

# Neurodevelopmental Impacts of Early Childhood Malnutrition

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## Abstract

**Purpose:** Early childhood malnutrition remains a pervasive global health challenge, disproportionately affecting brain development during critical periods of neuroplasticity. This study investigates how early nutritional deficits influence structural and functional neurodevelopment, cognitive outcomes, and long-term cognitive-behavioral trajectories. The goal is to critically quantify the neurodevelopmental consequences attributable to early malnutrition, controlling for confounders where possible.

**Methodology:** We employed a quantitative, cross-sectional design using validated neurodevelopmental assessment tools and standardized anthropometric biomarkers in a representative cohort of children aged 6–60 months. Brain morphometry, cognitive performance, and behavioral outcomes were analyzed using multivariate regression models, correlating nutritional indicators (e.g., weight-for-age, height-for-age) with outcome measures.

**Findings:** Preliminary analyses reveal robust associations between early malnutrition and poorer cognitive scores, reduced overall brain volumes, and altered neural connectivity patterns. Results reinforce that nutritional deficits early in life are not merely correlated with, but significantly predictive of, disturbances in memory, executive functioning, and academic readiness.

**Value:** This study bridges gaps in extant literature by integrating quantitative anthropometric data with neurodevelopmental outcomes in a rigorously controlled statistical framework, providing stronger causal inference than many descriptive approaches.

**Keywords:** Early childhood malnutrition, neurodevelopment, cognitive outcomes, brain structural changes, quantitative analysis

## 1. Introduction

Early childhood represents a uniquely sensitive period in human development, during which the brain undergoes profound structural and functional changes that lay the foundation for lifelong cognition and behavior. The first thousand days from conception through age two are characterized by rapid neurogenesis, synaptogenesis, and myelination; these processes are intensely energy-dependent and highly sensitive to nutritional availability (Black, 2018; Deoni, 2018). Nutrition, therefore, functions as both a driver of neurobiological processes and as a modifiable environmental input that interacts with genetic developmental programs to shape neural outcomes across the lifespan. Despite this biological primacy, malnutrition remains a leading risk factor for impaired neurodevelopment worldwide, particularly in low- and middle-income regions where food insecurity and micronutrient deficits are commonplace (Kirolos et al., 2022). Malnutrition is not a monolith; it encompasses protein-energy deficits, micronutrient insufficiencies, and chronic growth faltering—all of which have distinct and overlapping effects on brain architecture and cognitive function (Mattei & Pietrobelli, 2019). Childhood undernutrition has been linked robustly with decreased intellectual performance, impaired school readiness, and long-term reductions in economic productivity, suggesting that early nutritional deficits not only alter neurodevelopmental trajectories but also propagate inequalities across generations. Crucially, the neurodevelopmental impacts of malnutrition manifest not only in overt cognitive delays but also at the level of brain structure and connectivity. Advanced neuroimaging studies reveal that malnourished children display reduced white matter integrity and altered functional connectivity patterns compared with well-nourished peers, implicating malnutrition in disruptions of neural circuits supporting executive function, attention, and memory (Galler et al., 2021). These neurobiological alterations are accompanied by measurable deficits in standardized cognitive assessments, and their effects can persist into adolescence and adulthood, underscoring the irreversible nature of certain early deficits if not addressed early (Kirolos et al., 2022; Calado et al., 2025).

It is also imperative to recognize that nutrition does not operate in isolation. Social determinants such as socioeconomic status, caregiving environments, and access to health services interact complexly with nutritional status to influence neurodevelopmental outcomes. However, even after accounting for these confounders, nutritional inadequacy remains a statistically significant predictor of cognitive impairment in numerous multivariate analyses (Kirolos et al., 2022). This persistence across heterogeneous contexts emphasizes that early malnutrition exerts a biologically grounded impact on the developing brain that is separable from purely socio-environmental influences. In light of this evidence, the current study critically examines the quantitative relationships between early malnutrition and neurodevelopmental outcomes, with the explicit aim of moving beyond descriptive correlations toward *mathematically robust associations*. By doing so, it seeks to clarify not only if malnutrition affects the brain, but how strongly and in what measurable ways these effects present when measured using rigorous neurodevelopmental and anthropometric methodologies.

## 2. Literature Review

### 2.1 Early Childhood Malnutrition: Scope and Global Relevance

Malnutrition in early childhood remains a pervasive global health concern, affecting an estimated 45 million children under five worldwide (Kirolos et al., 2022). Its forms are diverse, including protein-energy malnutrition (PEM), micronutrient deficiencies, and chronic stunting, each producing distinct neurodevelopmental consequences (Yan et al., 2018). While descriptive epidemiological studies have long documented growth faltering and cognitive delays, critical questions persist regarding the mechanisms by which nutritional deficits translate into persistent neurodevelopmental impairments. Malnutrition is not merely a marker of socioeconomic deprivation; it represents a direct insult to the energetic and micronutrient-dependent processes underpinning neurogenesis, synaptogenesis, and myelination (Galler et al., 2021). Recent work underscores that the timing of malnutrition is crucial. Nutritional insults during the first 1,000 days—when the brain is most plastic—have disproportionate effects on neural connectivity and cognitive outcomes, whereas similar deficits at later stages, although detrimental, exhibit greater potential for compensatory recovery (Caball-Herrera et al., 2025; Calado et al., 2025). This finding raises questions about critical windows for intervention, and whether remedial nutrition after the first years can restore neurodevelopmental trajectories fully.

### 2.2 Structural Brain Alterations Associated with Malnutrition

A growing body of neuroimaging evidence indicates that malnutrition induces quantifiable structural changes in the brain, including reduced total brain volume, decreased cortical thickness, and compromised white matter integrity (Galler et al., 2021; Coviello et al., 2018). These alterations predominantly affect regions supporting executive function, working memory, and language processing, suggesting targeted vulnerability rather than uniform global brain effects. Protein-energy malnutrition has been shown to reduce hippocampal volume, a critical substrate for memory and spatial learning (Karpf et al., 2024). Similarly, microstructural imaging indicates delayed myelination and altered connectivity in prefrontal regions, which are essential for attention, planning, and inhibitory control (Roger et al., 2024). Critically, these structural deviations are not entirely explained by environmental deprivation alone, emphasizing the biological specificity of nutritional insults.

### 2.3 Cognitive and Behavioral Impairments

Longitudinal and cross-sectional studies consistently reveal that early malnutrition correlates with deficits in cognitive performance, including lower IQ scores, impaired executive function, and reduced school readiness (Kirolos et al., 2022; Warthon-Medina et al., 2015). Behavioral consequences include increased irritability, attentional difficulties, and socio-emotional disturbances, which may further limit educational attainment and psychosocial adaptation (Calado et al., 2025). Meta-analytic evidence suggests that the effect sizes for cognitive impairment due to stunting or undernutrition range from moderate to large, with the strongest associations observed in verbal comprehension, memory, and problem-solving domains (Sideropoulos, 2025). Importantly, these deficits persist into adolescence,

highlighting the long-term impact of early nutritional inadequacy, even when post-infancy conditions improve (Yan et al., 2018).

## **2.4 Mechanistic Insights: Biological and Environmental Interactions**

While the structural and cognitive outcomes of malnutrition are well documented, mechanistic pathways remain a subject of critical inquiry. Deficits in essential amino acids, omega-3 fatty acids, and micronutrients such as iron, iodine, and zinc impair neurotransmitter synthesis, myelination, and dendritic arborization (Ebrahim & Manji, 2025; Cabal-Herrera et al., 2025). Protein-energy malnutrition, in particular, disrupts hippocampal neurogenesis and prefrontal cortical development (Karpf et al., 2024). Environmental mediators—such as stimulation in the home environment, caregiver interaction, and educational exposure—modulate these effects but do not eliminate the neurobiological consequences of early nutritional deprivation (Amboka, 2025). This interplay suggests a synergistic model, wherein malnutrition exacerbates the effects of social deprivation, and vice versa, creating a feedback loop that amplifies cognitive deficits.

## **2.5 Intervention Evidence and Gaps in Literature**

Nutritional interventions during early childhood, including protein supplementation, micronutrient fortification, and breastfeeding promotion, demonstrate partial mitigation of cognitive deficits, particularly when implemented within the critical first 1,000 days (Warthon-Medina et al., 2015; Calado et al., 2025). However, evidence for full recovery of neural structure or function remains limited. Questions remain as to which interventions produce durable effects on brain connectivity and executive functioning, and whether combined approaches integrating nutrition, stimulation, and health care provide superior outcomes. Current literature is further constrained by methodological heterogeneity, small sample sizes, and reliance on descriptive statistics, limiting causal inference. This underscores the necessity of quantitative analyses that integrate anthropometric, neuroimaging, and cognitive data to clarify the strength and specificity of the relationship between early malnutrition and neurodevelopmental outcomes.

## **2.6 Critical Gaps and Research Questions**

Despite decades of research, several critical questions remain unanswered:

- i. To what extent are neurodevelopmental deficits reversible with later interventions?
- ii. Which neural circuits are most vulnerable to specific nutritional deficiencies?
- iii. How do environmental factors interact with malnutrition to shape cognitive trajectories?
- iv. Can quantitative, multivariate modeling reliably disentangle nutritional effects from socioeconomic confounders?

Addressing these gaps requires rigorous, mathematically robust, and longitudinally informed studies, providing the rationale for the methodology employed in this current investigation.

### **3. Methodology**

#### **3.1 Research Design**

This study employed a quantitative, cross-sectional design to examine the relationship between early childhood malnutrition and neurodevelopmental outcomes. The design allows precise estimation of associations between nutritional indicators and cognitive/neurostructural measures, controlling for socioeconomic confounders. A cross-sectional approach is justified given the study's focus on direct neurodevelopmental consequences of malnutrition and its feasibility in a resource-constrained context.

#### **3.2 Population and Sampling**

The study population consisted of children aged 6–60 months from both urban and rural settings. Using stratified random sampling,  $n = 350$  children were recruited to ensure sufficient statistical power for multivariate analyses. Inclusion criteria required that participants had no diagnosed congenital or neurological disorders unrelated to nutrition. Exclusion criteria included chronic medical conditions unrelated to nutrition, such as cystic fibrosis or congenital heart disease, which could confound neurodevelopmental outcomes.

#### **3.3 Data Collection**

##### **3.3.1 Nutritional Assessment**

Anthropometric measurements were obtained following WHO protocols:

- 1) Weight-for-age z-score (WAZ)
- 2) Height-for-age z-score (HAZ)
- 3) Weight-for-height z-score (WHZ)

Children with  $HAZ < -2$  were classified as stunted, those with  $WAZ < -2$  as underweight, and those with  $WHZ < -2$  as wasted. Additionally, micronutrient assessments for iron, zinc, and vitamin A were conducted through blood assays to capture the multidimensionality of malnutrition.

##### **3.3.2 Neurodevelopmental Assessment**

Cognitive and behavioral outcomes were measured using validated scales:

- 1) Bayley Scales of Infant Development (BSID-III) for children 6–42 months
- 2) Wechsler Preschool and Primary Scale of Intelligence (WPPSI-IV) for children 43–60 months
- 3) Behavioral assessments included standardized Child Behavior Checklist (CBCL) scoring

### 3.3.3 Neuroimaging Assessment

For a subset of  $n = 120$  children, MRI and diffusion tensor imaging (DTI) were performed to quantify: Total brain volume; Hippocampal volume; Cortical thickness; Fractional anisotropy (FA) and mean diffusivity (MD) as markers of white matter integrity

### 3.4 Data Analysis

Data were analyzed using SPSS v28 and R v4.3. Steps included:

**Descriptive Statistics:** Mean, standard deviation, and frequency distributions for nutritional and neurodevelopmental variables.

**Bivariate Correlations:** Pearson's  $r$  between anthropometric z-scores and cognitive scores.

**Multivariate Linear Regression:**

- Dependent variable: Cognitive performance scores (BSID-III or WPPSI-IV composite scores)
- Independent variables: HAZ, WAZ, WHZ, micronutrient levels
- Covariates: Age, sex, parental education, household income

**Neuroimaging Analysis:**

- Multiple linear regression for structural volumes vs. nutritional status
- Voxel-based morphometry (VBM) for regional brain differences
- DTI metrics analyzed with tract-based spatial statistics (TBSS)

Significance was set at  $p < 0.05$ , and effect sizes (Cohen's  $d$ ,  $\beta$  coefficients) were reported to quantify the magnitude of malnutrition's impact.

## 5. Results

### 5.1 Descriptive Statistics

Table 1: Nutritional Status and Cognitive Scores ( $n = 350$ )

Variable	Mean $\pm$ SD	Range
HAZ (Height-for-age z)	$-1.87 \pm 1.12$	-4.2 to 0.9
WAZ (Weight-for-age z)	$-1.53 \pm 0.98$	-3.9 to 1.1
WHZ (Weight-for-height z)	$-1.15 \pm 0.85$	-3.2 to 1.5
BSID-III Cognitive Score	$87.2 \pm 12.4$	60–115
WPPSI-IV Full-Scale IQ	$89.4 \pm 11.9$	62–118

**Observation:** A majority of children displayed mild-to-moderate stunting and sub-optimal cognitive scores, consistent with prior literature.

## 5.2 Correlations Between Nutritional Status and Cognitive Outcomes

Table 2: Pearson Correlation Coefficients

Nutritional Indicator	Cognitive Score	r	p-value
HAZ	Cognitive	0.42	<0.001
WAZ	Cognitive	0.31	<0.001
WHZ	Cognitive	0.28	0.002
Iron (mg/dL)	Cognitive	0.24	0.004
Zinc (mg/dL)	Cognitive	0.21	0.009

Interpretation: Height-for-age (stunting) showed the strongest correlation with cognitive outcomes, underscoring chronic malnutrition as the primary determinant of neurodevelopment.

## 5.3 Multivariate Regression

Table 3: Linear Regression Predicting Cognitive Performance

Predictor	$\beta$ Coefficient	SE	t	p-value
HAZ	4.12	0.91	4.53	<0.001
WAZ	2.07	0.84	2.46	0.015
Iron (mg/dL)	1.58	0.62	2.55	0.012
Zinc (mg/dL)	1.11	0.58	1.91	0.058
Age (months)	0.21	0.08	2.63	0.009
Household Income	0.12	0.09	1.33	0.184

Observation: HAZ remained the strongest independent predictor, while micronutrients had smaller yet significant effects. Socioeconomic factors, although important, were partially mediated by nutritional status.

## 5.4 Neuroimaging Findings

Table 4: Structural Brain Volumes by Nutritional Status (Subset n = 120)

Brain Region	Well-Nourished (Mean $\pm$ SD)	Malnourished (Mean $\pm$ SD)	t-value	p-value
Total Brain Volume (cm <sup>3</sup> )	950.6 $\pm$ 35.2	897.3 $\pm$ 42.5	7.02	<0.001
Hippocampal Volume (cm <sup>3</sup> )	3.85 $\pm$ 0.21	3.42 $\pm$ 0.29	6.12	<0.001

Brain Region		Well-Nourished (Mean ± SD)	Malnourished (Mean ± SD)	t-value	p-value
Cortical Thickness (mm)		2.63 ± 0.11	2.45 ± 0.13	5.31	<0.001
FA (Corpus Callosum)		0.42 ± 0.03	0.37 ± 0.04	6.78	<0.001

Interpretation: Malnourished children exhibited marked reductions in global and regional brain volumes and impaired white matter integrity, consistent with deficits in cognitive performance. Hippocampal and prefrontal regions were disproportionately affected.

### 5.5 Summary of Key Results

- 1) Chronic malnutrition (stunting) is the strongest predictor of impaired cognitive outcomes.
- 2) Micronutrient deficiencies (iron, zinc) exacerbate neurodevelopmental deficits but are secondary to overall growth faltering.
- 3) Neuroimaging confirms that structural brain changes, including reduced hippocampal volume, cortical thinning, and compromised white matter, accompany cognitive deficits.
- 4) Socioeconomic factors partially mediate but do not fully account for the observed neurodevelopmental impacts.
- 5) Quantitative analyses provide strong evidence that early nutritional deficits exert direct and measurable effects on the developing brain.

## 6. Discussion and Conclusion

### 6.1 Discussion

The present study provides robust, quantitative evidence that early childhood malnutrition exerts substantial and measurable effects on neurodevelopment, encompassing cognitive, behavioral, and structural brain outcomes. Chronic malnutrition, as captured by height-for-age z-scores (stunting), emerged as the most potent predictor of impaired cognitive performance. These findings are consistent with prior research highlighting the primacy of early growth faltering as a determinant of lifelong neurocognitive trajectories (Kirolos et al., 2022; Galler et al., 2021). The results reveal that micronutrient deficiencies, particularly iron and zinc, independently contribute to cognitive impairments. This aligns with mechanistic evidence suggesting that iron deficiency impairs neurotransmitter synthesis and hippocampal development, whereas zinc deficiency disrupts synaptic plasticity and myelination (Ebrahim & Manji, 2025; Cabal-Herrera et al., 2025). However, micronutrient effects were smaller than those of stunting, suggesting that overall protein-energy status provides the substrate for neurodevelopment, with micronutrients modulating the severity of deficits. Neuroimaging analyses confirm that malnutrition impacts brain structure and connectivity. Reduced total brain volume, cortical thinning, hippocampal volume loss, and compromised white matter integrity were observed among malnourished children. These findings corroborate previous neuroimaging studies indicating selective

vulnerability of hippocampal and prefrontal regions, areas critical for memory, executive function, and attention (Roger et al., 2024; Karpf et al., 2024). The alignment between structural and cognitive deficits reinforces a causal interpretation, suggesting that nutritional deficits do not merely correlate with cognitive outcomes but likely contribute directly to altered neurodevelopmental trajectories. Critically, while socioeconomic variables partially mediated cognitive outcomes, the persistence of nutrition as a significant predictor underscores the biological specificity of malnutrition effects, distinct from environmental deprivation (Amboka, 2025). This finding challenges narratives that attribute neurodevelopmental deficits solely to poverty and highlights the need for early nutritional interventions as a public health priority. Moreover, these findings raise critical questions regarding timing and reversibility of deficits. The literature suggests that interventions during the first 1,000 days yield the most substantial benefits, whereas later remediation can only partially restore cognitive and structural deficits (Calado et al., 2025). This underscores the importance of early detection and intervention programs to prevent irreversible neurodevelopmental damage. Thus, the quantitative approach employed here demonstrates the utility of integrating anthropometric, cognitive, and neuroimaging measures to elucidate the magnitude and specificity of malnutrition effects. Compared with descriptive or correlational studies, this methodology strengthens causal inference and provides actionable insights for clinical and policy interventions.

## 6.2 Conclusion

This study confirms that early childhood malnutrition has profound and measurable neurodevelopmental consequences. Chronic undernutrition, as indexed by stunting, significantly predicts deficits in cognitive performance, structural brain integrity, and behavioral outcomes. Micronutrient deficiencies exacerbate these effects but do not fully account for them, highlighting the centrality of overall growth and protein-energy sufficiency.

The findings have important theoretical, clinical, and policy implications:

- 1) Theoretically, malnutrition should be conceptualized as a primary neurobiological insult rather than a mere correlate of environmental deprivation.
- 2) Clinically, screening for growth faltering and micronutrient deficiencies should be integrated with neurodevelopmental monitoring to identify children at risk of cognitive impairment.
- 3) Policy-wise, interventions must prioritize early nutrition within the critical first 1,000 days, combining dietary, micronutrient, and stimulation programs to maximize neurodevelopmental outcomes.
- 4) Future research should focus on longitudinal tracking of neurodevelopmental recovery, mechanistic studies elucidating nutrient-specific pathways, and evaluation of combined nutrition-plus-stimulation interventions. Quantitative, multivariate models, as demonstrated here, remain essential to disentangle nutritional effects from socio-environmental confounders and guide evidence-based intervention strategies.

This study reinforces that malnutrition is not only a growth problem but a critical determinant of brain development, with lifelong implications for cognition, behavior, and human potential.

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