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(RESEARCH ARTICLE)

The Sokoto Syndemic: A call for a paradigm shift in tackling childhood malnutrition in Nigeria

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Abstract

Childhood malnutrition and infection are known to interact synergistically, yet the biochemical evidence for this bidirectional relationship in high-burden settings remains inadequately characterized. To examine the association between protein-energy malnutrition, micronutrient deficiencies, and systemic inflammation (C-reactive protein, CRP) among under-five children in Sokoto State, Nigeria, and to characterize the malnutrition-inflammation syndemic. A community-based cross-sectional study was conducted among 150 children aged 6–59 months attending primary health centers in Sokoto State. Anthropometric measurements (weight, height, MUAC) were collected, and venous blood samples were analyzed for prealbumin, CRP, serum retinol (vitamin A), hemoglobin, albumin, and zinc. Children were classified by nutritional status using WHO growth standards and MUAC cut-offs. Statistical analyses included independent t-tests, Pearson correlation, chi-square tests for trend, and logistic regression. The prevalence of elevated CRP (>3 mg/L) was 78.0% (n=117), with 10.0% (n=15) exhibiting severe inflammation (>10 mg/L). Malnourished children (MUAC <12.5 cm) had significantly higher CRP levels than well-nourished children (6.7 ± 2.4 vs. 4.3 ± 1.9 mg/L, $p < 0.001$). A strong dose-response relationship was observed: CRP elevation increased from 51.1% in normal children to 78.7% in MAM and 92.9% in SAM ($p < 0.001$). CRP showed significant negative correlations with all anthropometric indices ($r = -0.41$ to -0.53, $p < 0.01$) and with prealbumin ($r = -0.58$, $p < 0.001$). Among children with SAM, 92.9% had elevated CRP and 100% had concurrent protein-energy and micronutrient deficiencies. Logistic regression revealed that children with SAM had 12.2 times higher odds of elevated CRP compared to normal children (AOR = 12.19, 95% CI: 3.38–43.95, $p < 0.001$). The malnutrition-inflammation syndemic is highly prevalent among under-five children in Sokoto State, with nearly all severely malnourished children exhibiting systemic inflammation. The bidirectional relationship between malnutrition and inflammation creates a self-perpetuating cycle that exacerbates morbidity and mortality. These findings mandate integrated interventions that simultaneously address nutritional rehabilitation and infection control.

Keywords: Malnutrition; Inflammation; C-Reactive Protein; Syndemic; Protein-Energy Malnutrition; Under-Five Children; Sokoto State; Nigeria

1. Introduction

Childhood malnutrition remains one of the most formidable public health challenges globally, contributing to approximately 45% of all deaths among children under five years of age (Black et al., 2013). In Nigeria, the burden is disproportionately concentrated in the northern regions, with Sokoto State consistently reporting some of the worst

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nutritional indicators in the country (National Population Commission & ICF, 2019). While the anthropometric consequences of malnutrition stunting, wasting, and underweight have been extensively documented, the underlying pathophysiological mechanisms remain incompletely understood.

The relationship between malnutrition and infection is classically described as synergistic and bidirectional (Scrimshaw & SanGiovanni, 1997). Undernutrition compromises immune function, increasing susceptibility to infectious diseases, while infections exacerbate nutritional deficiencies through anorexia, malabsorption, and increased metabolic demands. This cyclical interaction has been recognized for decades, yet the biochemical evidence for this syndemic in contemporary high-burden settings remains limited (Raiten et al., 2015).

Systemic inflammation, as measured by C-reactive protein (CRP), serves as a critical biomarker of this interaction. CRP is an acute-phase protein synthesized by the liver in response to pro-inflammatory cytokines, particularly interleukin-6 (Pepys & Hirschfield, 2003). Elevated CRP indicates ongoing infection or inflammation and has been associated with poor outcomes in malnourished children (Briend et al., 2021). Importantly, inflammation confounds the interpretation of nutritional biomarkers such as albumin, prealbumin, and ferritin, which are suppressed during the acute-phase response (Thurnham et al., 2003).

The concept of a "syndemic" the synergistic interaction of co-occurring diseases that together produce a greater burden than the sum of their individual effects has gained increasing recognition in global health (Singer & Clair, 2003). In the context of child malnutrition, the syndemic framework posits that undernutrition and infection do not merely co-exist but interact in ways that amplify their combined pathological effects (Mertens et al., 2023). Understanding this syndemic is essential for designing effective interventions that address both dimensions simultaneously.

This study was designed to address critical evidence gaps regarding the malnutrition-inflammation syndemic in Sokoto State. The primary objectives were: (1) to quantify the prevalence and severity of systemic inflammation (elevated CRP) among under-five children; (2) to examine the association between nutritional status (anthropometric and biochemical) and CRP levels; (3) to characterize the dose-response relationship between malnutrition severity and inflammation; and (4) to provide biochemical evidence for the syndemic interaction between malnutrition and inflammation.

2. Materials and methods

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 twelve Local Government Areas (LGAs) in Sokoto State, Northwest Nigeria, between September 2024 and September 2025. Sokoto State 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 6 to 59 months and their mothers/caregivers who presented at the selected PHCs for routine child welfare services. A total of 150 mother-child pairs were enrolled using a multi-stage sampling technique. Children with physical deformities that could impede accurate anthropometric measurement, those who were critically ill requiring immediate emergency care, or those with known chronic illnesses (e.g., sickle cell disease) were excluded.

2.3. Data Collection

2.3.1. Anthropometric Measurements

Weight was measured to the nearest 0.1 kg using a calibrated Seca electronic scale with children wearing minimal clothing. Recumbent length was measured for children under 24 months using a calibrated infantometer; while standing height was measured for older children using a portable stadiometer, both recorded to the nearest 0.1 cm. Mid-upper arm circumference (MUAC) was measured at the midpoint of the left upper arm using a non-stretchable tape and recorded to the nearest 0.1 cm.

2.3.2. Blood Sample Collection and Biomarker Analysis

Venous blood samples (approximately 3-5 mL) were collected from each child by a trained phlebotomist under aseptic conditions. Samples were transported in a cold chain to a diagnostic laboratory for analysis. Prealbumin and C-reactive protein (CRP) were measured by high-sensitivity immunoturbidimetry. Serum retinol (vitamin A) was quantified by high-performance liquid chromatography (HPLC). Hemoglobin was analyzed using a hematology analyzer. Serum albumin was determined by the bromocresol green (BCG) dye-binding method. Serum zinc was measured by atomic absorption spectrophotometry.

2.4. Nutritional Status Classification

Anthropometric indices (Weight-for-Age Z-score [WAZ], Height-for-Age Z-score [HAZ], and Weight-for-Height Z-score [WHZ]) were calculated using WHO Anthro software (version 3.2.2). Nutritional status was classified according to WHO Child Growth Standards (World Health Organization, 2006). MUAC was categorized using standard cut-offs: normal (>12.5 cm), moderate acute malnutrition (MAM: 11.5–12.5 cm), and severe acute malnutrition (SAM: <11.5 cm) (World Health Organization & UNICEF, 2009).

2.5. Biomarker Thresholds

Biomarker deficiencies were defined using internationally recognized thresholds: prealbumin deficiency (<16 mg/dL; severe <10 mg/dL) (Ingenbleek & Young, 1994), elevated CRP (>3 mg/L; severe >10 mg/L) (Pearson et al., 2003), vitamin A deficiency (<0.70 μmol/L; severe <0.35 μmol/L) (World Health Organization, 2011a), anemia (hemoglobin <11.0 g/dL; severe <7.0 g/dL) (World Health Organization, 2011b), hypoalbuminemia (<3.5 g/dL; severe <2.5 g/dL) (Gibson, 2005), and zinc deficiency (<70 μg/dL; severe <60 μg/dL) (International Zinc Nutrition Consultative Group, 2004).

2.6. Statistical Analysis

Data were entered and analyzed using IBM SPSS Statistics version 26. Descriptive statistics (frequencies, percentages, means, standard deviations) were used to summarize all variables. Comparisons of mean biomarker levels across nutritional categories were conducted using independent t-tests and one-way ANOVA. Pearson correlation coefficients were calculated to examine relationships between continuous variables. Chi-square tests for trend were used to assess dose-response relationships. Binary logistic regression was performed to quantify the association between MUAC category and elevated CRP, with results reported as adjusted odds ratios (AOR) with 95% confidence intervals (CI). Statistical significance was set at $p < 0.05$, and all tests were two-tailed.

2.7. 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). Written informed consent was obtained from all mothers or caregivers. Children identified with severe acute malnutrition or severe illness were immediately referred for appropriate care.

3. Results

3.1. Demographic Characteristics of the Cohort

The study cohort consisted of 150 children under five years. The mean age was 30.2 ± 16.8 months (range: 6–59 months). The age distribution showed that 64.7% of children were under 36 months, with the largest age group being 13–24 months (30.0%). The sex distribution was near-equitable: 53.3% female (n=80) and 46.7% male (n=70).

3.2. Prevalence and Severity of Systemic Inflammation

The overall prevalence of elevated CRP (>3 mg/L) was 78.0% (n=117). Of these, 68.0% (n=102) exhibited mild-to-moderate elevation (3–10 mg/L), and 10.0% (n=15) exhibited severe inflammation (>10 mg/L). The mean CRP level was 5.8 ± 2.3 mg/L (range: 1.4–23.6 mg/L).

Table 1 presents the distribution of CRP categories across the cohort.

Table 1 **C-**Reactive Protein (CRP) Categories (N=150)

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

3.3. Association Between Nutritional Status and CRP Levels

3.3.1. Comparison by MUAC Classification

Children classified as malnourished by MUAC (<12.5 cm) had significantly higher CRP levels than well-nourished children (6.7 ± 2.4 vs. 4.3 ± 1.9 mg/L, mean difference 2.4 mg/L, t = -5.12, p < 0.001). Table 2 presents the comparison of mean CRP levels by nutritional status.

Table 2 Comparison of Mean CRP Levels by MUAC Classification

Nutritional Status	N	Mean CRP ± SD (mg/L)	Mean Difference	t-statistic	p-value
Malnourished (<12.5 cm)	103	6.7 ± 2.4	-2.4	-5.12	<0.001
Normal (>12.5 cm)	47	4.3 ± 1.9			

3.3.2. Comparison by Stunting Status

Stunted children (HAZ < -2SD) had significantly higher CRP levels than non-stunted children (6.3 ± 2.5 vs. 5.2 ± 2.1 mg/L, mean difference 1.1 mg/L, t = -2.89, p = 0.004).

3.3.3. Dose-Response Relationship

A striking dose-response relationship was observed between the severity of acute malnutrition (MUAC category) and the prevalence of elevated CRP. The proportion of children with elevated CRP increased progressively from normal (51.1%) to MAM (78.7%) to SAM (92.9%) (χ^2 trend = 19.34, p < 0.001).

3.4. Correlation Between CRP and Anthropometric Indices

Pearson correlation analysis revealed significant negative correlations between CRP and all anthropometric indices, indicating that higher inflammation is associated with poorer nutritional status (Table 3).

Table 3 Pearson Correlation Coefficients Between CRP and Anthropometric Indices

Anthropometric Index	Correlation Coefficient (r)	p-value
MUAC (cm)	-0.53	<0.001
Weight (kg)	-0.45	<0.001
Weight-for-Age Z-score (WAZ)	-0.48	<0.001
Height-for-Age Z-score (HAZ)	-0.41	<0.001
Weight-for-Height Z-score (WHZ)	-0.44	<0.001

3.5. Correlation Between CRP and Nutritional Biomarkers

CRP showed significant negative correlations with prealbumin (r = -0.58, p < 0.001) and albumin (r = -0.49, p < 0.001), consistent with the known suppression of negative acute-phase proteins during inflammation. CRP also showed negative correlations with serum retinol (r = -0.41, p < 0.001) and hemoglobin (r = -0.38, p < 0.001).

Table 4 presents the correlation matrix.

Table 4 Pearson Correlation Coefficients Between CRP and Nutritional Biomarkers

Biomarker	**Correlation Coefficient (r)**	**p-value**
Prealbumin	-0.58	<0.001
Serum Albumin	-0.49	<0.001
Serum Retinol (Vitamin A)	-0.41	<0.001
Hemoglobin	-0.38	<0.001
Serum Zinc	-0.35	<0.001

3.6. Prevalence of Inflammation by Biochemical Deficiency Status

Children with biochemical deficiencies had significantly higher rates of elevated CRP compared to those without deficiencies. Table 5 presents these associations.

Table 5 Prevalence of Elevated CRP by Biochemical Deficiency Status

Biochemical Deficiency	**% with Elevated CRP in Deficient Group**	**% with Elevated CRP in Non-Deficient Group**	**p-value (χ^2)**
Severe Prealbumin Deficiency (<10 mg/dL)	84.3%	28.6%	<0.001
Vitamin A Deficiency (<0.70 µmol/L)	82.5%	27.3%	<0.001
Hypoalbuminemia (<3.5 g/dL)	89.6%	57.4%	<0.001
Anemia (<11.0 g/dL)	86.4%	40.6%	<0.001
Zinc Deficiency (<70 µg/dL)	83.6%	62.5%	0.008

3.7. The Syndemic: Co-occurrence of Inflammation and Multiple Deficiencies

Among children with severe acute malnutrition (SAM), 92.9% (n=39) had elevated CRP, and 100% had three or more concurrent biochemical deficiencies. Among children with elevated CRP, the mean number of concurrent biochemical deficiencies was 3.4 ± 1.2, compared to 1.8 ± 1.1 among children with normal CRP ($t = 7.23$, $p < 0.001$).

Table 6 illustrates the syndemic burden.

Table 6 Co-occurrence of Inflammation and Multiple Deficiencies by MUAC Category

MUAC Category	**% with Elevated CRP**	**Mean Number of Deficiencies (out of 5)**
Normal (>12.5 cm)	51.1%	2.0 ± 1.2
MAM (11.5–12.5 cm)	78.7%	3.6 ± 1.0
SAM (<11.5 cm)	92.9%	4.7 ± 0.5
p-value (trend)	<0.001	<0.001

Note: Deficiencies assessed: vitamin A deficiency, zinc deficiency, anemia, prealbumin deficiency, hypoalbuminemia.

3.8. Logistic Regression: MUAC Category as Predictor of Elevated CRP

Binary logistic regression analysis quantified the dramatically increased risk of elevated CRP associated with worsening anthropometric status. After adjusting for potential confounders (age, sex, and household dietary diversity), children with SAM had 12.2 times higher odds of elevated CRP compared to normal children (AOR = 12.19, 95% CI: 3.38–43.95, $p < 0.001$). Children with MAM had 3.5 times higher odds (AOR = 3.54, 95% CI: 1.58–7.92, $p = 0.002$).

Table 7 presents the logistic regression results.

Table 7 Logistic Regression Analysis – MUAC Category as Predictor of Elevated CRP

MUAC Category	Adjusted Odds Ratio (AOR)	95% Confidence Interval	p-value
Normal (>12.5 cm)	1.00 (Reference)	-	-
MAM (11.5–12.5 cm)	3.54	1.58–7.92	0.002
SAM (<11.5 cm)	12.19	3.38–43.95	<0.001

Model adjusted for child age, sex, and household dietary diversity. Nagelkerke R^2 = 0.32.

4. Discussion

This study provides the first comprehensive biochemical evidence for the malnutrition-inflammation syndemic among under-five children in Sokoto State, Nigeria. The findings reveal that systemic inflammation is not merely a co-occurring condition but an integral component of the malnutrition syndrome, affecting the vast majority of children and exhibiting a striking dose-response relationship with the severity of nutritional depletion.

4.1. The Pervasive Burden of Systemic Inflammation

The finding that 78.0% of children have elevated CRP, with 10.0% exhibiting severe inflammation, represents one of the highest documented prevalence rates of systemic inflammation in a pediatric population. This prevalence far exceeds estimates from other sub-Saharan African settings (Namaste et al., 2017; Suchdev et al., 2016) and indicates that children in Sokoto State are living in a state of near-constant immune activation.

This pervasive inflammation has multiple etiologies. The poor WASH conditions documented in this population including 35.3% open defecation create a high burden of fecal-oral pathogen exposure, leading to repeated clinical and subclinical infections (Humphrey, 2009). The high prevalence of diarrhea (22.7%) and respiratory infections (32.0%) in the two weeks preceding the survey confirms that children are experiencing frequent infectious episodes. Additionally, environmental enteric dysfunction (EED), a subclinical condition characterized by chronic intestinal inflammation and villous atrophy, is highly prevalent in settings with poor sanitation and contributes to sustained low-grade inflammation (Kosek et al., 2014).

4.2. The Bidirectional Relationship: Malnutrition as Both Cause and Consequence of Inflammation

The data reveal a bidirectional relationship that is central to the syndemic. First, malnutrition drives inflammation. Children with SAM had 12.2 times higher odds of elevated CRP compared to normal children, and the dose-response relationship shows that as nutritional status worsens, the prevalence and severity of inflammation increase correspondingly. This likely reflects the immune-compromised state of malnourished children, who are more susceptible to infections that trigger inflammatory responses (Rytter et al., 2014).

Second, inflammation drives malnutrition. The significant negative correlations between CRP and prealbumin (r = -0.58) and albumin (r = -0.49) demonstrate that the acute-phase response suppresses the synthesis of these critical proteins, contributing to protein-energy malnutrition independently of dietary intake (Thurnham et al., 2003). Inflammation also disrupts iron metabolism via hepcidin induction, contributing to anemia, and depresses retinol-binding protein, exacerbating vitamin A deficiency (Nemeth & Ganz, 2021). This creates a vicious cycle where inflammation-induced nutrient depletion worsens malnutrition, which in turn increases susceptibility to further infections and inflammation.

4.3. The Syndemic: Synergistic Interactions Amplifying the Burden

The finding that among children with SAM, 92.9% had elevated CRP and 100% had three or more concurrent deficiencies illustrates the syndemic nature of the crisis. The convergence of protein-energy malnutrition, multiple micronutrient deficiencies, and systemic inflammation represents more than the sum of its parts. These conditions interact synergistically: inflammation suppresses appetite, increases catabolism, and diverts nutrients away from growth toward immune function (Tomkins, 2003); protein-energy malnutrition impairs immune function, reducing the capacity to clear infections and prolonging inflammatory responses (Bhutta et al., 2013); and micronutrient deficiencies particularly zinc and vitamin A compromise both innate and adaptive immunity, further amplifying susceptibility (Wessells & Brown, 2012).

This syndemic explains the exceptionally high morbidity and mortality documented in this population. Children with concurrent malnutrition and infection have mortality risks that are multiplicative rather than additive (Victora et al., 2008).

4.4. Inflammation as a Confounder in Nutritional Assessment

The high prevalence of inflammation has critical implications for the interpretation of nutritional biomarkers. Standard clinical thresholds for biomarkers such as albumin, prealbumin, and ferritin were developed in populations with low inflammatory burden. In populations like Sokoto, where 78% of children have elevated CRP, these biomarkers will systematically underestimate true nutritional status because inflammation suppresses their synthesis (Thurnham et al., 2003; Thurnham et al., 2021).

This finding underscores the importance of adjusting nutritional biomarkers for inflammation using methods such as those developed by the BRINDA (Biomarkers Reflecting Inflammation and Nutritional Determinants of Anemia) project (Namaste et al., 2017). Failure to account for inflammation leads to misclassification of nutritional status and may result in inappropriate treatment decisions.

4.5. Programmatic Implications: Integrated Interventions

The syndemic framework provides a compelling rationale for integrated interventions that simultaneously address malnutrition and its infectious drivers. Nutritional rehabilitation alone is insufficient if the underlying inflammatory burden is not addressed. Conversely, infection treatment alone will have limited impact if underlying malnutrition is not corrected (Bhutta et al., 2017).

Table 8 outlines the recommended integrated approach.

Table 8 Recommended Integrated Interventions for the Malnutrition-Inflammation Syndemic

Component	**Specific Interventions**
Nutritional Rehabilitation	RUTF for SAM; multiple micronutrient powders; vitamin A supplementation; zinc for diarrhea
Infection Control	Deworming (6-monthly); malaria chemoprevention; complete immunization; antibiotics for infections
WASH Improvements	Sanitation facilities to eliminate open defecation; safe water storage; handwashing with soap
Health System Strengthening	Integrated CMAM programs; point-of-care CRP testing; supply chain for RUTF and supplements

4.6. Strengths and Limitations

The primary strength of this study is its comprehensive biochemical assessment, which provides objective evidence for the malnutrition-inflammation syndemic. The inclusion of multiple biomarkers allows for a nuanced understanding of the interactions between different aspects of nutritional status and inflammation.

Several limitations should be acknowledged. The cross-sectional design captures associations at a single point in time and cannot establish causality or temporal sequence. The sample was drawn from PHC attendees, which may introduce selection bias; however, this also ensures that the findings are relevant to the population that engages with the health system. The relatively small sample size limited the precision of some subgroup estimates, though the large effect sizes observed provide confidence in the main findings. Finally, we did not directly assess infectious etiologies (e.g., malaria parasitemia, bacterial infections), which would provide a more complete understanding of the drivers of inflammation.

5. Conclusions

This study provides unequivocal biochemical evidence that the malnutrition-inflammation syndemic is highly prevalent among under-five children in Sokoto State. Systemic inflammation affects more than three-quarters of children and exhibits a strong dose-response relationship with the severity of malnutrition. The convergence of protein-energy malnutrition, multiple micronutrient deficiencies, and systemic inflammation creates a self-perpetuating cycle that amplifies morbidity and mortality.

These findings have profound implications for clinical practice and public health programming. Nutritional assessment must account for the confounding effects of inflammation on biomarker interpretation. Nutritional rehabilitation must be integrated with infection control measures. And interventions must address the environmental drivers of inflammation, particularly poor WASH conditions, which perpetuate the syndemic.

Addressing the malnutrition-inflammation syndemic requires a paradigm shift from narrow, single-focus interventions to comprehensive, integrated approaches that simultaneously target nutrition, infection, and their environmental determinants. The children of Sokoto State cannot afford anything less.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical approval for this study was obtained from the Health Research Ethics Committee of the Sokoto State Ministry of Health (Reference Number: SKHREC/059/2022). All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments.

Statement of informed consent

Written informed consent was obtained from the parent or legal guardian of each child who participated in the study. The purpose, procedures, potential risks, and benefits of the study were thoroughly explained to each caregiver in their local language (Hausa or Fulfulde) before any data or sample collection. For illiterate participants, informed consent was documented by a thumbprint in the presence of an impartial witness. Participants were assured of their right to withdraw from the study at any time without any consequences. All data were anonymized to ensure confidentiality.

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