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THE EFFECT OF EGG FEEDING ON SERUM THIAMINE LEVELS IN CHILDREN WITH *STUNTING* USING THE *ENZYME-LABELLED PROTEIN LIGAND ASSAY* (ELPLA) METHOD

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Abstract

Stunting is a chronic nutritional problem that remains a public health challenge in Indonesia and is associated with impaired linear growth, metabolic changes, and micronutrient deficiencies, including thiamine (vitamin B1). As an essential cofactor in energy metabolism, thiamine deficiency has the potential to exacerbate growth impairments in stunted children. This study aimed to assess thiamine levels using plasma samples from stunted children aged 24–59 months who had received a 30-day egg intervention and to compare them with healthy children. Thiamine levels were measured using the Enzyme-Labeled Protein Ligand Assay (ELPLA), an alternative method that is simpler and more economical than instrumental methods such as HPLC and fluorometry. The results showed that the mean thiamine levels increased from 1.06 ± 1.95 µg/mL to 3.76 ± 1.51 µg/mL after the intervention ($p<0.001$). Thiamine levels in stunted children before the intervention were lower than those in normal children (6.33 ± 0.57 µg/mL; $p < 0.001$) and remained lower after the intervention ($p < 0.001$). Thus, one month of egg consumption was associated with an increase in serum thiamine levels in stunted children, but it was not yet sufficient to restore levels to those of normal children.

Keywords Stunting, Thiamine, Egg Intervention, Enzyme-Labelled Protein Ligand Assay, Thiamine-Binding Protein.

INTRODUCTION

Stunting is a condition of height/age below -2 SD according to WHO standards, reflecting the cumulative effects of malnutrition and infection since before birth. The main causes include long-term malnutrition, repeated infections, and lack of water and sanitation infrastructure. Nutritional interventions are important because they increase macronutrients for tissue formation, muscle mass, growth hormone activity, and micronutrients for metabolic enzyme function that support linear growth. Based on the 2025 SSGI, the prevalence of child stunting in Indonesia is 19.8%, with a target of reducing it to 18.8% in 2025 and 14.4% in 2029. Stunting remains a major health problem with a high prevalence in children under five, also influenced by micronutrient deficiencies. Indonesia needs local biochemical diagnostic methods that are accurate, inexpensive, and easy to use in primary health facilities for early detection of micronutrient deficiencies, without relying on high-cost imported methods such as HPLC or ELISA. Thiamine is a water-soluble vitamin found in abundance in lean meat, wheat germ, poultry, eggs, fish, nuts, legumes, and whole grains. In its natural form, thiamine exists as thiamine pyrophosphate (TPP), which can be hydrolyzed to thiamine monophosphate (TMP). Thiamine's structure is less stable, requiring thiamine-binding proteins (TPPs/TBPs) for activation. TBPs have numerous uses in medical biotechnology, including diagnostic functions for detecting disease biomarkers.

Thiamine, or vitamin B1, functions as an essential cofactor in metabolic reactions, particularly energy production and enzymatic functions in the nitric acid cycle, glycolysis, and the pentose phosphate pathway. Thiamine acts as an essential coenzyme in carbohydrate metabolism through the pyruvate dehydrogenase (PDH) and transketolase complexes. Thiamine deficiency reduces the activity of these enzymes, thus reducing ATP production for bone and muscle cell proliferation, inhibiting linear growth in children and contributing to stunting. Thiamine deficiency can also cause disorders of the central nervous system and carbohydrate metabolism, as well as diseases such as beriberi and Wernicke-Korsakoff syndrome. Therefore, thiamine can be used as a biochemical biomarker for metabolic diseases such as diabetes mellitus and stunting.

Stunting due to thiamine deficiency can be detected using thiamine-binding protein (TPP). TP from grains and legumes can bind thiamine with high affinity. Thiamine levels can be determined using biological fluids such as serum and blood plasma containing free thiamine, then PPT is added to measure its affinity. Current methods for measuring thiamine are expensive, inaccurate, or require complex equipment. Gunarti et al. developed the Enzyme-Labeled Protein Ligand Assay (ELPLA) method, which is more accurate than HPLC and ELISA, and in this study was used to measure blood plasma thiamine levels in stunted and healthy children.

LITERATURE REVIEW

a. Stunting

Stunting is defined as impaired linear growth and development in children due to chronic malnutrition and/or recurrent infections, characterized by a length/height-for-age Z-score less than two standard deviations below the age-sex median according to the World Health Organization (WHO) growth standards. Stunting is an irreversible process that occurs in the first 1,000 days of life, involving biological, nutritional, infectious, and social and environmental factors, with both direct and indirect causes. Pathophysiologically, stunting is a manifestation of linear growth failure due to impaired bone formation and elongation at the growth plate through endochondral ossification. In carbohydrate metabolism, stunted children maintain blood glucose levels through increased glycogenolysis and gluconeogenesis, using amino acids and glycerol as glucose precursors. Energy is prioritized for vital organs such as the brain, heart, and immune system, resulting in reduced availability for new tissue synthesis, cell proliferation, and bone growth. The long-term impacts of stunting include decreased cognitive and physical development, low productive capacity, poor health, and an increased risk of metabolic diseases such as diabetes. Nutritional interventions in children under 5 years of age are an important factor in overcoming stunting, by increasing macronutrients as substrates for tissue and energy formation, and micronutrients as cofactors for metabolic enzymes, supporting energy production, enzymatic activity, and growth hormone regulation for optimal linear growth.

b. Vitamin B1/Thiamine

Vitamins are essential micronutrients that cannot be synthesized by the body and must therefore be obtained from food. They are needed in small amounts because most vitamins, such as vitamin B, function as cofactors for metabolic enzymes. Excess vitamins are generally excreted through the excretory system, while deficiencies cause a decline in biological functions such as intellectual and nervous system development. Vitamin B1 or thiamine has a pyrimidine ring structure and a thiazolium ring joined by a methylene bridge, with the molecular formula C12H17N4OS+, a molecular weight of 265.36 g/mol, soluble in water, insoluble in alcohol, and decomposes when heated. Sources of vitamins are found in grains, nuts, rice, wheat, lean meat, liver, organ meats, poultry, eggs, and fish, with a recommended daily intake of 0.5 mg per 1000 kcal.

Thiamine was first linked to beriberi in the 1880s by Takaki. Eijkman then discovered an antiberiberi factor in rice, crystallized it in 1926, synthesized it in 1936 by Williams and named it thiamine, and Peters demonstrated its role in glucose metabolism through the effects of catatorulin. Thiamine plays a role in brain enzymes, especially the production of acetylcholine, in the form of a cation (T^+) at physiological pH that affects nerve signals, neuronal transmission, cerebellum and spinal cord activity, and tissue repair. T^+ is phosphorylated to thiamin monophosphate (TMP) as a reserve. Thiamine also plays a role in the immune system through the regulation of immune cells, immunoglobulin expression, inflammatory responses, antioxidant effects, suppression of NF-κB, and tumor growth, and as an anti-inflammatory agent preventing recurrent inflammation, affecting the p53 protein, and functioning in immune activation in plants and yeast. Thiamine as a water-soluble vitamin is found in abundance in nuts, seafood, meat, and whole grains. Absorption begins with food digested by digestive enzymes into a cofactor or vitamin B1 binding protein, then released by pancreatic and intestinal enzymes, dissolved in water, and entered intestinal cells through the ThTr1 transporter. Thiamine functions as a coenzyme in the form of TDP in the metabolism of carbohydrates and branched chain amino acids, together with important Mg^{2+} ions in the dehydrogenase complex for the oxidation of a-keto acids and the formation of a-ketol between hexose and pentose phosphate catalyzed by transketolase which is important in the formation of ATP.

c. Thiamine Binding Protein

Binding proteins are specific proteins that interact with other molecules through specific binding sites, functioning to bind substances such as vitamins, hormones, or ions and transport them through body fluids because many substances are hydrophobic. Binding proteins maintain the biological availability of substances, regulate stability during transport, protect against enzymatic degradation, and control the active concentration of substances in target tissues. In medical biotechnology, binding proteins play an important role in therapeutic and diagnostic applications, with diagnostic advantages such as being non-invasive, rapid, sensitive, can be used for early screening, and is suitable for personalized diagnostics.

Thiamine Binding Proteins (TBP) are specific proteins with a molecular weight of approximately 103.81 kDa that function to bind thiamine, found in monocotyledonous plants such as corn and rice, dicotyledonous plants such as buckwheat, and gymnosperms such as pine and ginkgo which are rich in thiamine. The interaction of thiamine with binding proteins is in the form of association, namely the combination of proteins with their ligands without permanently changing the chemical structure of thiamine, but locking thiamine in a stable conformation for protection and storage. PPT functions in storage, transport, protection, regulation, and specificity of thiamine interactions at its source, with the first function as thiamine storage during seed dormancy so that it is not degraded or oxidized, and when germination begins thiamine is released from PPT for the initial energy metabolism of the plant.

d. Enzyme Labeled Protein Ligand Assay (ELPLA)

Enzyme Labeled Protein Linked Assay (ELPLA) is a method analogous to Enzyme Linked Immunosorbent Assay (ELISA), with almost the same principle. While ELISA uses antibodies, ELPLA uses specific binding proteins that bind thiamine from its ligand. ELPLA was first discovered by Gunarti et al. with a protein in the form of Thiamine Binding Protein and its liga d in the form of thiamine, and is used to measure thiamine levels in biological fluids such as serum and plasma. The working principle of ELPLA is analogous to indirect ELISA, where the antigen is adsorbed onto the plate and detection is carried out in two steps. In ELPLA, a specific binding protein is used to bind thiamine, followed by enzyme labeling and color formation which is measured spectrophotometrically. Validation of the ELPLA method was carried out by Gunarti et al. on the serum of normal individuals, patients with type 2 diabetes mellitus, and individuals with a history of chronic alcohol consumption by comparing it with a commercial ELISA and High Performance Liquid Chromatography (HPLC) as the gold standard. The results showed that ELPLA was able to detect significant differences between groups, where the serum thiamine levels of patients with diabetes mellitus and alcoholic individuals were much lower than those of healthy individuals ($p < 0.05$).

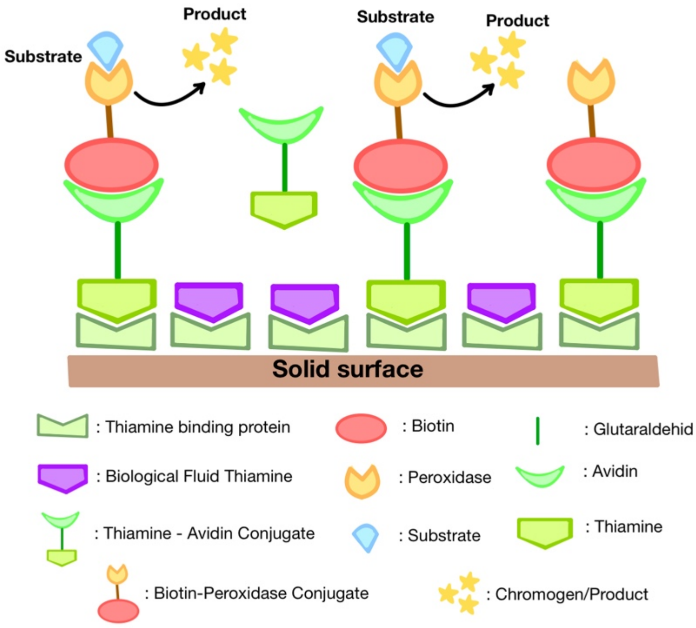

Figure: ELPLA Method Scheme

Theoretical Framework

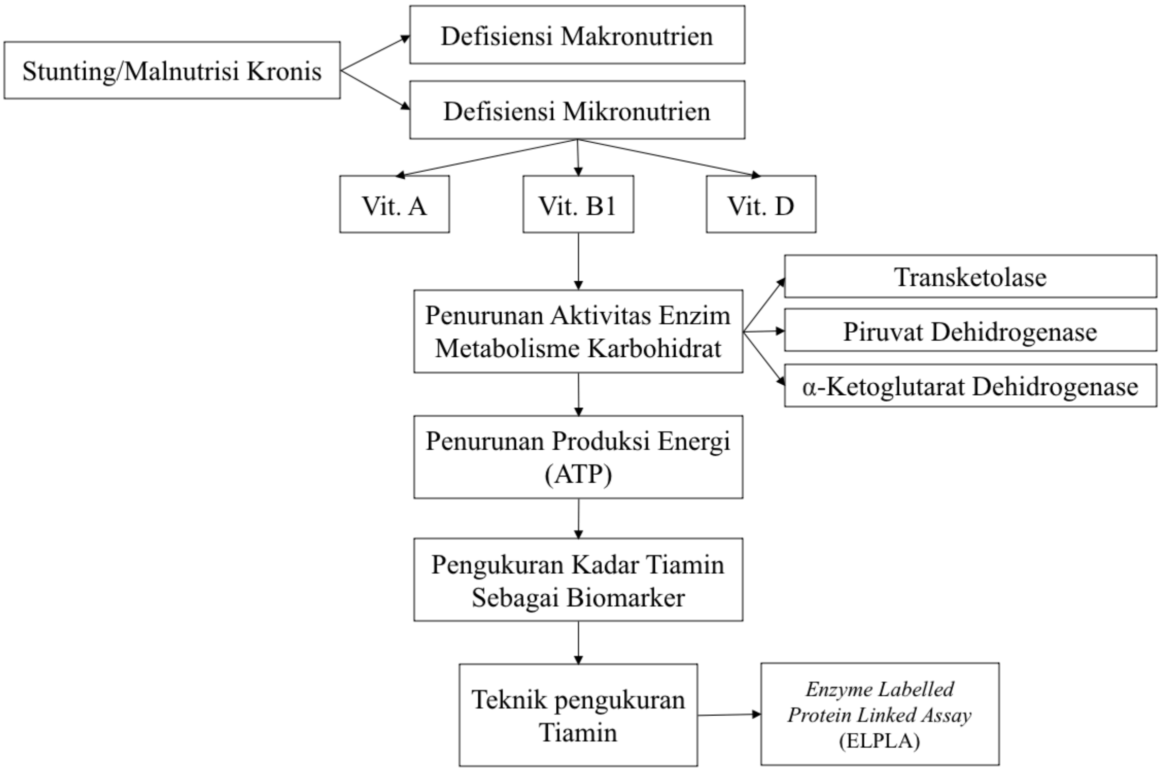

Image: Theoretical Framework

METHOD

This research method includes the fields of medical biochemistry and nutritional science that examines the effect of nutritional intervention in the form of eggs on thiamine levels in the serum of stunted children aged 2–5 years. Thiamine levels were tested using **the Enzyme-Linked Protein Labeled Assay (ELPLA) method** which is analogous to ELISA. The research design was quasi-experimental by comparing pre/before intervention and post/after intervention groups, and compared with a normal group as a control. The population included stunted children and healthy children aged 2–5 years in Buton Regency, Southeast Sulawesi. The sample was selected with the following inclusion criteria: children aged 2–5 years, diagnosed with stunting with HAZ <-2SD, registered at the Siontapina Community Health Center, guardians of children who can read, write, cooperate, and sign *informed consent* . The independent variable is categorical and the dependent variable is numeric, so the sample calculation uses the hypothesis test formula for the difference in the means of two groups. The data collected were the serum of stunted children before and after the intervention, the serum of normal children, and thiamine levels from the three groups.

Data collection was carried out by taking 3 mL of venous blood in the cubital fossa, centrifuged at 2000 rpm for 3 minutes to obtain plasma, stored at -80°C, then transported to the FKUI Laboratory. Measurement of thiamine levels was carried out using the ELPLA method using a 96-well plate coated with thiamine-binding protein isolated from mung beans, blocking with 1% BSA, adding sample plasma, thiamine-avidin and biotin-peroxidase conjugates, adding TMB/chromogen, giving a stop solution of H2SO4, and reading absorbance at a wavelength of 450 nm. Data were checked for errors, the mean of the duplicate absorption results was recalculated, a standard curve was created, and then input into SPSS. Analysis was performed using SPSS version 26 with descriptive univariate analysis. Numerical variables were tested for normality using the Shapiro-Wilk test, with $p>0.05$ presented as the mean and standard deviation. $P<0.05$ presented as the median and minimum–maximum range. Bivariate analysis, which examined the relationship between groups, used the t-test if the distribution was normal, and *the Wilcoxon* and *Mann-Whitney tests* if the distribution was not normal.

RESULTS AND DISCUSSION

Preparation and testing of the ELPLA method

This is done by coating a 96-well plate using Thiamine Binding Protein that has been isolated and purified from mung beans, then blocking with 1% BSA. After that, the well plate can be directly used for testing biological fluids such as blood plasma. A thiamine standard curve is made with a series of dilutions of thiamine solution of 20 µg/mL, 10 µg/mL, 5 µg/mL, 2.5 µg/mL and measured at a wavelength of 450 nm. A straight line equation is obtained $y = -0.0461x + 0.392$ with an R2 value of 0.9661. Based on this straight line equation, the measurement of thiamine levels from the results of plasma absorbance testing using the ELPLA method can be determined.

4.2 Thiamine Level Testing Using the ELPLA Technique

Plasma that has been measured by the ELPLA method at a wavelength of 450 nm is then calculated using a thiamine standard curve. Based on the calculation results, for the group of stunted children before the intervention, the mean thiamine level was 1.057 µg/mL (n=21) and SD was 1.952. Then, for the group of stunted children after the intervention, the mean thiamine level was 3.761 µg/mL (n=21) and SD was 1.513. Finally, for the group of healthy children, the mean thiamine level was 6.326 µg/mL with SD 0.569.

4.3 Test of the Relationship between Thiamine Levels in the Stunting Group and the Normal Group

Based on the normality test with the *Shapiro-Wilk test* , the significance of the pre-intervention group was 0.567 ($p>0.05$), the post-intervention group was 0.327 ($p>0.05$), and the normal group was 0.02 ($p<0.05$), where the pre- and post-intervention groups were considered normally distributed while the normal group was considered abnormally distributed. Therefore, to determine the relationship between the pre- and post-intervention groups with eggs, *a paired t-test was performed* . Meanwhile, to determine the relationship between the stunting group and the healthy group and the group with stunting after the intervention and the healthy group, a *Mann-Whitney test was performed* .

4.3.1 Comparison of thiamine levels between before and after intervention in stunted children

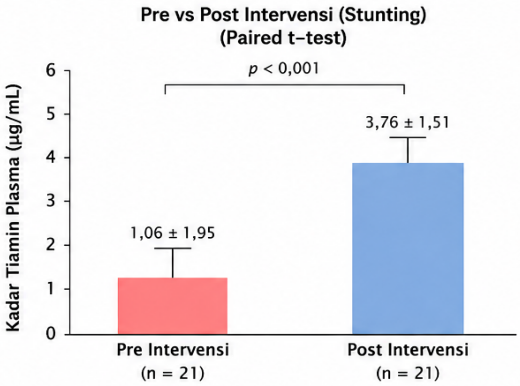

Figure 4.2 1Comparison Graph of Pre and Post Intervention

Paired t-test results on 21 stunted children showed a significant difference between serum thiamine levels before and after the intervention ($t = -5.644$; $p < 0.001$). The average plasma thiamine level increased from 1.06 ± 1.95 to 3.76 ± 1.51 (by 2.70 units) after the intervention. These results indicate that the intervention was effective in increasing plasma thiamine levels in stunted children.

4.3.2 Comparison of thiamine levels between stunted children and healthy children

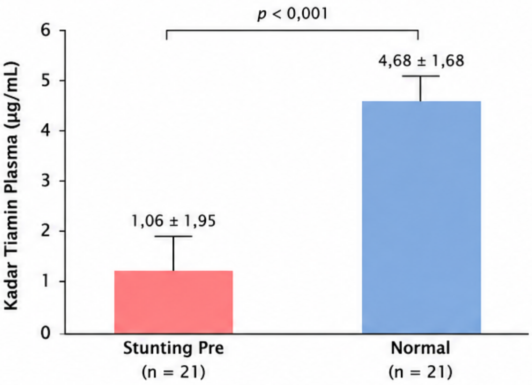

Figure 2. Comparison Chart of Stunted Children with Healthy Children

Mann–Whitney test showed a significant difference in plasma thiamine levels between stunted children before the intervention and healthy children ($U = 0.000$; $Z = -5.547$; $p < 0.001$). The mean serum thiamine level in stunted children before the intervention was 1.06 ± 1.95 µg/mL, while in normal children it was 6.33 ± 0.57 µg/mL. These results indicate that the serum thiamine levels of stunted children before the intervention were significantly lower than those of normal children.

4.3.3 Comparison of thiamine levels between stunted children after intervention and healthy children

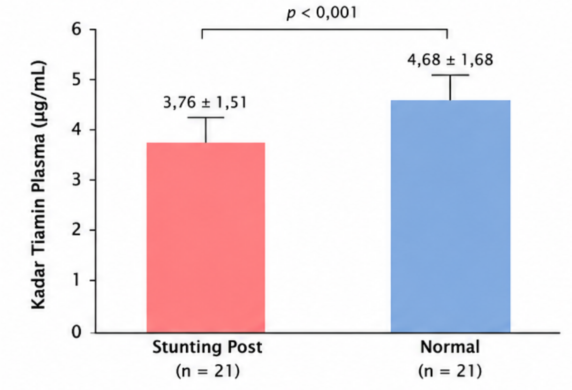

Figure 3. Comparison Chart of Stunting Children After Intervention with Healthy Children

Mann–Whitney test showed a significant difference in serum thiamine levels between stunted children after the intervention and children with normal weight (U = 14.500; Z = -5.183; $p < 0.001$). The mean serum thiamine level in stunted children after the intervention was 3.76 ± 1.51 µg/mL, while in normal children it was 6.33 ± 0.57 µg/mL. Although there was an increase in serum thiamine levels after the intervention, the value was still lower than the normal group so that the difference between the two groups remained statistically significant.

DISCUSSION

Thiamine Levels of Research Subjects

The serum thiamine levels of stunted children before intervention were significantly lower than those of normal children. The mean thiamine level in the stunted group was 1.06 ± 1.95 µg/mL, while in the normal group it was 6.33 ± 0.57 µg/mL. The Mann–Whitney test showed a significant difference (U = 0.000; Z = -5.547; $p < 0.001$). These findings align with previous research that found stunted children have lower micronutrient status due to poor food quality and quantity, high nutrient requirements, and recurrent infections. Micronutrient deficiencies often occur in conjunction with other nutrient deficiencies. Differences in thiamine levels indicate that chronic malnutrition in stunting affects not only linear growth but also micronutrient status. Low serum thiamine levels indicate an imbalance between nutrient needs and availability, making micronutrient status assessment crucial in stunting management, particularly in toddlers.

Interventional Thiamine Level Changes

The results showed that a one-month nutritional intervention in the form of eggs significantly increased serum thiamine levels in stunted children. Mean thiamine levels increased from 1.06 ± 1.95 µg/mL to 3.76 ± 1.51 µg/mL, with a significant paired t-test result (t = -5.644; $p < 0.001$). Increased thiamine levels are associated with improved dietary quality. Eggs, a nutrient-dense animal food, contain thiamine, quality protein, B-complex vitamins, choline, and essential minerals that support metabolism and growth. Improved nutritional status also improves gastrointestinal function and nutrient absorption efficiency, resulting in optimal nutrient utilization. Despite significant improvements, serum thiamine levels in stunted children after intervention remained lower than those in normal children. This suggests that recovery of micronutrient status requires more time, as chronic metabolic adaptation, inflammation, recurrent infections, and other micronutrient deficiencies continue to contribute to nutritional disparities.

Implications of Increased Thiamine Levels in Serum

Increased serum thiamine levels after intervention have important implications for the energy metabolism of stunted children. Thiamine is a precursor of thiamine pyrophosphate (TPP), the active form of vitamin B1, which functions as a cofactor for carbohydrate metabolism enzymes such as pyruvate dehydrogenase, α-ketoglutarate dehydrogenase, branched-chain α-ketoacid dehydrogenase, and transketolase. Increased thiamine levels reflect improvements in the body's metabolic capacity to utilize nutrients, supporting growth and physiological functions impaired by chronic malnutrition.

In thiamine deficiency, the activity of TPP-dependent enzymes decreases, resulting in inefficient glucose utilization, reduced cellular energy production, and impaired ATP-requiring biosynthesis. This is crucial for stunted children because energy needs remain high despite limited nutritional supply. Increasing serum thiamine levels has the potential to improve the efficiency of energy metabolism. In addition to ATP production, thiamine plays a role in the pentose phosphate pathway through transketolase, which generates NADPH for redox balance and protection against oxidative stress. In stunted children, oxidative stress and chronic inflammation contribute to impaired growth. Therefore, increasing thiamine status supports energy production, antioxidant defense mechanisms, and optimal cellular function.

The Relationship of Increased Thiamine to Stunting

Stunting is a condition of growth failure due to chronic malnutrition, characterized by impaired linear growth and metabolic changes as a result of adaptation to nutrient limitations. The body's metabolic rate and anabolic processes are reduced, hindering protein synthesis, new tissue formation, and bone and muscle growth. One mechanism involved is decreased activity of the mTOR pathway, a key regulator of protein synthesis and cell growth. Thiamine acts as a cofactor for energy metabolism enzymes, and its deficiency reduces acetyl-CoA formation and ATP production, thus reducing the efficiency of energy metabolism. This impairment has the potential to exacerbate the decline in mTOR activity, limiting protein synthesis, new tissue formation, and linear growth. Increased serum thiamine levels after intervention indicate improvements in key components of energy metabolism. Improved thiamine status increases cellular energy availability, supports anabolic processes, and possibly improves the function of the GH–IGF-1 axis, which is often impaired in stunted children. This improves tissue responsiveness to growth signals, supporting cell proliferation, protein synthesis, and linear growth. Although the study did not directly evaluate height or growth biomarkers, increased serum thiamine levels indicate improved metabolic conditions that could potentially support growth. Therefore, improved thiamine status may be a biological mechanism contributing to the metabolic capacity of stunted children and supporting catch-up growth when accompanied by adequate and sustained nutritional interventions.

CONCLUSION

This study showed that a one-month nutritional intervention using eggs in stunted children aged 24–59 months was associated with a statistically significant increase in serum thiamine levels. The mean serum thiamine level increased from 1.06 ± 1.95 µg/mL before the intervention to 3.76 ± 1.51 µg/mL after the intervention ($p < 0.001$). Furthermore, serum thiamine levels in stunted children before the intervention were lower than in normal children (1.06 ± 1.95 µg/mL vs. 6.33 ± 0.57 µg/mL; $p < 0.001$). Despite the increase after the intervention, serum thiamine levels in stunted children remained lower than in the normal group ($p < 0.001$). These results suggest that egg consumption for 30 days has the potential to improve micronutrient status, particularly thiamine, in stunted children. As a vitamin that acts as a precursor to thiamine pyrophosphate (TPP), thiamine is an essential cofactor for various enzymes involved in energy metabolism, including pyruvate dehydrogenase, α-ketoglutarate dehydrogenase, branched-chain α-ketoacid dehydrogenase, and transketolase. Therefore, increased serum thiamine levels after the intervention indicate an improvement in one of the essential components supporting energy metabolism and physiological functions required during growth. However, the increase in serum thiamine levels obtained was not sufficient to restore stunted children's thiamine levels to those of normal children. This suggests that improving the micronutrient status of stunted children likely requires longer and more comprehensive interventions. Therefore, it can be concluded that egg consumption has the potential to be a simple, accessible, and relatively affordable nutritional strategy to help improve thiamine status in stunted children.

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