *Plasmodium*-infected children, the increase in neutrophil count reflects the body's response to the parasitic infection, as neutrophils play a key role in fighting off *Plasmodium* species. The further increase in the co-infection group supports the hypothesis that multiple infections stimulate a stronger immune response, necessitating higher production of neutrophils to manage the increased pathogenic load.[10]

However, the high increase in neutrophils in co-infected children raises concerns about potential neutrophil-induced tissue damage, which can occur when the immune response becomes overly aggressive. This can lead to complications, particularly in children whose bodies are still developing. The correlation between parasite count and neutrophil count is 0.924, indicating a strong positive correlation. While neutrophils are typically associated with bacterial infections, they also respond to parasitic infections, particularly in the early stages when the immune system is activated.[11] The increase in neutrophil count in response to higher parasite load may reflect the body's attempt to prevent secondary bacterial infections that can arise due to the compromised immune system.

Eosinophils are typically elevated in allergic reactions and parasitic infections. Their reduction in Giardia-infected children aligns with the report of Nash et al.,[12] indicating a mild eosinophilic response to Giardia, a common parasitic infection in children.

The lower eosinophil count in Plasmodium-infected children could be due to suppression of eosinophil production by the *Plasmodium* parasite, as observed in other studies.[13] However, the dramatic increase in eosinophil count in the co-infection group may likely be due to the combined effect of both parasitic infections, triggering a strong eosinophilic response. Elevated eosinophils in this context may indicate a more severe immune reaction, with both protective and pathological consequences.[14] Eosinophils play a crucial role in combating parasitic infections by releasing cytotoxic granules containing enzymes that target and kill parasites.[15] Additionally, eosinophils can modulate inflammation and recruit other immune cells.[16] The elevated eosinophil count in the co-infected group reflects the activation of this response in defence against the protozoan parasites. Eosinophilia is often seen in parasitic infections, particularly those involving tissue invasion.[9]

Basophils are involved in inflammatory responses, particularly in allergies and parasitic infections. The lower basophil counts in both Giardia- and Plasmodium-infected children indicate that basophils may not be substantially mobilised in response to both infections. However, the dramatic increase in the co-infection group may likely reflect an enhanced inflammatory response due to the presence of both parasites, triggering the release of histamine and other inflammatory mediators from basophils.[17] Elevated basophils are often associated with severe or chronic infections, indicating a more complex immunopathology in co-infected children. Basophils can amplify immune responses by promoting the recruitment and activation of other immune cells, enhancing inflammation, and facilitating tissue repair.[18] They also interact with eosinophils and mast cells to coordinate immune responses against parasites[19] However, excessive basophil activity could exacerbate allergic reactions, potentially leading to complications for children with pre-existing allergies.[17] The increase in basophil count with higher parasite loads underscores their active involvement in combating parasitic infections.

Monocytes are crucial for phagocytosis and activating the adaptive immune response. The elevated monocyte count in both Giardia- and Plasmodium-infected children reflects the body's attempt to clear the infections through phagocytosis. Monocytes differentiate into macrophages, which engulf pathogens and produce inflammatory cytokines that regulate the immune response.[20] Thus, the elevated monocyte count in co-infected children likely indicates a heightened immune response to both parasites. However, elevated monocyte levels may also point to chronic inflammation, which can lead to immune dysregulation, increasing susceptibility to infections and chronic diseases.

The higher lymphocyte count in Giardia-infected children indicates activation of the adaptive immune response to the parasitic infection, whereas the decrease in the *Plasmodium*-infected group may be due to lymphocyte sequestration or apoptosis induced by *Plasmodium*, impairing the immune response.[21] The elevated count in the co-infection group may reflect an attempt by the immune system to respond to multiple antigens, although it could lead to immune system overactivation and potential exhaustion over time.[22,23] The in-

creased lymphocytes in Giardia and co-infected children suggest an active adaptive immune response, while the decrease in *Plasmodium*-infected children could point to lymphocyte sequestration or apoptosis, common in *Plasmodium*.[21] The mixed response in co-infected children may indicate a complex immune interaction between both parasites, potentially leading to an imbalanced immune response.

The correlation between parasite load and lymphocyte count (0.522) indicates a moderate positive correlation. Lymphocytes are involved in immune responses to parasitic infections, such as antibody production by B cells and killing infected cells by T cells.[24]

CONCLUSION

This study shows the high immunohaematological burden of co-infection of *G. lamblia* and *Plasmodium* spp. among children. Co-infected individuals exhibited elevated total and differential WBC counts, reflecting intensified immune activity. Consequently, public health interventions should prioritise surveillance of *G. lamblia* and *Plasmodium* spp. co-infection among children and integrated management strategies in endemic communities.

Ethics Approval: Approved by the Institutional Review Board, Federal University of Technology Owerri. Approval Number: FUTO/SOBS/REC/2025/0017

Conflicts of Interest

The authors declare no conflicts of interest.

Funding

This research did not receive any specific grant from funding agencies.

Author Contributions

J. C. Nwachukwu: Conceptualization, Methodology, Investigation, Writing – Original Draft.

E. A. A. Anyalogbu: Methodology, Data Curation, Formal Analysis.

A. A. Nwachukwu: Investigation, Writing – Review & Editing.

Competing Interests Disclaimer: Authors have declared that they have no known competing financial interests OR non-financial interests, OR personal relationships that could have appeared to influence the work reported in this paper.

REFERENCES

1. World Health Organisation, WHO. World Plasmodium report 2022: Tracking progress and gaps in the global response to Plasmodium. 2022. Retrieved from https://www.who.int/teams/global-Plasmodium-programme/reports/world-Plasmodium-report-2022
2. Meibalan E, Marti M. Biology of malaria transmission. Cold Spring Harbour Perspectives in Medicine, 2017: 7(3), 25-34.
3. Ryan U, Zahedi A. Giardia: Taxonomy, molecular epidemiology and drug resistance. Pathogens, 2019: 8(4), 227-238
4. Mahende C, Mandara CI, Ngasala B, Mwambuli A, Ngatunga D. Multiplex PCR assays for the detection of multiple parasitic infections in endemic regions. BMC Infectious Diseases, 2021: 21(1), 123-133.
5. Farthing MJ. Giardiasis. Gastroenterology Clinics of North America, 1996: 25(3), 493-515.
6. Perlmann P, Troye-Blomberg M. Plasmodium blood-stage infection and its control by the immune system. Folia Biologica, 2000: 46(6), 210-218.
7. McCullers JA. The co-pathogenesis of influenza viruses with bacteria in the lung. Nature Reviews Microbiology, 2014: 12(4), 252-262.
8. Ecker A, Donkor S, Wutor V, Opara J. Giardia lamblia infection alters haematological parameters and plasma cytokine profiles in children and adults in Ghana. Pathogens, 2022: 11(6), 679-690.
9. Ali S, Rashid M, Malik S. Immune response in Giardia lamblia infection: A review. Journal of Immunology Research, 2020: 1-10.
10. Taylor SM, Haldar K. Haemolysis and haemoglobinopathy in Plasmodium falciparum: An evolving hypothesis. Parasitology International, 2013: 62(4), 565-573.
11. Cummings DAT. Neutrophils and parasitic infections: Immune defence mechanisms in children. Immunology and Infection, 2020: 132(4), 564-572.
12. Nash TE, Herrington DA, Losonsky GA. Clinical and immunologic aspects of giardiasis: The saga of human giardiasis continues. Journal of Infectious Diseases, 2021: 223(9), 1514-1521.
13. White NJ. Anaemia and Plasmodium. Plasmodium Journal, 2018: 17(1), 371-386.
14. Katz N, Aquino EM, Andrade JP. Eosinophils and parasitic infections: New insights into an old association. Frontiers in Immunology, 2019: 10, 1642-1653.
15. Ramalho-Pinto FJ, Gazzinelli-Guimaraes PH, Gomes KX, de Mattos MCFI, de Castro EPF, Bueno LL. Eosinophils as key players in Giardiasis. Frontiers in Immunology, 2022: 13, 836-850.
16. Quintero JP, Dutta S, Saha B. Eosinophils and monocytes: Emerging roles in immune regulation in Plasmodium. Frontiers in Immunology, 2021: 12, 699-714.
17. Ghosh K, Ghosh S, Maitra A. Role of basophils in parasitic infections. Asian Pacific Journal of Tropical Medicine, 2017: 10(11), 997-1002.
18. Mukai K, Tsai M, Starkl P, Marichal T, Galli SJ. IgE and mast cells in host defence against parasites and venoms. Seminars in Immunopathology, 201638(5), 581-603.
19. Zarebavani M, Karimzadeh K, Naserifar R. Basophils: Key players in Plasmodium infection and therapeutic strategies. Frontiers in Immunology, 2022: 13, 926-936.
20. Cerban R, Moromizato MC, Chung G, Ranasinghe S. Giardia lamblia infection: A narrative review of the immunological mechanisms and potential therapeutic targets. Frontiers in Immunology, 2022: 13, 898-919.
21. Ndungu FM, Urban BC, Marsh K, Langhorne J. Regulation of immune responses by the spleen during Plasmodium infection. Parasite Immunology, 2018: 40(8), 400-413.
22. Okell LC, Bousema T, Griffin JT, Ouédraogo AL, Ghani AC, Drakeley CJ. Factors determining the occurrence of submicro-

scopic Plasmodium infections and their relevance for control. Nature Communications, 2019: 10(1), 1-13.
23. Dutta S, Saha B. Differential immune response in co-infections: A roadmap to therapeutic interventions. Frontiers in Immunology, 2020: 11(6), 196-208.
24. Cai H, Mu Q, Xiong H, Liu M, Yang F, Zhou L, Zhou B. Regulatory B cells in parasitic infections: Roles and therapeutic potential. Parasitology Research, 2025:124, 5

Table 1: Distribution of *Parasites* (Plasmodium spp and Giardia Species) in the Test group of selected sample population

	Infection					
	Giardia	**Plasmodium**	**Gir. + Pla.**	**Total infected**	**None/Control**	**Total sample**
Parasite count (µm)	$76.2x10^5 \pm 1.24^a$	$104.4x10^5 \pm 0.88^b$	$91.76x10^5 \pm 1.08^c$	$272.36x10^5 \pm 1.32$	Nil	-

Values are means ± standard deviations of triplicate determinations. Values with dissimilar alphabet superscripts are statistically significant ($p<0.05$) from each other across the row.

Table 2: Mean Total and Differential white blood cell count (x 10^9/L)

Parameter	**Tests**			**Control**
	Giardia	**Plasmodium**	**Pla. + Giar**	
Sample size	20	34	24	30
Total WBC	7.00±0.01	7.3±0.04	15.4±0.03	7.05±0.04
Neutrophils	3.00±0.02	3.8±0.07	4.3±0.04	3.2±0.07
Eosinophils	0.51±0.01	0.1±0.01	2.4±0.08	0.52±0.02
Basophils	0.20±0.02	0.18±0.02	1.4±0.07	0.21±0.01
Monocytes	0.73±0.03	1.10±0.01	1.4±0.02	0.52±0.03
Lymphocytes	3.3±0.05	2.1±0.01	3.8±0.11	3.00±0.10

Values are Means ± Std deviations of triplicate determinations

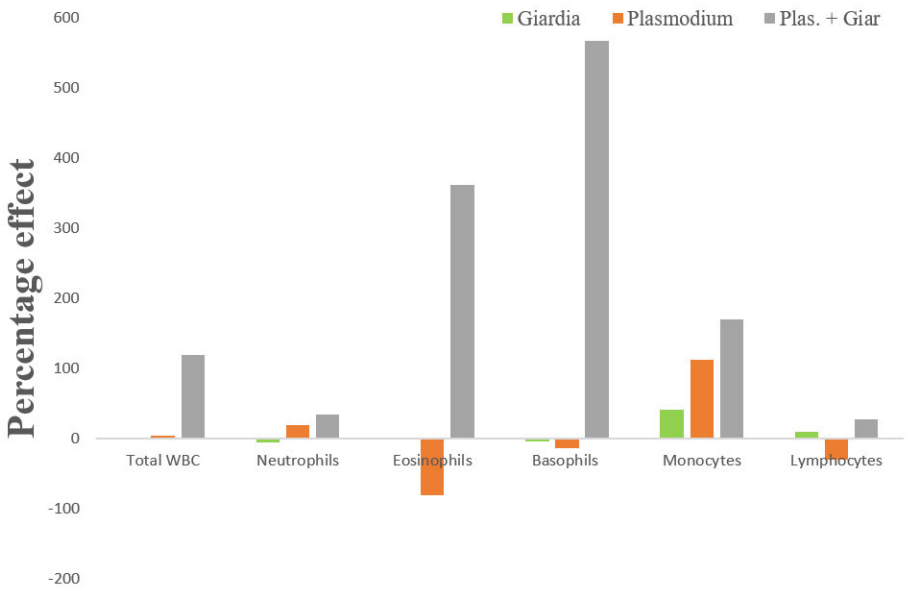

Figure 1: Percentage effect of parasites on total and differential white blood cell count.