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# Serum Cytokine and Haematological Profiles of Anaemic Children Aged 6 to 60 Months Old in Port-Harcourt, Nigeria

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## ABSTRACT

**Background:** Childhood anaemia is a serious paediatric health challenge in Sub-Saharan Africa. The accompanying high mortality rates in this region may be attributed to high prevalence of poverty, infections, malnutrition and poor healthcare facilities. Cytokines are thought to influence the development of anaemia in various pathologic conditions through mechanisms such as dyserythropoiesis and increased lysis of red blood cells.
**Objective:** This study was aimed at evaluating the relationship between serum levels of some cytokines viz. IL-6, IL-10 and IL-17 and anaemic parameters in children aged 6 to 60 months in University of Port-Harcourt Teaching Hospital, Nigeria.
**Methods:** This was a case-control study of 36 anaemic subjects and 36 non-anaemic controls. Full blood counts, levels of IL-6, IL-10 and IL-17 were compared between the two groups of children.
**Results:** There was a significant difference between the two groups (anaemic and controls) in the distribution of various haematological parameters (haemoglobin (Hb), haematocrit, MCV, MCHC, MCH, reticulocyte count), ($P < 0.05$). IL-6 level was significantly higher in anaemic children than in controls ($P = 0.001$). There were no statistically significant differences in the IL-10 and IL-17 levels between the groups. Significant correlations were observed between IL-6 and Hb ($r = -0.34$, $P = 0.041$). No correlation was found between IL-10, IL-17 and Hb.
**Conclusion:** These results suggest that IL-6 rather than IL-10 and IL-17 might have played a role in inducing the anaemia and could be a potential marker for therapeutic monitoring in anaemia.

***Keywords:*** Anaemia, Cytokines, Haemoglobin, Interleukin-6, Interleukin-10, Interleukin-17

## INTRODUCTION

Anaemia, a condition where haemoglobin level is below 11g/dL, [1] is a serious health challenge among children in the Sub-Saharan region of Africa. [2] Children aged 6 to 60 months with haemoglobin concentration below 7g/dL [3] or haematocrit value less than 21% are considered to have severe anaemia. [4] Aetiological factors of childhood anaemia include micronutrient deficiencies (such as iron, vitamin A, folate), human immunodeficiency virus (HIV) infections, bacteraemia, glucose-6-phosphate-dehydrogenase (G6PD) deficiency, hookworm infestations, malaria, [5-7] haemoglobinopathies such as sickle cell anaemia [8, 9] and thalassemias, helminth infections such as hookworm and *Schistosoma haematobium*, [10] and cancer. [11] Anaemia may have serious consequences such as impaired cognitive and motor development, impaired immune function and reduced growth [7] and decreased survival rates. [10]

The role of cytokines in the development of anaemia is not well understood.[12]Pro-inflammatory cytokines such as interleukin (IL)-17 are known to be potent inducers of inflammation. [13, 14] IL-17 is a novel cytokine produced by a subset of CD4+ T helper cells called Th17 cells. [14] It upregulates the expression of other pro-inflammatory cytokines mainly IL-6 and tumour necrosis factor (TNF)-α which are known to be involved in dysregulation of iron homeostasis and erythropoiesis thereby causing anaemia. [11, 14] IL-17 has been linked with inflammatory disorders such as rheumatoid arthritis, asthma, multiple sclerosis, lupus, aplastic anaemia [14] and autoimmune haemolytic anaemia. [15] It is yet to be associated with uncategorized anaemia, iron deficiency anaemia or iron homeostasis [12] in a clinical setting. This is despite the finding that it could inhibit both granulocytic and erythroid lineages in vivo and in laboratory mice, with more mature haematopoietic progenitors being most susceptible to its effect. [16]

IL-6 is a pleiotropic cytokine with pro-inflammatory, haematopoietic and immunomodulatory effects. [17] IL-6 negatively correlates with haemoglobin concentrations in various diseases and it has been linked with anaemia of inflammation. [18] Studies have shown that administration of recombinant IL-6 induces anaemia with decreases seen in haemoglobin level, haematocrit level and red blood cell numbers. [17] By stimulating production of the iron-regulatory hormone, hepcidin, IL-6 promotes iron sequestration thereby restricting the availability of iron for red cell production, hence anaemia develops. [19] IL-10 produced by T helper 2 (Th2) cells is an anti-inflammatory cytokine that inhibits production of cytokines by Th1 cells. It has been demonstrated both in vitro and in mice to stimulate bone marrow activity and counteract anaemia by mediating feedback regulation of TNF-alpha. [20] IL-10 is believed to provide protective effects against severe anaemia, especially in malaria by preventing overproduction of pro-inflammatory mediators, [21, 22] hence it is possible that insufficient levels of this cytokine may contribute to pathogenesis of anaemia. [20]

This study was therefore aimed at evaluating the relationship between serum concentrations of cytokines and anaemia in children aged 6 to 60 months in University of Port-Harcourt Teaching Hospital, Nigeria. The objectives were: to determine the levels of serum cytokines (IL-17, IL-6 and IL-10) in anaemic children aged 6 to 60 months; to compare these cytokines (IL-17, IL-6 and IL-10) levels in anaemic children with the controls; and to determine the severity of anaemia in children thereby making these cytokines a potential therapeutic target.

## MATERIALS AND METHODS

This case-control study was conducted in the Paediatrics and Child Health Department of University of Port-Harcourt Teaching Hospital, Port-Harcourt, Nigeria. Seventy-two children aged 6 to 60 months were consecutively enrolled into the study. Thirty-six children who had haemoglobin level of less than 11.0g/dl were included as the subjects while thirty-six children who had haemoglobin level of 11.0g/dl or higher served as the controls. Children were excluded from the study if they had received blood transfusion in the past three months and/or received anthelmintic drugs and iron supplements in the past one month prior to the beginning of the study. Ethical permission to conduct this study was issued by the Hospital Ethical Committee of University of Port-Harcourt Teaching Hospital. Written informed consents were obtained from the parents of each enrolled child prior to being included in the study. Three millilitres (3 ml) of whole blood was obtained from each patient and control. One ml (1 ml) of the blood was dispensed into EDTA anticoagulant tubes/specimen bottles for determination of haematological parameters using Rayto RT-7200 Automated Haematology Analyzer

(Rayto, Shenzhen, China). Two millilitres (2 ml) was transferred into plain polypropylene tubes (anti-coagulant free) and processed to obtain serum by centrifuging at 1000×g for 15 mins. The serum samples were aliquoted and stored at – 20$^{o}$C until cytokine assays were performed.

Measurement of serum IL-6, IL-10 and IL-17 levels were done separately with the help of commercially available human enzyme-linked immunosorbent assay (ELISA) kits from Aviva Systems Biology, San Diego, CA, USA. The assays employed quantitative sandwich enzyme immunoassay technique and were performed following manufacturer's instructions.

**Statistical Analysis:**

The data were analysed using SPSS statistical package version 20. Data were summarized as frequency, percentage, median and presented as tables and pie charts. The Mann-Whitney *U* test was used for comparisons of non-parametric data between the two groups. Fisher's exact test was used to analyse categorical variables between groups. Pearson's correlation test was used to study correlations between various variables. A two-tailed *p*-value of less than 0.05 was considered significant for all statistical comparisons.

## RESULTS

The age and gender distribution of the subjects and controls are shown in Table 1. In our study, the mean age of the anaemic children was 27.08 ± 18.81 months while the mean age of the non-anaemic group was 38.86 ± 15.78 months.

**Table 1: Age and gender distribution of the study population**

| Variable | ANAEMIC SUBJECTS (n = 36) | CONTROL (n = 36) | *P* value |
|---|---|---|---|
| | Median (range) or N (%) | Median (range) or N (%) | |
| Age of Child (Months) | 21.0 (7.0 – 60.0) | 36.0 (11.0 – 60.0) | 0.003 |
| Sex | | | 1.000 |
| Male | 22 (61.1) | 22 (61.1) | |
| Female | 14 (38.9) | 14 (38.9) | |

There were statistically significant differences between all the haematologic parameters in the two groups (subjects and control) except for the total white blood cells (WBC), platelets and WBC differential as shown in Table 2.

**Table 2: Haematological profile of the cases compared to controls**

| Parameter | ANAEMIC SUBJECTS (n=36) | CONTROL (n=36) | *P* value |
|---|---|---|---|
| | Median (Range) | Median (Range) | |
| Haemoglobin (g/dl) | 9.65 (3.50 – 10.90) | 11.85 (11.00 – 13.50) | < 0.001 |
| Total WBC (×10$^{9}$/L) | 9.75 (3.60 – 87.60) | 8.25 (4.40 – 17.90) | 0.186 |
| Haematocrit (%) | 32.00 (11.00 – 37.00) | 39.00 (36.00 – 44.00) | < 0.001 |
| RBC (×10$^{12}$/L) | 4.15 (1.20 – 5.40) | 4.60 (4.20 – 5.20) | < 0.001 |
| MCV (fl) | 79.95 (62.1 – 98.0) | 83.90 (77.10 – 97.70) | 0.003 |
| MCH (pg) | 23.35 (13.70 – 29.70) | 25.05 (23.00 – 30.50) | < 0.001 |
| MCHC (g/dl) | 29.15 (22.10 – 31.90) | 30.05 (28.20 – 33.60) | < 0.001 |
| RDW (%) | 18.85 (13.30 – 28.60) | 17.20 (13.40 – 20.00) | 0.002 |
| Platelets (×10$^{9}$/L) | 306.00 (56.00 – 982.00) | 282.00 (138.00 – 614.00) | 0.510 |
| Reticulocyte count (%) | 3.20 (1.60 – 4.20) | 2.00 (0.90 – 3.20) | < 0.001 |
| Neutrophil (×10$^{9}$/L) | 2.40 (0.00 – 11.90) | 2.50 (0.10 – 5.80) | 0.888 |
| Lymphocyte (×10$^{9}$/L) | 5.25 (2.40 – 43.80) | 4.80 (2.40 – 10.60) | 0.143 |
| Monocyte (×10$^{9}$/L) | 0.70 (0.10 – 8.40) | 0.55 (0.10 – 1.80) | 0.273 |
| Eosinophil (×10$^{9}$/L) | 0.50 (0.00 – 3.30) | 0.50 (0.20 – 1.80) | 0.932 |
| Basophil (×10$^{9}$/L) | 0.01 (0.00 – 1.30) | 0.01 (0.00 – 0.40) | 0.667 |

**WBC** = white blood cells, **RBC** = red blood, **MCV** = mean cell volume, **MCH** = mean cell haemoglobin, **MCHC** = mean cell haemoglobin concentration, **RDW** = red cell distribution width.

Eleven subjects (30.6%) had mild anaemia, 23 (63.9%) had moderate anaemia and 2 (5.6%) had severe anaemia as shown in Figure 1.

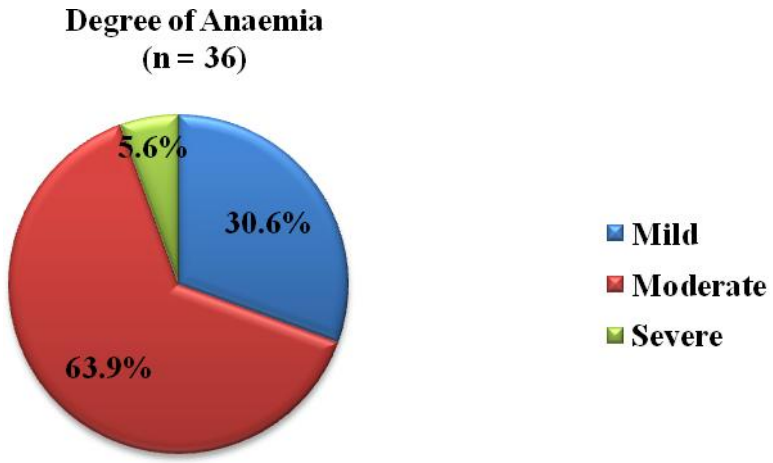


**Figure 1:** Severity of anaemia among the subjects. This shows that majority (63.9%) of children in the anaemic group had moderate anaemia.

The concentrations of IL-6 between the two groups were significantly higher in the anaemic children. The mean level of IL-6 in the subjects was 41.22 ± 57.13 pg/ml compared to the mean in the control group 15.35 ± 6.12 pg/ml ($p$ = 0.001) as shown in Table 3.

**Table 3: Cytokine profile of the studied population**

| Parameter | ANAEMIC SUBJECTS (n = 36) | CONTROL (n = 36) | *P* value |
|---|---|---|---|
| | Median (Range) | Median (Range) | |
| IL-6 (pg/ml) | 20.80 (9.60 – 256.10) | 14.07 (8.50 – 40.00) | 0.001 |
| IL-10 (pg/ml) | 38.71 (29.60 – 80.70) | 37.21 (28.80 - 58.20) | 0.057 |
| IL-17 (pg/ml) | 64.05 (28.50 – 113.00) | 58.04 (30.20 – 108.90) | 0.474 |

Also, there were no statistically significant differences in the serum concentration of IL-10 and IL-17 between the anaemic subjects and controls. In anaemic subjects, statistically significant correlation were observed between Hb, haematocrit, RBC and IL-6 (r = - 0.34, r = - 0.39, r = - 0.41, respectively) as shown in Table 4.

**Table 4: Correlation of anaemic parameters with cytokines levels in anaemic subjects**

| Parameter | Hb r (*p* value) | Hct r (*p* value) | RBC r (*p* value) | MCV r (*p* value) | MCH r (*p* value) | MCHC r (*p* value) |
|---|---|---|---|---|---|---|
| **IL-6** | -0.34 (0.041)* | -0.39 (0.020)* | -0.41 (0.013)* | 0.32 (0.055) | 0.31 (0.067) | 0.15 (0.379) |
| **IL-10** | 0.06 (0.738) | -0.03 (0.862) | -0.22 (0.208) | 0.45 (0.006)* | 0.43 (0.009)* | 0.27 (0.109) |
| **IL-17** | -0.15 (0.930) | -0.07 (0.677) | -0.26 (0.132) | 0.41 (0.013)* | 0.38 (0.021)* | 0.22 (0.197) |

In the anaemic children, there was a significant association between IL-6 and IL-10 (r = 0.60, $p < 0.001$), between IL-6 and IL-17 (r = 0.46, $p$ = 0.005) and between IL-10 and IL-17 (r = 0.36, $p$ = 0.033) as presented in Table 5.

**Table 5: Correlation between cytokines in the anaemic children and controls**

| | ANAEMIC SUBJECTS r (*P* value) | CONTROLS r (*P* value) |
|---|---|---|
| 1L-6 vs IL-10 | 0.60 (< 0.001) | -0.08 (0.664) |
| IL-6 vs IL-17 | 0.46 (0.005) | 0.05 (0.757) |
| IL-10 vs IL-17 | 0.36 (0.033) | 0.04 (0.833) |

## DISCUSSION

Different studies have presented varying reports on cytokine levels and their roles in anaemia. In the present study, a strong inverse correlation was found between IL-6 and Hb, between IL-6 and RBC and between IL-6 and haematocrit in the subjects. This is in agreement with the findings of previous studies. IL-6 was found to be significantly elevated in anaemic patients with rheumatoid arthritis [23] and also in anaemic patients suffering from systemic lupus erythematosus. [24] Both studies reported that IL-6 may be

responsible for the development of anaemia in these patients. [23, 24] It was observed that IL-6 may cause anaemia through its ability to inhibit red cell progenitors, BFU-E and CFU-E. [23] A correlation between IL-6 and Hb levels was reported in geriatric syndrome of frailty. [25] Likewise, Ripley et al. (2005) found an inverse correlation between IL-6 and Hb levels. A similar observation was made in this present study. In experimental studies, administration of IL-6 in rats led to the development of anaemia. [26] Similarly, in human cancer subjects, recombinant IL-6 led to a rapid decrease in Hb concentration [27] while administration of IL-6 to rhesus monkeys correlated with a decline in PCV. [24] Many other studies have also shown that IL-6 drives anaemia, especially in inflammatory states by inhibiting red cell precursors and erythropoietin, [28] stimulating ferritin which causes iron retention in reticuloendothelial cells [26] and also by increasing the synthesis of hepcidin by liver cells. In turn, hepcidin inhibits iron absorption in the intestine and release of iron by macrophages. [29]

Also, IL-6 had a positive correlation with IL-10 and IL-17 in this study. This is in line with previous finding that IL-6 induces IL-17 expression and likewise IL-17 upregulates IL-6. [14] Just like IL-6, IL-17 also induces anaemia by inhibiting or suppressing growth of late-stage/mature erythroid progenitors (CFU-E). [30] It had been reported that IL-17 induced IL-8 and IL-6 expression in aplastic anaemia and normal controls. [31] The implication of this is that the anaemia may worsen as the circulating levels of these two cytokines rise. However, the findings in this present study did not suggest any relationship between IL-17 and RBC or Hb in the anaemic children but an inverse correlation was found between haematocrit, RBC and IL-17 in the non-anaemic controls. This implies that IL-17 may actually influence haematocrit and RBC levels and possibly decrease as RBC and haematocrit levels rise to normal values. A larger sample size possibly made up of more severe cases of anaemia may be required to detect this effect.

Although IL-10 and IL-17 were both elevated in the anaemic subjects when compared to the controls, the difference in the serum levels of both cytokines between these groups was not statistically significant. IL-10 is well-known for its anti-inflammatory properties and is thought to counteract anaemia by stimulating haematopoiesis and preventing destruction of red cell progenitors by pro-inflammatory cytokines. [12, 32] This may be the reason for the significant relationship observed between increased levels of IL-10 and IL-6 in this study.

There was no correlation between IL-10 and Hb levels in this study. Insufficient production of IL-10 has also been associated with severity of anaemia in malaria endemic areas [33] but this present study did not observe any significant difference in IL-10 levels between patients with mild, moderate and severe anaemia as well as malaria parasitaemia. The correlation between IL-10 and anaemia in this study may have been undetectable due to the small sample size however further studies involving more severe cases of anaemia may be helpful in confirming this observation.

Experimental studies have demonstrated a role for IL-10 in the suppression of IL-17 production in autoimmune arthritis patients. [34] IL-10 exerted significant inhibitory effect on IL-17-induced secretion of IL-1β and TNF-α by human macrophages *in vitro*. [35] IL-10 deficient mice macrophages produced significantly high levels of IL-17 *in vitro* whereas the wild-type macrophages secreted low levels of IL-17. [31] Unlike previous reports, the present study demonstrated a positive correlation between IL-10 and IL-17 in children with anaemia and this does not suggest a negative regulatory relationship between IL-10 and IL-17 in anaemia.

## CONCLUSION

This study indicated that IL-6 rather than IL-10 and IL-17 may be responsible for the pathogenesis of anaemia in children. Thus, IL-6 might be a potential marker for therapeutic monitoring in anaemia. A similar study utilizing larger samples, different age groups and a panel of cytokines should be conducted in the future to investigate the relationship between multiple cytokines and anaemia in specific diseases.

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**Conflicts Of Interest**

The authors do not have any conflicts of interest

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