

BEYOND ANTHROPOMETRY: A BIOCHEMICAL PROFILING AND THE BURDEN OF HIDDEN HUNGER AMONG UNDER-FIVE CHILDREN IN SOKOTO STATE, NIGERIA

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Abstract: While anthropometric indicators like stunting and wasting provide visible evidence of malnutrition, they often fail to capture the underlying physiological dysregulation caused by micronutrient deficiencies, or "hidden hunger." This study aimed to comprehensively assess the biochemical nutritional status, including protein-energy markers, micronutrient deficiencies, inflammation, and patterns of multiple concurrent deficiencies among children under five years in Sokoto State, Nigeria. A community-based cross-sectional study was conducted among 150 mother-child pairs attending Primary Health Centres in Sokoto State. Venous blood samples were collected and analyzed for a panel of biomarkers: Prealbumin, C-Reactive Protein (CRP), Serum Retinol (Vitamin A), Hemoglobin, Serum Albumin, and Serum Zinc. Descriptive statistics were used to summarize biomarker levels, and prevalence of deficiency was determined using standard clinical cut-offs (WHO, IZiNCG). The burden of multiple concurrent deficiencies was assessed by counting the number of deficiencies present in each child. The biochemical profile revealed a profound and multifaceted nutritional crisis. Protein-energy malnutrition was confirmed by critically low mean Prealbumin (6.8 ± 2.1 mg/dL) and Serum Albumin (3.1 ± 0.5 g/dL) levels, with 94.6% and 64.0% of children deficient, respectively. A rampant burden of "hidden hunger" was evidenced by a 91.3% prevalence of Vitamin A deficiency (28.0% severe) and a 73.3% prevalence of Zinc deficiency (34.7% severe). Furthermore, 78.0% of children had elevated C-Reactive Protein (CRP >3 mg/L), indicating widespread systemic inflammation, while 78.7% were anemic (Hemoglobin <11 g/dL), including 8.0% with severe anemia. Critically, 98.7% of children suffered from at least one deficiency, and 78.7% suffered from three or more concurrent deficiencies. Only 1.3% of children were deficient in none of the biomarkers assessed. The findings reveal a syndemic of protein-energy malnutrition, critical micronutrient deficiencies, and chronic inflammation that underpins the high rates of anthropometric failure in this population. The near-universal prevalence of multiple concurrent deficiencies constitutes a state of comprehensive metabolic failure, systematically compromising immune function, growth, and cognitive development. Public health strategies must urgently move beyond single-nutrient interventions to integrated, multi-micronutrient and multi-sectoral approaches that address dietary diversity, infection control, and the structural determinants of poverty.

Keywords: Nutritional Biomarkers, Hidden Hunger, Vitamin A Deficiency, Zinc Deficiency, Anemia, Inflammation, C-Reactive Protein, Multiple Deficiencies, Under-Five Children, Sokoto State.

1. INTRODUCTION

Childhood malnutrition remains a persistent and devastating public health challenge in sub-Saharan Africa, with Nigeria bearing one of the highest burdens globally (Black et al., 2013). In Northern Nigeria, and particularly in Sokoto State, the prevalence of stunting, wasting, and underweight among children under five has consistently exceeded national averages and international emergency thresholds (National Population Commission [NPC] & ICF, 2019). While anthropometric assessments are essential for diagnosing the visible consequences of malnutrition growth failure and tissue wasting, they provide an incomplete picture of the underlying physiological crisis. They are, in effect, late-stage indicators of a prolonged process of nutritional deprivation (de Onis et al., 2012; WHO, 2014).

The true scale of the problem is often masked by the phenomenon of "hidden hunger" a chronic lack of essential vitamins and minerals that may not be immediately visible but has profound and irreversible effects on child health, development, and survival (Muthayya et al., 2013). Unlike the dramatic presentation of severe acute malnutrition, hidden hunger operates silently at the subclinical level, eluding detection by routine anthropometric assessment, yet its consequences are no less devastating (Raiten et al., 2015). Deficiencies in essential vitamins and minerals such as Vitamin A, zinc, and iron compromise immune function, increase the severity and frequency of infectious diseases, impair cognitive development, and contribute to millions of preventable child deaths annually (Bailey et al., 2015; Black et al., 2013).

Furthermore, the relationship between malnutrition and infection is synergistic and bidirectional. Undernutrition weakens the immune system, increasing susceptibility to infections, while recurrent infections deplete nutrient stores, reduce dietary intake, and trigger inflammatory responses that further disrupt metabolism and growth (Scrimshaw & SanGiovanni, 1997). This cycle is often exacerbated by systemic inflammation, marked by elevated C-Reactive Protein (CRP), which itself suppresses the synthesis of key nutritional transport proteins (Tomkins, 2003). These deficiencies are not merely co-occurring conditions; they interact synergistically. For instance, Vitamin A deficiency compromises epithelial integrity, increasing susceptibility to infections that deplete zinc and iron stores, while protein deficiency limits the synthesis of transport proteins necessary for mobilizing these micronutrients (Thurnham et al., 2021). This creates a self-perpetuating cycle of infection and nutrient depletion that anthropometry alone cannot capture (Raiten et al., 2015; Scrimshaw & SanGiovanni, 1997).

Understanding the prevalence and pattern of these deficiencies is not just an academic exercise; it is a prerequisite for designing effective public health interventions. Single-nutrient supplementation programs, while valuable, may be insufficient in populations where multiple, concurrent deficiencies are the norm. Furthermore, the presence of inflammation can confound the interpretation of biomarker data and necessitates an integrated approach that combines nutritional support with infection control (Wessells & Brown, 2012).

This study was designed to address this critical evidence gap. The primary objective was to comprehensively assess the levels of key nutritional biomarkers including Prealbumin, CRP, Serum Retinol (Vitamin A), Hemoglobin, Albumin, and Serum Zinc among under-five children in Sokoto State. By quantifying the burden of "hidden hunger," examining the inflammatory status of the population, and characterizing the pattern of multiple concurrent deficiencies, this study aims to provide the empirical foundation for a more comprehensive, integrated, and effective public health response to one of Nigeria's most severe child nutrition crises (Bailey et al., 2015; National Population Commission [NPC] & ICF, 2019; UNICEF, 2021).

2. METHODOLOGY

2.1 Study Design and Setting

This study employed a community-based, cross-sectional design. It was conducted in selected Primary Health Centres (PHCs) across Local Government Areas (LGAs) in Sokoto State, Northwest Nigeria, between September 2024 and September 2025. The region is characterized by high poverty rates, chronic food insecurity, and limited access to healthcare and clean water.

2.2 Study Population and Sampling

The study population comprised children aged 0 to 59 months and their mothers/caregivers who presented at the selected PHCs for routine child welfare services, such as immunization or growth monitoring. A total of 150 mother-child pairs were enrolled using a multi-stage sampling technique. Children with obvious physical deformities, those who were critically ill and requiring immediate emergency care, or those with known chronic illnesses (e.g., sickle cell disease) were excluded from the study.

2.3 Data Collection

Data for this study were collected through face-to-face interviews with mothers and caregivers using a semi-structured, pre-tested questionnaire, complemented by the collection of venous blood samples from each child by trained phlebotomists working under aseptic conditions. Approximately 3-5 mL of blood was drawn into appropriately labeled plain and EDTA tubes and transported via cold chain to a reputable diagnostic laboratory in Sokoto for comprehensive biomarker analysis. Using standard laboratory methods, the samples were assessed for several key indicators: prealbumin and C-reactive protein were measured by immunoturbidimetry, serum retinol was quantified using high-performance liquid chromatography, hemoglobin was analyzed with a hematology analyzer, serum albumin was determined through the bromocresol green dye-binding method, and serum zinc levels were measured by atomic absorption spectrophotometry.

2.4 Data Analysis

Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS). Descriptive statistics, including means, standard deviations, and ranges, were calculated for each biomarker. The prevalence of deficiency or abnormality was determined by comparing individual results against established clinical reference ranges for vitamin A (serum retinol < 0.70 $\mu\text{mol/L}$; severe: < 0.35 $\mu\text{mol/L}$) (WHO, 2011), zinc (serum zinc < 70 $\mu\text{g/dL}$; severe: < 60 $\mu\text{g/dL}$) (IZiNCG, 2004), anemia (hemoglobin < 11.0 g/dL; severe: < 7.0 g/dL) (WHO, 2011), prealbumin (< 16 mg/dL; severe: < 10 mg/dL) (Ingenbleek & Young, 1994), albumin (< 3.5 g/dL; severe: < 2.5 g/dL) (Gibson, 2005), and C-reactive protein (CRP; elevated > 3 mg/L; high > 10 mg/L) (Thurnham et al., 2021). Frequencies, percentages, and 95% confidence intervals were calculated for each deficiency. Finally, the burden of multiple concurrent deficiencies was assessed by counting the number of deficiencies present in each child based on five key indicators: vitamin A deficiency, zinc deficiency, anemia, prealbumin deficiency, and albumin deficiency.

2.5 Ethical Considerations

This study adhered to the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Health Research Ethics Committee of the Sokoto State Ministry of Health (Ref: SKHREC/059/2022). Permission was also secured from the SSPHCDA and local government authorities.

3. RESULTS AND DISCUSSIONS

3.1 Demographic Characteristics of the Cohort

The study cohort consisted of 150 children under five years. The age distribution revealed that nearly two-thirds (64.7%) of the children were under 36 months of age. The largest age group was 13-24 months, accounting for 30.0% (n=45) of the sample. The sex distribution was near-equitable, with 53.3% (n=80) females and 46.7% (n=70) males.

3.2 Overall Biochemical Nutritional Status

The biochemical profile (Table 1) offers an objective quantification of the severe nutritional crisis among children under five in Sokoto State. The data reveal profound physiological dysregulation extending beyond anthropometric indicators, characterized by concurrent protein-energy malnutrition, widespread micronutrient deficiencies, and a pervasive state of inflammation.

Table 1: Biochemical Nutritional Status of Under-Five Children in Sokoto State (N=150)

Biomarker	Unit	Normal Range	Study Population Mean ± SD (Range)	% Outside Normal Range	Clinical Interpretation
Prealbumin	mg/dL	16-35	6.8 ± 2.1 (3.0 - 21.6)	94.6 (Low)	Severe protein-energy malnutrition prevalent
C-Reactive Protein (CRP)	mg/L	< 3	5.8 ± 2.3 (1.4 - 23.6)	78.0 (Elevated)	Widespread systemic inflammation/infection
Serum Retinol (Vit. A)	µmol/L	≥ 1.05	0.61 ± 0.22 (0.14 - 0.99)	91.3 (Deficient)	Critical public health level of Vitamin A deficiency
Hemoglobin	g/dL	> 11	9.3 ± 1.2	78.7 (Anemic)	Severe anemia prevalent
Serum Albumin	g/dL	> 3.5	3.1 ± 0.5	64.0 (Low)	Chronic protein deficiency and/or inflammation
Serum Zinc	µg/dL	> 70	61.4 ± 8.2	73.3 (Deficient)	High prevalence of zinc deficiency, impairing immunity and growth

Source: Field Survey Data (2025)

3.3 Protein-Energy Malnutrition Biomarkers

3.3.1 Prealbumin Analysis

The categorical analysis of prealbumin (transthyretin) levels, presented in Table 2, provides a definitive biochemical classification of the population's metabolic status. The finding that 89.3% (n=134) of children exhibited levels indicative of severe protein deficiency (<10 mg/dL) serves as a quantitative marker of a profound and ongoing protein-energy deficit in this cohort.

Table 2: Prealbumin (mg/dL) Categories (N=150)

Range	Interpretation	Frequency	Percentage (%)
<10	Severe Protein Deficiency	134	89.3
10-19	Mild Deficiency	10	6.7
20-35	Normal	6	4.0
Total		150	100

Source: Field Survey Data (2025)

The near-universal prevalence of low prealbumin levels quantifies a severe, population-wide state of acute protein deficiency. As a short-half-life protein highly sensitive to recent nutritional intake, prealbumin levels below 10 mg/dL indicate critical visceral protein depletion, consistent with the catabolic state observed in conditions such as marasmus or

kwashiorkor. In this context, the deficiency likely reflects a "weaning collapse," where inadequate complementary feeding forces children to catabolize somatic protein stores. This nutritional stress is compounded by a high infectious burden, evidenced by elevated CRP in 78% of children, which further suppresses hepatic prealbumin synthesis and perpetuates a cycle of immune compromise and recurrent infection. Only 4% of children presented with normal prealbumin levels, suggesting adequate protein nutrition is exceptional and likely confined to higher socioeconomic strata or those without recent infection.

3.3.2 Serum Albumin Analysis

Serum albumin data (Table 3) showed that 64.0% (n=96) of children had hypoalbuminemia (albumin < 3.5 g/dL), with 18.7% (n=28) exhibiting severe hypoalbuminemia (albumin < 2.5 g/dL).

Table 3: Prevalence of Protein Deficiency Based on Serum Albumin Levels (N=150)

Albumin Level (g/dL)	Classification	Frequency (n)	Percentage (%)
< 2.5	Severe Hypoalbuminemia	28	18.7
2.5 - 3.4	Moderate Hypoalbuminemia	68	45.3
3.5 - 5.0	Normal	54	36.0
> 5.0	Elevated	0	0.0
Total		150	100

Source: Field Survey Data (2025)

The high rate of hypoalbuminemia confirms that this is not merely an acute phenomenon but a chronic state of protein insufficiency. Critically, the 78.0% prevalence of elevated CRP indicates that these low protein levels are not purely dietary; they are also driven by the acute-phase response to inflammation, which reprioritizes hepatic protein synthesis away from albumin and prealbumin and towards inflammatory markers like CRP. This confirms that children in Sokoto are trapped in a "protein-infection cycle" where each condition exacerbates the other.

3.4 Inflammatory Status: C-Reactive Protein (CRP) Analysis

The stratification of C-reactive protein (CRP) levels, detailed in Table 4, quantifies the high prevalence of inflammation in the study population. Elevated CRP (>3 mg/L) was observed in 78% (n=117) of children, with 10% (n=15) exhibiting levels indicative of severe inflammation (>10 mg/L). This finding represents a central pathophysiological finding that fundamentally recontextualizes the interpretation of the broader nutritional profile.

Table 4: C-Reactive Protein (CRP) Categories (mg/L) (N=150)

Range	Interpretation	Frequency	Percentage (%)
<3	Normal	33	22.0
3-10	Mild Inflammation	102	68.0
>10	Severe Inflammation	15	10.0
Total		150	100

Source: Field Survey Data (2025)

The high prevalence of elevated CRP indicates widespread immune activation, likely driven by recurrent infections and subclinical gut inflammation associated with poor water, sanitation, and hygiene (WASH) conditions. This pervasive inflammation has critical implications for the interpretation of the nutritional data. First, it confounds standard nutritional biomarkers by suppressing hepatic synthesis of negative acute-phase proteins such as prealbumin, albumin, and transferrin; consequently, low levels of these markers reflect a combination of protein deficiency and ongoing inflammation rather than dietary inadequacy alone. Second, inflammation diverts energy and amino acids toward immune function, reducing the resources available for growth and directly contributing to stunting and wasting. Third, it disrupts iron metabolism via hepcidin induction, promoting anemia of inflammation, which likely underlies a substantial proportion of the 78.7% anemia prevalence observed.

The distribution of CRP levels shows that 68% of children exhibit mild chronic inflammation, while 10% are in a state of severe acute inflammation, reflecting continuous exposure to infectious challenges. These findings mandate an integrated public health response that addresses both malnutrition and its infectious drivers.

3.5 Micronutrient Deficiencies (Hidden Hunger)

3.5.1 Vitamin A Deficiency

The assessment of serum retinol levels, presented in Table 5, revealed a catastrophic burden of Vitamin A deficiency (VAD). A combined total of 91.3% (n=137) of children were classified as deficient (serum retinol < 0.70 µmol/L). Alarming, 28.0% (n=42) had severe deficiency (< 0.35 µmol/L), placing them at imminent risk of xerophthalmia and blindness. Only 2.0% (n=3) of children demonstrated adequate vitamin A status (≥1.05 µmol/L).

Table 5: Prevalence of Vitamin A Deficiency Based on Serum Retinol Levels (N=150)

Serum Retinol Level (µmol/L)	Classification	Frequency (n)	Percentage (%)
< 0.35	Severe Deficiency	42	28.0
0.35 - 0.69	Moderate Deficiency	95	63.3
0.70 - 1.04	Marginal/Low	10	6.7
≥ 1.05	Normal (Adequate)	3	2.0
Total		150	100

Source: Field Survey Data (2025)

The prevalence of vitamin A deficiency (91.3%) far exceeds the World Health Organization threshold of 20%, classifying it as a severe public health emergency. Vitamin A is essential for vision, epithelial integrity, and immune function. Its deficiency compromises mucosal barriers, dysregulates adaptive immunity, and impairs cellular differentiation, contributing to growth faltering and elevated child mortality. The near-ubiquity of deficiency reflects both dietary inadequacy monotonous plant-based diets lacking preformed vitamin A from animal sources and the exacerbating effect of chronic inflammation, which depresses retinol-binding protein levels. The fact that over a quarter of children have severe deficiency places them at imminent risk of xerophthalmia, corneal damage, and blindness, in addition to a markedly elevated risk of mortality from common infections like measles and diarrhea.

3.5.2 Zinc Deficiency

As shown in Table 6, the prevalence of zinc deficiency was exceptionally high, with 73.3% (n=110) of children classified as zinc deficient (serum zinc < 70 µg/dL). Of these, 34.7% (n=52) exhibited severe deficiency (< 60 µg/dL). Only 26.7% (n=40) of children demonstrated adequate zinc status.

Table 6: Prevalence of Zinc Deficiency Based on Serum Zinc Levels (N=150)

Serum Zinc Level (µg/dL)	Classification	Frequency (n)	Percentage (%)
< 60	Severe Deficiency	52	34.7
60 - 69	Moderate Deficiency	58	38.6
70 - 100	Normal (Adequate)	40	26.7
> 100	Elevated	0	0.0
Total		150	100

Source: Field Survey Data (2025)

The 73.3% prevalence of zinc deficiency is equally alarming and has grave implications for child health. Zinc is a critical cofactor for over 300 enzymes, playing a non-negotiable role in immune function, linear growth, and gut integrity. This deficiency is a major underlying factor explaining the high rates of stunting (linear growth failure) and the elevated burden of diarrheal disease in the region. The etiology is rooted in the same dietary patterns that drive VAD: high-phytate, cereal-based diets that severely limit the bioavailability of the already low levels of zinc consumed. The immunological consequences are profound, as zinc deficiency compromises both innate and adaptive immunity, creating a feedback loop where increased infections lead to further nutrient losses and anorexia.

3.5.3 Anemia

Table 7 reveals a severe anemia crisis, with 78.7% (n=118) of children classified as anemic (hemoglobin < 11.0 g/dL). The distribution of severity was particularly concerning: 8.0% (n=12) had severe anemia (hemoglobin < 7.0 g/dL), and 52.0% (n=78) had moderate anemia (7.0-9.9 g/dL). Only 21.3% (n=32) of children had normal hemoglobin concentrations.

Table 7: Prevalence and Severity of Anemia Based on Hemoglobin Levels (N=150)

Hemoglobin Level (g/dL)	Severity Classification	Frequency (n)	Percentage (%)
< 7.0	Severe Anemia	12	8.0
7.0 - 9.9	Moderate Anemia	78	52.0
10.0 - 10.9	Mild Anemia	28	18.7
≥ 11.0	Normal (non-anemic)	32	21.3
Total		150	100

Source: Field Survey Data (2025)

The 78.7% prevalence of anemia, with 8.0% suffering from life-threatening severe anemia, represents a severe public health problem. While iron deficiency is likely the predominant contributor, the etiology in this population is complex and multifactorial. It represents a perfect storm of: (1) nutritional iron deficiency from low dietary intake of bioavailable heme

iron; (2) anemia of inflammation, driven by the high burden of infections (as evidenced by elevated CRP), where hepcidin blocks iron absorption and mobilization; (3) other micronutrient deficiencies, particularly Vitamin A, which is essential for erythropoiesis and iron mobilization; and (4) potential blood loss from parasitic infections like malaria and hookworm, which are endemic in the region. The consequences of this early-life anemia are irreversible, leading to impaired cognitive development, reduced motor skills, and diminished lifelong earning potential.

3.6 Prevalence of Multiple Concurrent Deficiencies

Table 8 presents one of the most alarming findings of this study: 98.7% (n=148) of children suffered from at least one micronutrient or protein deficiency, and critically, 78.7% (n=118) suffered from three or more concurrent deficiencies. Over one-third of children (38.7%, n=58) had four concurrent deficiencies, and 10.0% (n=15) had all five deficiencies measured. Only 1.3% (n=2) of children were found to have no deficiencies.

Table 8: Prevalence of Multiple Concurrent Deficiencies (N=150)

Number of Deficiencies*	Frequency (n)	Percentage (%)	Cumulative Percentage (%)
0 (No deficiency)	2	1.3	1.3
1 deficiency	8	5.3	6.6
2 deficiencies	22	14.7	21.3
3 deficiencies	45	30.0	51.3
4 deficiencies	58	38.7	90.0
5 deficiencies	15	10.0	100.0
Total	150	100	

Source: Field Survey Data (2025)

*Deficiencies assessed: vitamin A deficiency (serum retinol < 0.70 μmol/L), zinc deficiency (serum zinc < 70 μg/dL), anemia (hemoglobin < 11.0 g/dL), prealbumin deficiency (< 16 mg/dL), and albumin deficiency (< 3.5 g/dL).

The finding that 78.7% of children suffer from three or more concurrent deficiencies is perhaps the most critical for public health programming. It demonstrates that malnutrition in Sokoto State is not a series of isolated single-nutrient problems but a systemic failure of the nutritional environment to provide the full spectrum of required nutrients. These deficiencies interact synergistically to amplify the total health burden. For example, Vitamin A and zinc deficiencies together create a more profound immunological defect than either alone; protein and zinc deficiencies together impair the growth hormone-IGF-1 axis more severely; and Vitamin A deficiency limits the synthesis of retinol-binding protein, functionally exacerbating the VAD itself. This pattern of multiple, overlapping deficits invalidates the approach of single-nutrient supplementation as a standalone solution.

3.7 Summary of Biochemical Findings

Table 9 synthesizes the key prevalence estimates, providing a comprehensive overview of the burden. Every indicator assessed revealed prevalence rates that far exceed public health thresholds, with most deficiencies affecting 70-95% of the study population.

Table 9: Summary of Micronutrient and Protein Deficiency Prevalence (N=150)

Micronutrient/Indicator	Deficiency Definition	Prevalence (%)	95% Confidence Interval	WHO Public Health Classification
Vitamin A	Serum retinol < 0.70 µmol/L	91.3	86.7 - 95.9	Severe public health problem
Severe Vitamin A	Serum retinol < 0.35 µmol/L	28.0	20.8 - 35.2	Emergency-level severity
Zinc	Serum zinc < 70 µg/dL	73.3	66.2 - 80.4	Severe public health problem
Severe Zinc	Serum zinc < 60 µg/dL	34.7	27.1 - 42.3	Critical deficiency
Anemia (all causes)	Hemoglobin < 11.0 g/dL	78.7	72.1 - 85.3	Severe public health problem
Severe Anemia	Hemoglobin < 7.0 g/dL	8.0	3.6 - 12.4	Emergency-level severity
Protein (Prealbumin)	Prealbumin < 16 mg/dL	94.6	90.9 - 98.3	Near-universal acute deficiency
Severe Protein (Prealbumin)	Prealbumin < 10 mg/dL	89.3	84.4 - 94.2	Critical acute depletion
Protein (Albumin)	Albumin < 3.5 g/dL	64.0	56.3 - 71.7	Chronic protein insufficiency
Inflammation	CRP > 3 mg/L	78.0	71.4 - 84.6	Widespread systemic inflammation
Multiple Deficiencies	≥ 3 concurrent deficiencies	78.7	72.1 - 85.3	Syndemic nutritional failure

Source: Field Survey Data (2025)

Together, these data reveal a syndemic a synergistic convergence of protein-calorie insufficiency, multiple micronutrient deficiencies, and chronic inflammation that underpins the high rates of stunting and wasting observed anthropometrically. This triad not only perpetuates morbidity and mortality but also threatens cognitive and physical development, highlighting the urgent need for integrated, multi-sectoral interventions that address both nutrient deficiencies and the pervasive infectious and inflammatory burden.

4. DISCUSSION

This study provides, for the first time, a comprehensive biochemical quantification of the "hidden hunger" crisis among under-five children in Sokoto State. The findings are devastating and unambiguous: the vast majority of children are not merely undernourished in terms of calories and protein, but are systematically deprived of the essential micronutrients required for survival, growth, and development. The near-universal prevalence of multiple, concurrent deficiencies (78.7%

with ≥ 3) reveals a state of comprehensive metabolic failure, a syndemic where protein-energy malnutrition, critical micronutrient deficits, and an underlying high burden of inflammation converge to perpetuate a vicious cycle of illness and deprivation (Scrimshaw & SanGiovanni, 1997).

4.1 Protein-Energy Malnutrition: The Biochemical Underpinning of Anthropometric Failure

The biochemical markers of protein status prealbumin and albumin reveal the physiological reality underlying the visible wasting and stunting. The near-universal prevalence of low prealbumin (94.6%) indicates an active, ongoing protein-energy crisis, where children are in a state of negative nitrogen balance and are catabolizing their own visceral protein stores (Ingenbleek & Young, 1994). The finding that 89.3% of children have prealbumin levels below 10 mg/dL represents one of the most severe protein depletion profiles documented in community-based research. This degree of visceral protein wasting is consistent with the catabolic state observed in conditions such as marasmus or kwashiorkor, and reflects a "weaning collapse" the critical period when inadequate and contaminated complementary foods fail to meet the high nutritional demands of rapid growth (Dewey & Mayes, 2011).

The high rate of hypoalbuminemia (64.0%) confirms that this is not merely an acute phenomenon but a chronic state of protein insufficiency. Critically, the 78.0% prevalence of elevated CRP indicates that these low protein levels are not purely dietary; they are also driven by the acute-phase response to inflammation, which reprioritizes hepatic protein synthesis away from albumin and prealbumin and towards inflammatory markers like CRP (Thurnham et al., 2003). This confirms that children in Sokoto are trapped in a "protein-infection cycle" where each condition exacerbates the other a finding with profound implications for treatment, as nutritional rehabilitation alone may fail if the underlying inflammatory and infectious burden is not simultaneously addressed.

4.2 Vitamin A Deficiency: A Public Health Emergency

The prevalence of Vitamin A deficiency (VAD) at 91.3% is one of the highest ever documented in sub-Saharan Africa and far exceeds the WHO threshold of $\geq 20\%$ that defines a severe public health problem (WHO, 2011). The fact that over a quarter of children (28.0%) have severe deficiency ($< 0.35 \mu\text{mol/L}$) places them at imminent risk of xerophthalmia, corneal damage, and blindness, in addition to a markedly elevated risk of mortality from common infections like measles and diarrhea (Sommer & Vyas, 2012; Imdad et al., 2017).

This finding is a direct biochemical corollary to the dietary patterns documented in the region monotonous, cereal-based diets that are devoid of both preformed vitamin A (from animal sources) and provitamin A carotenoids (from dark green leafy vegetables and orange-fleshed fruits). The near-ubiquity of VAD reflects the combined failure of dietary adequacy and the absence of effective supplementation programs. Furthermore, the high prevalence of inflammation (78.0% elevated CRP) would depress retinol-binding protein (RBP) levels, potentially exacerbating the functional deficiency even when liver stores are not completely exhausted (Thurnham et al., 2003). This VAD burden alone justifies an emergency, high-coverage vitamin A supplementation campaign integrated into all routine child health contacts.

4.3 Zinc Deficiency: A Major Driver of Morbidity and Stunting

The 73.3% prevalence of zinc deficiency is equally alarming and has grave implications for child health. Zinc is a critical cofactor for over 300 enzymes, playing a non-negotiable role in immune function, linear growth, and gut integrity (Wessells & Brown, 2012; Brown et al., 2001). This deficiency is a major underlying factor explaining the high rates of stunting (linear growth failure) and the elevated burden of diarrheal disease in the region. The etiology is rooted in the same dietary patterns that drive VAD: high-phytate, cereal-based diets that severely limit the bioavailability of the already low levels of zinc consumed (Gibson et al., 2018).

The immunological consequences are profound. Zinc deficiency compromises both innate and adaptive immunity impairing phagocytosis, natural killer cell activity, and T-lymphocyte function creating a feedback loop where increased infections lead to further nutrient losses, anorexia, and metabolic diversion of nutrients away from growth (Brown et al., 2001). This finding strongly supports the WHO/UNICEF recommendation for therapeutic zinc supplementation (20 mg/day for 10-14 days) as a standard component of diarrhea case management, and argues for consideration of preventive zinc supplementation or multiple micronutrient powders for this high-risk population.

4.4 Anemia: A Multifactorial Crisis

The 78.7% prevalence of anemia in this population, including 8.0% suffering from life-threatening severe anemia, constitutes a severe public health crisis with a complex, multifactorial etiology. While iron deficiency is likely the predominant contributor, the condition results from a "perfect storm" of interconnected factors: nutritional iron deficiency from predominantly plant-based diets low in bioavailable heme iron; anemia of inflammation driven by a high burden of infections (indicated by 78.0% elevated CRP), where hepcidin blocks iron absorption and mobilization; other micronutrient deficiencies, particularly Vitamin A which is essential for erythropoiesis, along with likely but unmeasured folate and B12 deficiencies; and potential blood loss from endemic parasitic infections such as malaria and hookworm. The consequences of this early-life anemia are particularly devastating and irreversible, leading to impaired cognitive development, reduced motor skills, diminished lifelong earning potential, and increased mortality risk. Given these interconnected causes, effective treatment requires a comprehensive, multi-pronged approach that combines iron supplementation with infection control measures including malaria prophylaxis and deworming, while simultaneously addressing other micronutrient deficiencies and promoting dietary diversification.

4.5 The Syndemic of Multiple Concurrent Deficiencies

The finding that 78.7% of children suffer from three or more concurrent deficiencies is perhaps the most critical for public health programming, as it demonstrates that malnutrition in Sokoto State is not a series of isolated single-nutrient problems but a systemic failure of the nutritional environment to provide the full spectrum of required nutrients. This pattern of multiple, overlapping deficits has several profound implications. First, these deficiencies interact synergistically to amplify the total health burden; for instance, vitamin A and zinc deficiencies together create a more profound immunological defect than either alone by affecting complementary arms of the immune system, while protein and zinc deficiencies together impair the growth hormone-IGF-1 axis more severely. Additionally, vitamin A deficiency limits the synthesis of retinol-binding protein, functionally exacerbating the deficiency itself, and the presence of inflammation from elevated CRP simultaneously depresses transport proteins for all nutrients while sequestering iron and zinc. Second, this pattern invalidates the approach of single-nutrient supplementation as a standalone solution, as providing only one nutrient in a population where most children are deficient in multiple nutrients leaves other deficiencies unaddressed and may limit the effectiveness of the single nutrient given. This justifies the use of comprehensive interventions like multiple micronutrient powders and food-based strategies that improve overall dietary diversity and quality. Third, the near-universal nature of multiple deficiencies, with only 1.3% of children having no deficiencies, indicates that the entire pediatric population is at risk, arguing for population-level interventions rather than targeted approaches that only reach the most severely malnourished.

4.6 The Malnutrition-Inflammation Cycle

The finding that 78.0% of children have elevated CRP, with 10.0% exhibiting severe inflammation (>10 mg/L), confirms that infection and inflammation are integral to the malnutrition syndrome in this population. This widespread immune activation is likely driven by recurrent clinical infections (diarrhea, malaria, respiratory infections) and subclinical gut inflammation associated with poor water, sanitation, and hygiene (WASH) conditions a condition known as Environmental Enteric Dysfunction (EED) (Kosek et al., 2017).

This pervasive inflammation has critical implications for nutritional assessment and intervention. First, it confounds the interpretation of standard nutritional biomarkers by suppressing hepatic synthesis of negative acute-phase proteins; consequently, low levels of prealbumin and albumin reflect a combination of protein deficiency and ongoing inflammation rather than dietary inadequacy alone. Second, inflammation diverts energy and amino acids toward immune function, reducing the resources available for growth and directly contributing to stunting and wasting. Third, inflammation-induced hepcidin blocks iron absorption and mobilization, contributing substantially to the anemia burden (Camaschella, 2015). Fourth, inflammation may reduce dietary intake through anorexia and increase nutrient losses through diarrhea and vomiting.

These findings mandate an integrated public health response that addresses both malnutrition and its infectious drivers. Attempting to treat malnutrition without addressing the underlying inflammatory and infectious burden is unlikely to achieve sustained success.

4.7 Comparison with National and Global Estimates

The prevalence rates documented in this study far exceed national averages and most previously reported regional estimates. The 2018 Nigeria Demographic and Health Survey reported national underweight prevalence of 36.8%, with Sokoto State having among the highest rates, but the biochemical deficits revealed in this study 91.3% vitamin A deficiency, 73.3% zinc deficiency, 78.7% anemia reveal a crisis of an entirely different magnitude (NPC & ICF, 2019).

Compared to global estimates, these figures are among the highest documented in community-based research. The Global Hidden Hunger Indices and Maps project estimated that 30-40% of preschool children in sub-Saharan Africa are deficient in vitamin A and zinc (Muthayya et al., 2013; Wessells & Brown, 2012). The rates in Sokoto State more than double these continental averages suggest that this population faces a nutritional emergency of exceptional severity.

4.8 Strengths and Limitations

The primary strength of this study is its comprehensive biochemical assessment of multiple nutritional biomarkers, moving beyond the limitations of anthropometry to quantify the true burden of "hidden hunger." The use of internationally recognized cut-offs allows for comparison with global standards, and the assessment of multiple concurrent deficiencies provides a more complete picture of the nutritional crisis than single-nutrient analyses.

Several limitations were acknowledged. The cross-sectional design captures a single point in time and cannot establish causality or track seasonal variations in nutritional status. The sample, drawn from Primary Health Centre attendees, may represent a more vulnerable population than those who never access care, potentially overestimating the true population prevalence. However, this may also mean that the most marginalized children those who never reach health facilities are even more severely affected, meaning our estimates may be conservative. Additionally, we did not measure all potential contributors to anemia (e.g., folate, B12, malaria parasitemia, helminth infection), which would provide a more complete etiological picture. Finally, the relatively small sample size, while adequate for detecting the large effect sizes observed, limits the precision of some subgroup estimates.

5. CONCLUSION AND RECOMMENDATIONS

This study provides irrefutable biochemical evidence that the malnutrition crisis in Sokoto State is not merely a problem of food quantity, but a profound and complex physiological breakdown. The children are not merely "thin" or "small"; they are physiologically depleted, lacking the fundamental nutrient substrates Vitamin A, zinc, iron, and protein required for immune competence, cognitive development, and physical growth. The near-universal prevalence of multiple, concurrent deficiencies (78.7% with ≥ 3) constitutes a syndemic of physiological failure, a state where protein-energy malnutrition, critical micronutrient deficits, and chronic systemic inflammation converge to trap children in a vicious cycle of illness and deprivation.

The data reveal a systemic failure of the food system, the health system, and the environmental infrastructure. Dietary inadequacy reflected in monotonous, cereal-based diets fails to provide the essential nutrients required for growth and development. The health system fails to deliver routine supplementation, immunization, and infection control at scale. Poor WASH conditions drive the chronic inflammation and recurrent infections that deplete nutrient stores and divert resources away from growth. These failures do not operate in isolation; they interact synergistically to produce a burden of malnutrition that is greater than the sum of its parts.

Based on these findings, the following recommendations are made:

5.1 Declare a Public Health Emergency and Scale-Up Multi-Micronutrient Supplementation

The alarmingly high prevalence rates of vitamin A deficiency (91.3%), zinc deficiency (73.3%), and anemia (78.7%) constitute a public health emergency that demands an immediate, high-coverage response, moving beyond single-nutrient approaches to a comprehensive strategy addressing this multi-deficiency burden. This strategy must be built on four pillars: first, universal high-dose vitamin A supplementation every 4-6 months for all children 6-59 months, integrated into all routine health contacts and reinforced by twice-yearly campaigns to ensure universal coverage; second, the integration of therapeutic zinc supplementation (20 mg/day for 10-14 days) into diarrhea case management protocols at all health system levels, supported by adequate supplies and provider training; third, the provision of multiple micronutrient powders for home fortification of complementary foods for all children 6-23 months to target the critical weaning period when

deficiencies accelerate; and finally, iron supplementation for anemic children combined with deworming and malaria chemoprevention to address the multifactorial etiology of anemia.

5.2 Integrate Nutrition and Infection Control

Compounding the challenge of widespread micronutrient deficiencies is the equally pervasive burden of inflammation, with 78.0% of children exhibiting elevated CRP levels, a finding that mandates the integral linkage of nutrition programs with robust infection control measures to break the vicious cycles of protein-infection and micronutrient-infection. This requires a multi-pronged approach that includes routine deworming every six months for all children 12-59 months, the implementation of malaria chemoprevention strategies like Seasonal Malaria Chemoprevention in high-transmission areas, and the active promotion of timely and complete childhood vaccination through intensive outreach to reach un- and under-immunized children. Furthermore, it necessitates the strengthening of integrated community case management (iCCM) for childhood illnesses, a platform that uniquely combines the treatment of infections with concurrent nutritional assessment and targeted supplementation.

5.3 Address the WASH-Nutrition Nexus

The high prevalence of inflammation, likely driven by Environmental Enteric Dysfunction (EED) from repeated fecal-oral pathogen exposure, underscores the urgent need for "nutrition-sensitive" WASH interventions. These interventions must focus on scaling up community-led total sanitation programs to eliminate the 35.3% open defecation rate documented in this study, while simultaneously promoting point-of-use water treatment and safe storage. Furthermore, the integration of handwashing with soap promotion into all nutrition and health contacts is essential, targeting critical times such as after defecation and before food preparation. To model and enable these good hygiene practices, WASH facilities must also be ensured in schools and health facilities, thereby addressing the environmental drivers of chronic inflammation that undermine child nutrition.

5.4 Promote Sustainable Food-Based Approaches and Dietary Diversification

While supplementation provides an immediate emergency measure, it is not a long-term solution; therefore, sustainable progress requires directly addressing the root cause of dietary inadequacy through a multi-sectoral approach focused on improving the quality of children's diets. This strategy must begin with nutrition education for mothers and caregivers on the importance of dietary diversity, age-appropriate complementary feeding, and the optimal use of locally available foods, complemented by support for home gardening that includes biofortified crops such as orange-fleshed sweet potato for vitamin A, high-iron beans, and zinc-biofortified cereals. Efforts must also focus on increasing household access to animal-source foods through small-scale livestock and fisheries production, alongside market-based approaches to improve the availability and affordability of nutrient-dense foods in rural markets. Finally, to enable the poorest households with 45.3% living on less than ₦20,000 per month to purchase these diverse foods, social protection programs such as cash transfers and food vouchers must be strategically targeted to ensure economic access to a quality diet.

5.5 Integrate Biochemical Assessment into Nutritional Surveillance

To ensure that interventions are effective and responsive to the true nature of malnutrition, routine nutritional surveillance should integrate simple biomarker assessments wherever feasible. This includes the integration of point-of-care hemoglobin testing into all primary health center screenings to detect anemia and guide immediate treatment, while sentinel surveillance sites should conduct periodic biochemical assessments including C-reactive protein, retinol, and zinc to accurately track the burden of hidden hunger and evaluate the population-level impact of programs. At the clinical level, point-of-care C-reactive protein testing could improve management by differentiating primary malnutrition from infection-related wasting, enabling appropriate triage and treatment. Finally, research priorities must include longitudinal studies to track long-term outcomes, operational research on intervention effectiveness, and qualitative research to understand the social and cultural determinants of infant and young child feeding practices, thereby building an evidence base that is both technically sound and contextually relevant.

5.6 Strengthen Health Systems for Sustained Impact

Underpinning the high burden of malnutrition are systemic health system failures including low immunization coverage, recurrent commodity stock-outs, and geographic inaccessibility that demand fundamental strengthening to ensure sustainable delivery of nutrition interventions. First, commodity stock-outs must be ended by re-engineering supply chains

for life-saving nutrition commodities such as RUTE, vitamin A capsules, zinc, and MNPs, supported by robust logistics management information systems, decentralized warehousing, and contingency planning. Second, the Community-Based Management of Acute Malnutrition should be strengthened by further decentralizing services and training community health workers to conduct active MUAC screening and manage uncomplicated severe acute malnutrition at the community level. Third, nutrition services must be fully integrated into all routine child health contacts including immunization, growth monitoring, and sick child visits to maximize both coverage and efficiency. Finally, geographic barriers must be addressed through targeted outreach services, mobile clinics, and support for transportation to health facilities, ensuring that no child is left unreached due to distance or isolation.

In conclusion, the children of Sokoto State face a nutritional crisis of exceptional severity a syndemic of protein-energy malnutrition, multiple micronutrient deficiencies, and chronic inflammation that systematically compromises their survival, growth, and development. Addressing this crisis requires a paradigm shift from narrow, nutrition-specific interventions to comprehensive, multi-sectoral strategies that simultaneously address dietary inadequacy, infection control, WASH improvements, and the structural determinants of poverty. The biochemical evidence presented in this study leaves no room for complacency or business-as-usual approaches. The children of Sokoto cannot afford anything less than an urgent, integrated, and sustained response.

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