Severe Pediatric Malnutrition and Its Impact on Brain Development: A Comprehensive Review

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Abstract

Background: Severe pediatric malnutrition remains one of the most pressing global health challenges, particularly in low- and middle-income countries, where it contributes significantly to morbidity, mortality, and long-term disability. Beyond the immediate consequences of stunting, wasting, and increased susceptibility to infections, malnutrition exerts profound and lasting effects on the developing brain. The human brain undergoes rapid growth and structural reorganization in early childhood, a period during which nutritional adequacy is critical for neuronal proliferation, myelination, and synaptic connectivity. Deficiencies in macronutrients and micronutrients, especially proteins, essential fatty acids, iron, zinc, iodine, and vitamins, can disrupt these processes, resulting in cognitive, motor, and behavioral impairments that often persist into adolescence and adulthood.

The aim of this review is to synthesize current evidence regarding the impact of severe pediatric malnutrition on brain development from a comprehensive pediatric perspective. By examining epidemiological trends, biological mechanisms, neurodevelopmental outcomes, and structural brain alterations, this article seeks to highlight the multidimensional burden of malnutrition on child neurodevelopment. A particular emphasis is placed on the interaction between malnutrition, infections, and socioeconomic determinants, which together amplify the risk of impaired cognitive function and poor educational attainment.

Recent advances in neuroimaging and neuropsychology provide compelling evidence of reduced cortical volumes, altered white matter integrity, and delayed neural maturation among malnourished children. These structural changes correlate with deficits in attention, memory, executive functioning, and socio-emotional regulation. Furthermore, studies underscore the importance of the first 1,000 days of life as a critical window during which nutritional interventions have the highest potential to mitigate long-term neurological damage. Rehabilitation strategies that combine therapeutic feeding, micronutrient supplementation, and early cognitive stimulation show promise, although challenges in implementation remain in resource-limited settings.

In conclusion, severe pediatric malnutrition is not only a nutritional disorder but also a profound threat to optimal brain development and future human capital. A deeper understanding of its mechanisms and outcomes underscores the urgent need for integrated interventions that address both biological and social determinants of malnutrition. By framing malnutrition as a pediatric neurodevelopmental crisis, this review emphasizes the importance of early prevention, timely management, and sustained policy efforts to break the intergenerational cycle of poverty, undernutrition, and impaired cognitive potential.

Keywords: *-Severe Malnutrition, Brain Development, Pediatric*

Introduction

Severe pediatric malnutrition is a critical global health issue, affecting millions of children under five years of age, particularly in regions burdened by poverty, food insecurity, and limited access to healthcare. According to UNICEF and WHO estimates, approximately 45 million children worldwide suffer from wasting, while 149 million experience stunting, conditions that are strongly associated with increased risk of mortality and long-term disability [1]. While the life-threatening consequences of malnutrition, such as infections, electrolyte imbalances, and impaired immunity, are well recognized, its less visible but equally devastating impact on brain development is often underappreciated.

The developing brain is particularly vulnerable to nutritional deprivation due to its rapid growth and high metabolic demands in early life. During the first 1,000 days—from conception to two years of age—the brain undergoes critical processes including neuronal proliferation, synaptogenesis, myelination, and pruning. Each of these steps is highly dependent on adequate supplies of energy, proteins, essential fatty acids, and micronutrients such as iron, iodine, zinc, and vitamins A, B12, and D [2]. Deficiencies during this sensitive window may cause irreversible damage, impairing cognitive, motor, and socio-emotional capacities that extend far beyond childhood.

The aim of this review is to provide a comprehensive analysis of how severe pediatric malnutrition influences brain development, integrating insights from clinical, epidemiological, and neurobiological research. Unlike traditional discussions that focus primarily on survival outcomes, this review emphasizes the long-term neurodevelopmental consequences of malnutrition, thus reframing the disorder as a determinant of human capital and societal progress.

A major research gap lies in the limited integration of structural brain imaging, neuropsychological outcomes, and interventional data in children with severe malnutrition. Although substantial evidence links nutritional deficiencies to impaired cognitive performance, the underlying neurobiological mechanisms remain incompletely understood. Moreover, the compounded effects of recurrent infections, inflammation, and environmental adversity on neurodevelopment in malnourished children have yet to be fully elucidated [3]. Addressing these gaps is essential to inform effective prevention and intervention strategies.

This article, therefore, explores severe pediatric malnutrition through a pediatric lens, highlighting its epidemiology, pathophysiological mechanisms, neurocognitive consequences, and potential interventions. By consolidating current evidence, it seeks to strengthen the case for prioritizing malnutrition as both a medical and developmental emergency, warranting urgent action from clinicians, policymakers, and researchers alike [4].

Epidemiology of Severe Pediatric Malnutrition

Severe pediatric malnutrition continues to represent a pressing health burden worldwide, particularly in low- and middle-income countries. Current global estimates suggest that approximately 45 million children under the age of five suffer from wasting, while 149 million experience stunting, both of which are strongly associated with impaired survival and development [5]. The highest prevalence is seen in Sub-Saharan Africa and South Asia, where food insecurity, infectious diseases, and poverty converge to perpetuate malnutrition. Despite progress in reducing mortality from malnutrition over recent decades, the neurodevelopmental consequences remain underreported and under-prioritized [6].

The epidemiology of malnutrition is shaped not only by food scarcity but also by social, political, and environmental factors. Humanitarian crises, climate change, displacement, and regional conflicts exacerbate nutritional insecurity, disproportionately affecting children during their most vulnerable years of growth and brain development [7]. Children from rural areas, low-income households, and marginalized communities are at significantly higher risk of malnutrition compared to those in urban or wealthier settings. This unequal distribution reflects deep-rooted socioeconomic disparities that further

compound developmental disadvantages.

Severe acute malnutrition (SAM), characterized by severe wasting or nutritional edema, affects an estimated 14 million children globally at any given time. Of these, more than 90% live in Asia and Africa [8]. Chronic malnutrition, defined by stunting, not only increases the risk of mortality but also represents a marker of long-standing deprivation, which is strongly linked to impaired brain growth. Studies have shown that children who are stunted before the age of two years are at higher risk of poor school achievement, reduced productivity in adulthood, and intergenerational transmission of undernutrition [9].

Epidemiological studies highlight that malnutrition and its consequences on brain development are rarely isolated phenomena. Instead, they often coexist with recurrent infections such as diarrhea, malaria, and respiratory illnesses, all of which aggravate nutritional deficiencies and further compromise neurodevelopment [10]. This syndemic relationship underscores the need for integrated health, nutrition, and infection-control strategies to address the full spectrum of child health and developmental outcomes. Furthermore, regional disparities demonstrate that while high-income countries report relatively low rates of severe malnutrition, hidden forms of undernutrition, such as micronutrient deficiencies, remain prevalent and can still impair brain development. This highlights that malnutrition is a universal issue, though its manifestations and severity vary by context [11].

Pathophysiology of Malnutrition and Brain Development

The pathophysiology of severe pediatric malnutrition and its impact on brain development is multifaceted, involving deficits in macronutrients, micronutrients, and energy supply, which disrupt normal neurodevelopmental processes. The developing brain, particularly during the first 1,000 days of life, requires a continuous supply of nutrients to support rapid neuronal proliferation, dendritic arborization, synaptogenesis, and myelination. When these needs are not met, structural and functional alterations occur, many of which are irreversible if not corrected during critical developmental windows [12].

Protein-energy malnutrition directly impairs brain growth by reducing the availability of amino acids necessary for neurotransmitter synthesis, neuronal repair, and structural protein formation. Inadequate protein intake results in smaller brain size, decreased cell numbers, and impaired synaptic plasticity. Studies on children with marasmus and kwashiorkor have shown reduced dendritic complexity and lower cortical thickness, which correlate with delayed cognitive and motor development [13].

Micronutrient deficiencies further exacerbate brain dysfunction. Iron deficiency compromises myelination and neurotransmitter metabolism, particularly dopamine and serotonin pathways, leading to deficits in attention and memory. Iodine deficiency disrupts thyroid hormone production, which is critical for neuronal migration and differentiation. Zinc deficiency impairs synaptic signaling, while deficiencies in vitamins B12, B6, and folate affect one-carbon metabolism and DNA methylation, mechanisms that regulate gene expression in neurodevelopment [14].

Energy deficits caused by chronic undernutrition alter glucose metabolism, the primary fuel for the brain. Hypoglycemia and reduced energy availability impair neuronal function and can result in long-term deficits in hippocampal growth, with consequences for memory and learning. Additionally, reduced availability of essential fatty acids such as docosahexaenoic acid (DHA) impedes membrane fluidity and synaptic efficiency, further weakening neurocognitive development [15].

Inflammation plays a critical mediating role in the pathophysiology of malnutrition. Malnourished children frequently suffer from recurrent infections that trigger systemic inflammation, which disrupts the blood–brain barrier, induces oxidative stress, and activates microglia. These processes interfere with normal neuronal connectivity and repair. Emerging evidence suggests that chronic low-grade inflammation, in combination with nutrient deprivation, creates a hostile neurodevelopmental environment that amplifies brain injury [16].

Overall, the interplay of protein-energy deficiency, micronutrient deprivation, altered energy

metabolism, and inflammation illustrates how severe malnutrition undermines the structural and functional integrity of the developing brain. This pathophysiological understanding highlights the urgency of early nutritional interventions to mitigate long-term neurodevelopmental damage.

Critical Windows of Neurodevelopment Affected by Malnutrition

Brain development is a dynamic process that begins in utero and extends through adolescence, with specific periods of heightened vulnerability to nutritional deprivation. These "critical windows" represent stages where malnutrition can cause permanent structural and functional impairments if not corrected. Understanding these phases is essential for targeted interventions that maximize recovery and minimize long-term damage [17].

The prenatal period is the earliest and one of the most sensitive phases. Adequate maternal nutrition is crucial for fetal brain growth, neuronal proliferation, and the establishment of basic neural circuitry. Malnutrition during pregnancy, particularly deficiencies in folate, iodine, and iron, has been associated with neural tube defects, impaired myelination, and reduced hippocampal and cortical development. Such prenatal insults set the stage for cognitive delays, low IQ, and increased vulnerability to neurodevelopmental disorders [18].

The first two years of life, often described as the "first 1,000 days," represent another critical window. During this time, rapid synaptogenesis and myelination occur, processes that depend on energy sufficiency and micronutrients such as iron, zinc, and long-chain polyunsaturated fatty acids. Malnutrition during this stage has been linked to stunting, reduced head circumference, and impaired motor and language acquisition. Evidence suggests that recovery after this stage is limited, making early nutrition crucial for lifelong neurodevelopmental outcomes [19].

The preschool and school-age years also represent important, though more plastic, phases of brain development. During this time, higher-order cognitive functions such as executive control, problem-solving, and working memory are refined. Malnutrition at this stage may not cause gross structural deficits but significantly impairs functional capacities, leading to poor school performance, attention deficits, and social-behavioral difficulties. Interventions in this window can still improve outcomes, but often cannot fully compensate for early deficits [20].

Adolescence represents a later critical period when the brain undergoes synaptic pruning and maturation of prefrontal regions responsible for decision-making and emotional regulation. Nutritional deficiencies during adolescence, such as iron or vitamin D deficiency, may impair these processes and perpetuate cycles of poor educational attainment, risky behaviors, and reduced productivity in adulthood. Importantly, malnutrition during adolescence can also influence reproductive health, perpetuating intergenerational malnutrition and neurodevelopmental impairment [21].

Collectively, these critical windows highlight the lifelong consequences of malnutrition when experienced during vulnerable stages of brain growth. Early prevention, especially before and during the first 1,000 days, remains the most effective strategy to protect brain development and ensure optimal cognitive potential.

Nutritional Deficiencies and Specific Neurocognitive Outcomes

Different nutrient deficiencies within the spectrum of severe malnutrition contribute uniquely to impairments in brain development and neurocognitive functioning. Each nutrient plays a specialized role in neural growth, neurotransmitter synthesis, and synaptic efficiency, meaning deficiencies produce distinct patterns of developmental deficits. Understanding these nutrient-specific effects allows for targeted interventions to prevent and correct neurocognitive impairments in children [22].

Protein-energy deficiency is central to severe malnutrition and affects global brain development. Inadequate protein intake reduces the availability of essential amino acids required for neurotransmitters such as dopamine, serotonin, and glutamate, which regulate cognition and mood. Protein deprivation has been linked to smaller brain size, reduced dendritic arborization, and impaired synaptic plasticity. Clinically, children with severe protein-energy malnutrition often present with delayed speech, reduced IQ scores, and deficits in attention and executive functioning [23].

Iron deficiency is among the most common nutritional deficits affecting the brain. Iron is crucial for oxygen transport, myelin formation, and neurotransmitter metabolism. Chronic iron deficiency during infancy is strongly associated with impaired memory, reduced processing speed, and behavioral disturbances such as irritability and poor concentration. Longitudinal studies have shown that iron deficiency in early life predicts lower school achievement and lower cognitive test performance in adolescence [24].

Iodine deficiency represents a major cause of preventable intellectual disability worldwide. Thyroid hormones, which require iodine, are essential for neuronal differentiation, migration, and myelination. Severe iodine deficiency during pregnancy and infancy leads to cretinism, characterized by profound cognitive impairment, hearing loss, and motor dysfunction. Even mild to moderate iodine deficiency has been linked to reduced IQ scores and learning difficulties in school-aged children [25].

Zinc deficiency impairs synaptic transmission, neurogenesis, and brain signaling pathways. Clinical studies associate zinc deficiency with reduced attention span, poor motor development, and delayed language acquisition. Supplementation trials have demonstrated modest improvements in motor and cognitive function, underscoring zinc's critical role in early neurodevelopment [26].

Vitamin B12 and folate deficiencies disrupt one-carbon metabolism, a pathway central to DNA methylation and gene regulation. Deficiency states impair myelination and are associated with developmental delay, hypotonia, and long-term cognitive deficits. Infants with maternal B12 deficiency often exhibit delayed milestones, apathy, and impaired problem-solving skills [27].

Vitamin D deficiency has also emerged as a contributor to impaired brain function, given its role in neurotrophic signaling, calcium homeostasis, and regulation of neuroinflammation. Observational studies link vitamin D deficiency in early childhood with increased risk of language delay, autism spectrum disorder, and impaired memory [28].

In summary, nutrient-specific deficiencies in the context of severe pediatric malnutrition converge to produce profound neurocognitive consequences. Their recognition is essential in clinical practice to design comprehensive supplementation strategies that go beyond caloric rehabilitation to protect and restore brain development.

Structural and Functional Brain Changes in Malnourished Children

Severe pediatric malnutrition results in measurable structural and functional alterations in the developing brain, which can be detected through neuroimaging, neuropathological studies, and neurophysiological assessments. These changes reflect disruptions in neuronal proliferation, myelination, and synaptic connectivity, and they often correlate with long-term deficits in cognition, behavior, and learning [29].

Neuroimaging studies, particularly MRI, have demonstrated that children with a history of severe malnutrition often present with reduced total brain volume, smaller cortical thickness, and decreased hippocampal size. These structural deficits are most prominent in regions responsible for memory, attention, and executive functioning. For example, longitudinal studies in malnourished children in Jamaica and Bangladesh revealed smaller head circumference, which strongly correlated with lower cortical volume and delayed cognitive outcomes [30].

White matter development, which is crucial for rapid signal transmission and higher-order cognitive processing, is also profoundly affected. Diffusion tensor imaging (DTI) studies have shown disrupted white matter integrity in malnourished children, with reduced fractional anisotropy indicating impaired myelination. These abnormalities are especially prominent in the corpus callosum and frontal lobe pathways, which are essential for interhemispheric communication and executive control [31].

Functional brain changes are equally significant. Electroencephalography (EEG) studies have revealed slower background rhythms and reduced alpha activity in malnourished children, indicating delayed cortical maturation. Functional MRI (fMRI) findings also demonstrate hypoactivation of the prefrontal cortex and hippocampus during memory and attention tasks, correlating with poorer performance in cognitive testing [32].

Neuropathological studies support these imaging findings, showing reduced dendritic arborization,

fewer synapses, and diminished neuronal density in cortical regions. Animal models of protein-energy malnutrition have confirmed these structural changes, suggesting that early nutrient deprivation permanently alters brain architecture. Importantly, many of these deficits persist even after nutritional rehabilitation, underscoring the irreversibility of damage when malnutrition occurs during critical developmental periods [33].

Furthermore, malnutrition affects neurochemical balance, including reduced levels of dopamine, serotonin, and acetylcholine, which are essential for attention, learning, and mood regulation. Such neurochemical disruptions explain the increased risk of behavioral problems, including irritability, poor social interaction, and inattention, among malnourished children [34].

In summary, structural and functional brain changes caused by malnutrition provide compelling biological evidence of its long-lasting impact. These alterations, visible at both microstructural and macrostructural levels, highlight the urgent need for early prevention and intervention strategies to protect brain health and optimize neurodevelopmental outcomes.

Cognitive, Behavioral, and Educational Outcomes

Severe pediatric malnutrition has profound consequences on cognitive development, behavior, and educational attainment, which persist well into adolescence and adulthood. These outcomes reflect the combined impact of structural brain changes, neurotransmitter imbalances, and social disadvantages experienced by malnourished children [35].

Cognitive outcomes are among the most extensively documented consequences of severe malnutrition. Children affected in early life consistently score lower on intelligence tests, with deficits observed in working memory, problem-solving, and executive functioning. Longitudinal studies in Jamaica demonstrated that stunted children had significantly lower IQ scores and poorer cognitive flexibility compared to well-nourished peers, even after adjusting for socioeconomic status. These deficits remained detectable years after nutritional rehabilitation, suggesting long-lasting effects on intellectual potential [36].

Behavioral outcomes are equally significant. Malnourished children often display irritability, low frustration tolerance, poor attention span, and difficulties with emotional regulation. Such behavioral patterns are partly explained by disrupted dopaminergic and serotonergic pathways, which are essential for impulse control and mood regulation. Additionally, these children frequently exhibit social withdrawal, reduced play behavior, and lower levels of curiosity, behaviors that further restrict cognitive stimulation during critical developmental years [37].

Educational outcomes highlight the long-term societal consequences of malnutrition. Children who suffered from early-life malnutrition are more likely to demonstrate poor school readiness, delayed language acquisition, and reduced academic achievement. A landmark cohort study in Guatemala found that individuals who experienced chronic malnutrition in early childhood completed fewer years of schooling and had lower literacy rates compared to adequately nourished peers. These findings emphasize the role of nutrition in shaping not only health but also human capital development [38].

Beyond school performance, malnutrition affects productivity and socioeconomic outcomes in adulthood. Adults who were malnourished as children often earn lower incomes and are less likely to engage in skilled labor, perpetuating cycles of poverty and undernutrition across generations. This intergenerational effect underscores the broader economic and societal impact of pediatric malnutrition, extending beyond immediate health consequences [39].

Intervention studies demonstrate that while nutritional supplementation can partially improve cognitive outcomes, the greatest benefits are seen when combined with early psychosocial stimulation. Programs that provide enriched caregiving environments alongside therapeutic feeding have been shown to enhance IQ, school performance, and emotional well-being more effectively than nutrition alone. This highlights the importance of a multidisciplinary approach to address both biological and psychosocial dimensions of child development [40].

Socioeconomic and Environmental Determinants

Severe pediatric malnutrition and its impact on brain development cannot be understood solely in biological terms; socioeconomic and environmental determinants play a pivotal role in shaping both the onset and outcomes of malnutrition. Poverty, food insecurity, poor sanitation, and limited access to healthcare act as powerful drivers of undernutrition, influencing not only physical growth but also neurodevelopmental potential [41].

Poverty is the single strongest predictor of childhood malnutrition. Families with limited financial resources often lack access to diverse and nutrient-rich diets, leading to reliance on calorie-dense but nutrient-poor foods. In addition, poverty is associated with reduced access to healthcare services, increasing children's vulnerability to infections that further exacerbate nutrient deficiencies. Importantly, children growing up in impoverished households are also less likely to receive adequate psychosocial stimulation, compounding the developmental delays caused by nutritional deficits [42].

Environmental conditions, particularly inadequate sanitation and unsafe water, contribute to a condition known as environmental enteric dysfunction (EED), which impairs nutrient absorption and promotes chronic inflammation. Children living in unhygienic environments are at high risk of diarrheal diseases and parasitic infections, both of which worsen malnutrition and hinder brain development. The combination of nutrient loss, systemic inflammation, and reduced absorption creates a vicious cycle that amplifies neurocognitive deficits [43].

Maternal education and empowerment are also critical determinants of child nutrition and neurodevelopment. Studies consistently show that higher levels of maternal education are associated with better feeding practices, improved healthcare utilization, and enhanced child developmental outcomes. Educated mothers are more likely to provide dietary diversity, seek medical care early, and engage in stimulating caregiving practices, all of which protect children against the developmental harms of malnutrition [44].

Broader societal factors, including conflict, displacement, and climate change, further exacerbate malnutrition risks. Armed conflicts disrupt food systems, limit healthcare access, and displace vulnerable populations, creating environments where malnutrition thrives. Similarly, climate change threatens food security by reducing crop yields and increasing food price volatility, disproportionately affecting children in already food-insecure regions. These global determinants underscore the need for multisectoral interventions that address not only nutrition but also broader socioeconomic inequities [45].

Ultimately, the relationship between malnutrition and brain development is mediated by a complex web of social and environmental determinants. Effective strategies to combat malnutrition must therefore go beyond food provision, addressing poverty reduction, maternal empowerment, education, sanitation, and climate resilience to safeguard child neurodevelopment and future human capital.

Diagnosis and Management of Severe Pediatric Malnutrition

The diagnosis and management of severe pediatric malnutrition are critical not only for reducing mortality but also for minimizing the long-term neurodevelopmental consequences associated with nutrient deprivation. Early recognition, accurate assessment, and comprehensive management are key pillars in addressing this condition effectively [46].

Diagnosis of severe pediatric malnutrition is primarily based on anthropometric, clinical, and laboratory criteria. According to WHO guidelines, severe acute malnutrition (SAM) is defined by a weight-for-height z-score below –3 standard deviations, the presence of bilateral pitting edema, or a mid-upper arm circumference (MUAC) less than 115 mm. Clinical examination often reveals muscle wasting, growth faltering, irritability, apathy, and, in some cases, hepatomegaly due to fatty liver infiltration. Beyond anthropometry, assessment should also include developmental screening to identify early signs of cognitive and motor delays, as these may indicate malnutrition's impact on brain development [47].

Laboratory investigations may be necessary to assess micronutrient deficiencies, electrolyte imbalances, and coexisting infections. Common findings include anemia, hypoglycemia, hypokalemia, and low

serum levels of iron, zinc, and vitamin D. Developmental assessments, including tools such as the Bayley Scales of Infant Development or Denver Developmental Screening Test, can provide insight into the neurodevelopmental deficits associated with malnutrition, though these are often underutilized in resource-limited settings [48].

Management of severe pediatric malnutrition requires a stepwise and multidisciplinary approach. Immediate priorities include stabilization of life-threatening complications such as hypoglycemia, hypothermia, dehydration, and infections. WHO protocols recommend therapeutic feeding with F-75 formula during stabilization, followed by gradual transition to energy-dense F-100 formula or ready-to-use therapeutic foods (RUTF) for nutritional rehabilitation. These interventions aim to restore weight, replenish nutrient stores, and support catch-up growth [49].

Micronutrient supplementation is integral to management, particularly for iron, zinc, iodine, vitamin A, and vitamin D, as deficiencies in these nutrients disproportionately affect brain development. However, iron supplementation is typically delayed until after stabilization due to the risk of worsening infections. Beyond nutritional therapy, management must include treatment of coexisting infections such as pneumonia, malaria, and tuberculosis, which are common in malnourished children and further impair neurodevelopment [50].

Long-term management extends beyond acute rehabilitation and should incorporate developmental stimulation and caregiver education. Evidence from intervention studies indicates that children who receive psychosocial stimulation in combination with nutritional rehabilitation show better outcomes in IQ, school performance, and emotional regulation compared to those who receive nutrition alone. Follow-up programs must also address environmental determinants, including food insecurity, sanitation, and parental education, to prevent relapse and promote sustained brain development [51].

In summary, the diagnosis and management of severe pediatric malnutrition require integration of nutritional, medical, and developmental care. Early detection, timely therapeutic feeding, micronutrient supplementation, infection control, and psychosocial interventions form the cornerstone of comprehensive management. This holistic approach maximizes the chances of recovery and mitigates long-term neurocognitive impairment.

Conclusion

Severe pediatric malnutrition is far more than a disorder of growth; it is a profound threat to the developing brain and, by extension, to the child's lifelong potential. The evidence across clinical, neurobiological, and epidemiological studies highlights that malnutrition disrupts critical processes such as synaptogenesis, myelination, and neurotransmitter regulation, leaving lasting imprints on cognition, behavior, and educational outcomes. These changes are not only biologically mediated but also strongly influenced by social and environmental determinants such as poverty, maternal education, sanitation, and food security.

Diagnosis and management require more than identifying anthropometric deficits; they must encompass developmental assessment and early intervention to protect neurocognitive outcomes. Comprehensive management strategies that combine therapeutic feeding, micronutrient supplementation, infection control, and psychosocial stimulation offer the best chance of reversing some of the adverse impacts. However, the irreversibility of certain brain changes underscores the urgency of early prevention, particularly during the prenatal period and the first 1,000 days of life.

The long-term consequences of severe malnutrition extend beyond childhood, shaping adult health, productivity, and intergenerational cycles of disadvantage. By framing severe malnutrition as both a medical and developmental emergency, there is an opportunity to mobilize multidisciplinary and multisectoral responses that address biological, social, and environmental determinants simultaneously. Ultimately, protecting the developing brain from the effects of malnutrition is not just a matter of child survival but also of safeguarding the intellectual and economic future of societies. Early, integrated, and sustained interventions represent the most powerful tools to break the cycle of malnutrition and unlock the full developmental potential of children worldwide.

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