# Trace Element Imbalances and Their Impact on Community-Acquired Pneumonia in Pediatrics

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***Abstract***

**Background:** Community-acquired pneumonia (CAP) remains one of the leading causes of morbidity and mortality among children worldwide, particularly in low- and middle-income countries. Despite the availability of antibiotics and vaccines, CAP continues to place a substantial burden on pediatric health, often complicated by nutritional deficiencies that impair host defense mechanisms. Among these, imbalances in trace elements such as selenium (Se), zinc (Zn), iron (Fe), and copper (Cu) have emerged as important factors influencing both susceptibility to infection and disease outcomes. These micronutrients play critical roles in immune function, oxidative stress regulation, and microbial control, making their assessment in children with CAP highly relevant.

This review aims to evaluate the relationship between serum levels of Se, Zn, Fe, and Cu and the severity of CAP in pediatric populations. Evidence suggests that zinc deficiency is strongly associated with impaired innate immunity and higher pneumonia incidence, while selenium contributes to antioxidant defense and viral resistance. Iron status remains complex, as both deficiency and overload can predispose to infection by modulating bacterial growth and host immunity. Copper, though less studied, participates in enzymatic pathways critical for oxidative killing of pathogens and may act synergistically with zinc in maintaining immune competence. Understanding how these elements interact, and how their deficiency or excess correlates with CAP severity, is crucial for developing effective adjunctive strategies in pediatric pneumonia management.

Several observational studies indicate that lower serum zinc and selenium levels correlate with more severe pneumonia presentations, prolonged hospital stays, and higher inflammatory markers. Conversely, disturbances in iron and copper homeostasis may serve as predictors of disease progression and complications. However, inconsistencies in study methodologies, patient populations, and cutoff values limit generalizability.

In conclusion, trace element imbalances represent modifiable risk factors that could influence the clinical course of pediatric CAP. Comprehensive evaluation of Se, Zn, Fe, and Cu may provide valuable biomarkers for assessing disease severity and guiding therapeutic interventions. Further multicenter studies and randomized controlled trials are needed to validate these associations and to explore the potential role of micronutrient supplementation as an adjunctive treatment in pediatric CAP.

***Keywords:*** *Trace Element , Community-Acquired Pneumonia, Pediatrics*

## Introduction

Community-acquired pneumonia (CAP) is a major global health challenge and remains one of the leading causes of morbidity and mortality in children under five years of age, particularly in developing countries. According to the World Health Organization, pneumonia accounts for approximately 15% of all deaths in children within this age group, highlighting its persistent burden despite advancements in vaccination, antimicrobial therapy, and supportive care [1]. Beyond infectious etiology, the outcome of CAP is increasingly recognized to be influenced by host-related factors such as nutritional status, immune competence, and micronutrient balance.

Trace elements, including selenium (Se), zinc (Zn), iron (Fe), and copper (Cu), are integral to various physiological and immunological processes. Selenium functions as a cofactor for glutathione peroxidase, thereby reducing oxidative stress and enhancing viral and bacterial clearance [2]. Zinc is crucial for both innate and adaptive immune responses, supporting the function of macrophages, neutrophils, and natural killer cells, while also facilitating epithelial barrier integrity [3]. Iron plays a dual role: while essential for hemoglobin synthesis and immune activity, excess iron may promote bacterial proliferation by serving as a nutrient for pathogens [4]. Copper, though less extensively studied, contributes to enzymatic antioxidant defense and pathogen elimination through its role in superoxide dismutase and ceruloplasmin [5].

The relationship between trace element imbalance and CAP severity is complex and not fully understood. Several studies have demonstrated that deficiencies in zinc and selenium are associated with higher rates of respiratory infections, longer hospitalizations, and more severe disease manifestations [6,7]. Meanwhile, dysregulation of iron metabolism, particularly anemia of inflammation, has been reported as a predictor of poor clinical outcomes [8]. Copper status, which often fluctuates in response to infection, may also reflect disease severity through its link to inflammatory and oxidative processes [9].

Despite these associations, there is a lack of comprehensive synthesis examining the combined impact of these four trace elements on pediatric CAP severity. Research gaps exist in defining optimal serum thresholds, understanding element-element interactions, and exploring whether supplementation could reduce morbidity. Therefore, this review aims to evaluate existing evidence on Se, Zn, Fe, and Cu levels in children with CAP and their correlation with disease severity. By addressing these gaps, the review highlights the potential role of trace element monitoring as both a prognostic tool and a therapeutic target in pediatric pneumonia.

## Community-Acquired Pneumonia in Children: Epidemiology and Burden

Community-acquired pneumonia (CAP) is a leading cause of infectious morbidity in children globally, disproportionately affecting populations in low- and middle-income countries. The global incidence of CAP in children under five years is estimated at 150 million new cases annually, with approximately 20 million cases requiring hospital admission [10]. Mortality remains significant, with nearly 800,000 pediatric deaths reported worldwide in 2019, despite the availability of preventive strategies such as pneumococcal and Haemophilus influenzae type b (Hib) vaccines [11]. The burden is most pronounced in sub-Saharan Africa and South Asia, where malnutrition, poor access to healthcare, and high exposure to infectious pathogens converge to amplify disease risk.

The epidemiology of CAP varies depending on age, geography, season, and vaccination coverage. In industrialized nations, viral pathogens such as respiratory syncytial virus (RSV), influenza, and adenovirus are most commonly implicated, while in developing countries, bacterial agents like Streptococcus pneumoniae and Staphylococcus aureus predominate [12]. Malnutrition, including deficiencies in micronutrients like zinc and selenium, increases susceptibility to CAP by impairing immune defenses and epithelial integrity [13]. This highlights the interplay between nutritional status and infectious disease burden, making the assessment of trace elements highly relevant in pediatric

populations.

Socioeconomic determinants such as overcrowding, indoor air pollution from biomass fuels, and limited vaccination uptake further exacerbate the incidence and severity of CAP [14]. Children living in resource-limited settings often face a double burden of infectious exposure and inadequate nutrition, compounding their risk. Mortality rates are highest among infants and young children due to immature immune systems and high metabolic demands. Early recognition of risk factors and prompt management are critical to reducing case fatality, which can exceed 10% in severe cases requiring hospitalization [15].

The economic and healthcare burden of CAP is considerable. Hospital admissions, antibiotic use, and intensive care requirements place a strain on already overburdened health systems, particularly in low-income regions. In addition to immediate morbidity, CAP can lead to long-term sequelae such as reduced lung function, impaired growth, and increased risk of recurrent respiratory infections [16]. These outcomes underscore the need to address not only the infectious etiology but also host factors like micronutrient deficiencies that may worsen disease course.

In summary, pediatric CAP remains a major public health problem, with significant mortality, morbidity, and economic costs worldwide. Understanding how underlying nutritional and trace element imbalances contribute to this burden is essential to developing targeted preventive and therapeutic strategies. The following sections explore the immunological significance of selenium, zinc, iron, and copper in this context.

## Immunological Role of Trace Elements in Childhood Infections

Trace elements are essential micronutrients required in small amounts but exert disproportionately large effects on immune function and host defense. In children, deficiencies or imbalances in selenium, zinc, iron, and copper can compromise immune competence, predisposing them to infections such as community-acquired pneumonia (CAP). These elements function as cofactors in enzymatic systems, regulators of oxidative stress, and modulators of innate and adaptive immunity [17]. Their combined influence is particularly critical in pediatric populations, where rapid growth, immature immune systems, and frequent infections increase metabolic demands for micronutrients.

**Selenium** plays a vital role in antioxidant defense through incorporation into selenoproteins such as glutathione peroxidases and thioredoxin reductases. These enzymes neutralize reactive oxygen species (ROS), thereby limiting tissue damage during infection and supporting lymphocyte proliferation [18]. Selenium deficiency has been linked to impaired T-cell function, reduced antibody production, and increased susceptibility to viral and bacterial respiratory infections [19]. Experimental studies have shown that low selenium status enhances viral mutation rates and pathogenicity, further complicating respiratory illnesses [20].

**Zinc** is indispensable for both innate and adaptive immunity. It influences the activity of neutrophils, macrophages, and natural killer cells while also facilitating cytokine production and T-lymphocyte maturation [21]. Zinc deficiency impairs epithelial barrier integrity in the respiratory tract, increases vulnerability to pathogen invasion, and weakens the inflammatory response needed for effective clearance [22]. Importantly, zinc also regulates the balance between pro-inflammatory and anti-inflammatory pathways, preventing excessive tissue damage during infection [23].

**Iron** is a critical determinant of host-pathogen interactions. While iron is essential for DNA synthesis, cellular respiration, and immune cell proliferation, pathogens such as Streptococcus pneumoniae and Haemophilus influenzae also require iron for growth [24]. The host immune system regulates iron availability through proteins like hepcidin and lactoferrin, a process termed *nutritional immunity*. Dysregulated iron metabolism, whether from deficiency or overload, disrupts this balance, leading either to impaired host immunity or enhanced pathogen proliferation [25].

**Copper** contributes to immune defense primarily through its role in redox reactions and enzymatic activity. It serves as a cofactor for enzymes such as superoxide dismutase and ceruloplasmin, which

neutralize free radicals generated during infection [26]. Copper also has direct antimicrobial activity, as copper ions can damage bacterial membranes, proteins, and DNA [27]. Deficiency impairs neutrophil bactericidal activity, while elevated copper levels are often observed during acute infections, reflecting its role as an acute-phase reactant [28].

In summary, the immunological relevance of selenium, zinc, iron, and copper underscores their potential role as biomarkers of disease severity in pediatric CAP. Imbalances compromise immune resilience and recovery, while adequate levels may mitigate infection severity. Understanding these interactions provides a foundation for evaluating their role in CAP pathophysiology.

**Selenium and Pediatric Pneumonia**

Selenium is a trace element that plays a crucial role in antioxidant defense and immune function, largely through its incorporation into selenoproteins such as glutathione peroxidases, thioredoxin reductases, and selenoprotein P. These proteins mitigate oxidative stress generated during infection and maintain cellular redox balance [29]. In pediatric pneumonia, selenium deficiency has been associated with increased oxidative injury to lung tissues, impaired immune responses, and higher susceptibility to severe forms of disease [30]. Children in low-resource settings are particularly vulnerable, as soil selenium deficiency often translates into lower dietary intake.

Several studies have demonstrated a correlation between low selenium status and increased incidence and severity of respiratory infections in children. Beck et al. first highlighted the role of selenium in viral pathogenesis, showing that deficiency can enhance viral virulence and worsen clinical outcomes [31]. In pediatric populations, reduced serum selenium concentrations have been linked with more severe CAP, longer hospital stays, and higher inflammatory marker levels [32]. This is likely due to impaired antioxidant capacity, resulting in unchecked oxidative damage during pulmonary infection.

Selenium also exerts effects on the adaptive immune system. Adequate selenium levels are necessary for the proliferation and differentiation of T lymphocytes and for optimal antibody production by B cells [33]. In selenium-deficient children, impaired T-helper cell function compromises the immune response to bacterial pathogens such as Streptococcus pneumoniae, a leading cause of CAP [34]. Moreover, selenium deficiency can exacerbate inflammatory cascades by reducing the regulation of nuclear factor-kappa B (NF-κB) signaling, thereby amplifying cytokine-mediated lung injury [35].

Clinical trials evaluating selenium supplementation in pediatric pneumonia are limited but promising. Some randomized studies have reported that selenium supplementation reduces the duration of hospitalization, improves oxygenation, and decreases C-reactive protein levels in children with CAP [36]. However, heterogeneity in study designs, dosage regimens, and baseline selenium status makes it difficult to establish standardized guidelines. Concerns about selenium toxicity at high doses also necessitate careful evaluation before widespread supplementation is recommended [37].

In summary, selenium deficiency appears to be a significant risk factor for increased severity of CAP in children. While existing data suggest potential therapeutic benefits of supplementation, further large-scale, multicenter studies are required to clarify its role as an adjunctive treatment. Monitoring selenium levels in children with CAP could help identify those at higher risk of severe disease and guide personalized nutritional interventions.

**Zinc and Pediatric Pneumonia**

Zinc is one of the most extensively studied trace elements in relation to pediatric infections, including community-acquired pneumonia (CAP). It plays a vital role in cellular metabolism, growth, and especially immune defense. Zinc supports both innate and adaptive immune systems by promoting epithelial integrity, modulating cytokine production, and regulating the function of macrophages, neutrophils, and natural killer cells [38]. In its absence, the mucosal barrier of the respiratory tract becomes compromised, facilitating pathogen invasion and increasing susceptibility to pneumonia.

Zinc deficiency is highly prevalent in low- and middle-income countries, where dietary intake is insufficient and infections increase zinc losses. Epidemiological studies have shown that children with zinc deficiency are at significantly higher risk of developing pneumonia and other acute respiratory

infections [39]. In addition, low serum zinc levels are often associated with greater pneumonia severity, prolonged hospital stays, and increased mortality [40]. This makes zinc not only a risk factor for pneumonia incidence but also a determinant of disease outcome.

Mechanistically, zinc deficiency impairs the development and activation of T lymphocytes, decreases thymulin activity, and reduces antibody production [41]. Zinc also regulates the balance between pro- and anti-inflammatory cytokines, preventing excessive lung inflammation during infection [42]. Clinical studies suggest that zinc deficiency may lead to exaggerated inflammatory responses, worsening lung injury, and delayed recovery. Interestingly, zinc also influences antimicrobial peptide expression in the respiratory tract, strengthening the host's first line of defense against pathogens [43].

Supplementation studies have provided strong evidence of zinc's beneficial role in reducing pneumonia morbidity. Randomized controlled trials conducted in South Asia and sub-Saharan Africa demonstrated that zinc supplementation reduced the incidence of pneumonia and shortened recovery time in children with acute lower respiratory infections [44]. Moreover, prophylactic supplementation in zinc-deficient populations has been associated with reduced rates of respiratory infections overall [45]. However, some trials in well-nourished populations failed to show similar benefits, highlighting that baseline nutritional status plays a key role in determining zinc's clinical impact [46].

Overall, zinc deficiency is a major modifiable risk factor for pediatric pneumonia. Adequate zinc intake appears essential for both prevention and improved recovery from CAP. While supplementation has proven effective in high-risk groups, further research is needed to optimize dosing regimens, duration, and the integration of zinc therapy into standard pneumonia management guidelines.

**Iron and Pediatric Pneumonia**

Iron is an essential trace element required for hemoglobin synthesis, oxygen transport, energy metabolism, and immune cell function. However, its role in pediatric pneumonia is paradoxical, as both deficiency and excess may increase susceptibility to infection. Iron is a critical nutrient for many pathogens, including *Streptococcus pneumoniae* and *Haemophilus influenzae*, which thrive when host iron availability is increased [47]. To counter this, the host immune system limits iron access to microbes through mechanisms such as hepcidin-mediated sequestration and increased ferritin synthesis, a process known as *nutritional immunity* [48].

Iron deficiency is highly prevalent in children under five years, especially in resource-limited settings where CAP is also most common. Several studies have reported that iron-deficient children are more prone to recurrent respiratory infections, including pneumonia [49]. This may be due to impaired T-lymphocyte proliferation, reduced neutrophil activity, and diminished bactericidal capacity of macrophages in iron deficiency [50]. Conversely, some evidence suggests that iron deficiency may confer partial protection against certain bacterial infections by restricting iron availability to pathogens [51]. This duality makes iron's role in pneumonia particularly complex.

Anemia of inflammation, often observed in children with pneumonia, is mediated by cytokine-induced hepcidin upregulation, which decreases iron absorption and mobilization [52]. This adaptive response reduces iron availability to pathogens but simultaneously compromises erythropoiesis and oxygen delivery to tissues. Severe anemia has been associated with worse pneumonia outcomes, including prolonged hypoxemia, delayed recovery, and higher mortality rates [53]. Distinguishing between iron deficiency anemia and anemia of inflammation remains a clinical challenge, complicating decisions on iron supplementation.

Iron supplementation in children with pneumonia has shown mixed results. While supplementation may improve hemoglobin levels and overall immunity in deficient children, it also carries the risk of exacerbating infections by increasing iron availability to pathogens [54]. A large randomized trial in Zanzibar demonstrated higher rates of severe infections in children receiving iron and folate supplementation, raising concerns about indiscriminate use in high-burden settings [55]. Consequently, the World Health Organization recommends careful assessment before initiating iron therapy in children with acute infections.

In summary, iron status plays a dual role in pediatric pneumonia: deficiency impairs immune function, while excess iron may enhance microbial proliferation. Careful monitoring of iron metabolism markers is essential to guide therapy. Research into safe, context-specific iron supplementation strategies during pneumonia remains a pressing need to optimize outcomes without increasing infectious risks.

**Copper and Pediatric Pneumonia**

Copper is an essential cofactor for several redox-active enzymes—including cytochrome c oxidase, superoxide dismutase (Cu/Zn-SOD), and ceruloplasmin—that collectively shape oxidative burst capacity, iron trafficking, and epithelial repair during infection. In the respiratory tract, adequate copper supports neutrophil and macrophage microbicidal functions and helps limit collateral tissue injury by dismutating superoxide generated during host–pathogen encounters. Conversely, copper deficiency impairs neutrophil bactericidal activity and T-cell proliferation, while excess free copper can catalyze Fenton-like reactions and propagate oxidative damage—illustrating a narrow therapeutic window relevant to inflamed pediatric lungs. Clinically, ceruloplasmin behaves as an acute-phase reactant, and infection-driven rises in circulating copper often mirror systemic inflammation rather than repletion of functional copper at tissue level. These dynamics suggest that serum copper may track disease activity but not always reflect true intracellular sufficiency in children with CAP. [5,26,28]

Beyond systemic markers, copper intersects with *nutritional immunity* through its reciprocal relationship with iron. Ceruloplasmin's ferroxidase activity facilitates iron export via hephaestin/ferroportin; during acute infection, inflammatory signaling perturbs this axis, altering both copper and iron bioavailability. Such shifts can influence pathogen fitness (many bacteria are copper-sensitive yet iron-dependent) and host hypoxic stress in pneumonic lung. Experimental work at the host–pathogen interface shows that macrophages exploit copper's direct antimicrobial properties by elevating phagosomal copper to intoxicate microbes, while maintaining stringent intracellular copper handling to avoid host toxicity—physiology that may be compromised in deficiency states. In pediatrics, where growth demands are high and antioxidant reserves modest, perturbations in copper balance may therefore amplify CAP severity through combined effects on oxidative killing, barrier integrity, and iron handling. [25,26,27]

Therapeutically, routine copper supplementation for CAP is not currently recommended; evidence in children remains limited and heterogeneous, and infection-related hypercupremia can confound assessment. When copper deficiency is suspected (e.g., malnutrition, chronic diarrhea, malabsorption), targeted correction should be considered within comprehensive nutritional care, ideally guided by a panel that includes serum copper, ceruloplasmin, and—when feasible—functional indices. Given copper's redox reactivity, indiscriminate supplementation during acute inflammation could theoretically exacerbate oxidative injury, whereas correction of confirmed deficiency may restore neutrophil function and antioxidant defenses. Until pediatric CAP-specific trials clarify risk–benefit profiles, a prudent approach is individualized assessment integrated with overall trace-element status rather than single-nutrient interventions. [5,26,28]

**Interactions and Imbalances Among Trace Elements**

Trace elements rarely act in isolation; their biological effects are interconnected, and imbalances can amplify disease severity in pediatric pneumonia. **Zinc and copper** share common absorption pathways in the intestine, meaning excess intake of one can antagonize the other. For example, high zinc supplementation can induce copper deficiency, impairing antioxidant defenses [56]. Similarly, **iron and zinc** compete for absorption through divalent metal transporters, and deficiency of one can worsen the immune impact of the other [57].

The **selenium–zinc axis** is particularly relevant in pneumonia, as both support antioxidant and immune functions. Deficiency in one may exacerbate the detrimental effects of the other, compounding oxidative stress and impaired host defense [58]. Likewise, disturbances in **iron–copper interactions** can alter oxygen transport and iron mobilization, influencing both pathogen survival and host tissue resilience [59].

Overall, the delicate balance among selenium, zinc, iron, and copper is crucial for maintaining effective

immunity. Disruption of this equilibrium—whether from dietary insufficiency, infection-driven redistribution, or supplementation imbalance—can worsen CAP severity in children. Integrated assessment of multiple trace elements, rather than focusing on single nutrients, is therefore vital in both research and clinical management

**Biomarker Potential of Trace Elements in Pneumonia Severity**

The search for reliable biomarkers to assess pneumonia severity in children has gained momentum, and trace elements such as selenium, zinc, iron, and copper have emerged as potential candidates. Altered serum levels of these micronutrients often reflect both nutritional status and the systemic inflammatory response. For instance, **low zinc and selenium concentrations** have consistently been associated with more severe pneumonia presentations, higher inflammatory marker levels, and prolonged recovery times [60]. These findings suggest their potential as prognostic indicators in pediatric CAP.

Iron parameters—including serum ferritin, transferrin saturation, and soluble transferrin receptor—have also been explored as biomarkers. Elevated ferritin levels may indicate anemia of inflammation, while low serum iron and transferrin saturation often correlate with severe disease [61]. However, interpretation is challenging because these markers are strongly influenced by the acute-phase response, making it difficult to differentiate true iron deficiency from infection-driven redistribution.

Copper, primarily measured through ceruloplasmin, often rises during acute infections as part of the body's defense mechanisms [62]. Elevated copper levels may therefore serve more as markers of systemic inflammation than of nutritional status. Some studies propose that the **zinc-to-copper ratio** could be a better indicator of disease severity than either element alone, as it reflects the balance between immune support (zinc) and inflammatory activation (copper) [63].

Despite their promise, the use of trace elements as clinical biomarkers faces several limitations. Serum levels can be influenced by acute infection, malnutrition, or supplementation, reducing specificity. Moreover, reference ranges for children vary by age, geography, and baseline diet, complicating standardization. Nonetheless, combining trace element status with established clinical scores (such as WHO pneumonia severity classification) and inflammatory markers (CRP, procalcitonin) may enhance diagnostic accuracy and prognostic value [64].

In summary, trace elements hold potential as adjunctive biomarkers for assessing pneumonia severity in children. While zinc, selenium, and iron deficiencies often signal higher risk, copper behaves as an acute-phase reactant, and ratios among elements may better reflect disease status. Further studies are needed to validate cutoff values and integrate trace element assessment into routine pediatric pneumonia management.

The evidence linking selenium, zinc, iron, and copper with the incidence and severity of pediatric community-acquired pneumonia (CAP) highlights important avenues for clinical application. First, assessment of trace element status in children presenting with pneumonia could provide valuable insight into prognosis. Routine monitoring of serum zinc, selenium, iron indices, and copper, particularly in high-risk populations, may help identify children at greater risk of severe disease and prolonged recovery. Integrating micronutrient evaluation into pediatric pneumonia management protocols could thus enhance risk stratification.

Second, nutritional interventions hold promise as adjunctive therapies. Zinc supplementation has the strongest evidence base, with multiple clinical trials demonstrating its ability to reduce pneumonia incidence and improve recovery in deficient populations. Selenium supplementation appears beneficial in enhancing antioxidant defenses and reducing oxidative injury during infection, although larger pediatric trials are needed. In contrast, iron supplementation remains controversial, as it can both correct deficiency-related immunosuppression and potentially fuel pathogen growth. Copper supplementation is least studied in pneumonia, and its therapeutic role remains largely theoretical, though correction of frank deficiency is essential.

Third, public health strategies targeting the prevention of micronutrient deficiencies may reduce CAP burden on a population level. Fortification programs, dietary diversification, and maternal

supplementation during pregnancy and lactation may collectively improve baseline nutritional status in children, reducing susceptibility to respiratory infections. Tailoring such interventions to regional deficiencies and dietary patterns is critical to maximize benefit.

Finally, the interactions among trace elements must be considered in clinical practice. Supplementing one micronutrient can inadvertently affect the absorption or utilization of others, underscoring the importance of integrated approaches rather than isolated interventions. The zinc-to-copper ratio, for example, may prove to be more informative than single nutrient values in guiding supplementation strategies.

## Conclusion

Trace element imbalances significantly influence the susceptibility, progression, and severity of community-acquired pneumonia in children. Selenium and zinc deficiencies impair antioxidant and immune defenses, iron dysregulation contributes to both immunosuppression and pathogen proliferation, and copper plays a dual role as a cofactor in host defense and an acute-phase reactant. Together, these micronutrients form an intricate network that modulates the host-pathogen interaction in pediatric pneumonia.

While zinc and selenium show the most consistent associations with improved clinical outcomes, iron and copper remain more complex due to their infection-driven fluctuations. The integration of trace element monitoring into pediatric pneumonia care has the potential to improve disease stratification and guide adjunctive nutritional interventions. Moreover, population-level strategies aimed at preventing deficiencies may reduce the global burden of pneumonia in children, particularly in resource-limited settings.

Future research should focus on large, well-designed trials to clarify optimal supplementation regimens, establish standardized reference ranges for children, and evaluate combined trace element approaches. By bridging clinical care with nutritional science, the management of pediatric CAP can move toward more holistic, personalized, and preventive strategies that address not only the pathogen but also the child's nutritional resilience.

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