Prevention of Micronutrient Deficiencies in Young Children: Consensus Statement from Infant and Young Child Feeding Chapter of Indian Academy of Pediatrics

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Justification: Micronutrient deficiencies have significant impact on the overall health and well-being of society and potential targets for supplementations. It is important to formulate a consensus statement in view of current evidence, and put in place strategies to meet targets. **Objectives:** To formulate by endorsement or adoption and disseminate a consensus statement for prevention of micronutrients deficiencies in young children for office practices from an Indian perspective. **Process:** A National Consultative Meeting was convened by Infant and Young Child Feeding Chapter (IYCF) of Indian Academy of Pediatrics (IAP) on 17 December, 2016 at Mumbai. IYCF chapter, IAP, United Nations Children Fund, National Institute of Nutrition and Government of India were the participating agencies; and participants representing different parts of India were included. **Conclusions**: Micronutrient deficiencies are widespread. For its prevention proper maternal and infant-young child feeding strategies need to be practiced. Encourage delayed cord clamping, dietary diversification, germinated foods, soaking and fermentation processes. Existing Iron, Vitamin A, Zinc supplementation and universal salt iodization programs need to be scaled up, especially in high risk groups. Universal vitamin D supplementation need to be in place; though, the dose needs more research. Vitamin B_{12} deficiency screening and supplementation should be practiced only in high-risk groups. Availability of appropriately fortified foods needs to be addressed urgently.

Keywords: *Dietary diversification, Food fortification, Trace elements, Sustainable developmental goals, Multiple micronutrient powder.*

Micronutrients (vitamins and trace elements) are needed in amounts <100 mg/day and are crucial in development, production and functioning of enzymes (Zinc, Copper, Manganese, Selenium, Magnesium, Molybednum); hormones (Iodins, Chromium) and growth regulator proteins; reproductive and immune system; bone and membrane structure (Calcium, Phosphorus, Magnesium, Vitamin D); oxygen binding (Iron), etc.

Micronutrient deficiencies ('hidden hunger') are highly prevalent and affect far beyond the known effects like anemia, goiter, asymptomatic to devastating, often hard to recognize, mimic many diseases, have fewer signs but gamut of symptoms, and can involve multiple system. Only few have practicable laboratory diagnosis. Hence they need high index of suspicion and a detailed dietary history for diagnosis. It has potential to affect economic and overall development, as affected populations are unable to achieve full mental and physical potentials, have low work capacity, and are prone to infections [1].

Global health risk estimates in low income countries reveal 7% deaths and 10% total disease burden in children attributable to underweight, micronutrient deficiencies (especially iron, vitamin A and zinc) and suboptimal breastfeeding triage, almost equivalent to entire disease and injury burden of high-income countries [2]! National Nutrition Monitoring Bureau (NNMB) report reveals high micronutrient deficiency in rural population of major States. National nutrition program in the past have failed to achieve the five year plan goals of Government of India (GOI) [3]. Currently, apart from programatic approach by GOI, with Iron-folic acid, Universal salt iodization (USI), Zinc in diarrhea management, and vitamin A supplementation; Food Safety and Standards Authority of India (FSSAI) standards of fortification of foods with iron, zinc, iodine, vitamin A and D, vitamin B_{12} and other B-vitamins ensure micronutrient supply [4,5].

Objectives: Increasing awareness and information mandates need of consensus statement on micronutrient

supplementation in making informed decisions on the appropriate nutrition actions; to achieve the Sustainable Development Goals (SDGs) and the global targets set in the Comprehensive Implementation Plan on Maternal, Infant and Young Child Nutrition and the Global Strategy for Women's, Children's, and Adolescents' Health.

PROCESS

A National Consultative Meeting was convened by Infant and Young Child Feeding Chapter (IYCF) of Indian Academy of Pediatrics (IAP) on 17 December, 2016 at Mumbai. IYCF chapter, IAP, United Nations Children Fund, National Institute of Nutrition and Government of India were the participating agencies; and participants representing different parts of India were included.

Methods of search: Cochrane database, e-Library of Evidence for Nutrition Actions (eLENA), MEDLINE through Pubmed, and Google Scholar were searched with preference to recent systematic reviews, using combination of key words "iron (and other micronutrients) status, India, complementary feeding, children, deficiency and supplementation" and further expanded through "related articles" and reference lists of the articles.

RECOMMENDATIONS

Iron Deficiency

Clinical presentations: Apart from anemia, iron deficient young children are vulnerable to socio-emotional behavior issues; irreversible effects on psychomotor skills and cognition and later attention deficits. Presentation with pagophagia, dysphagia, decreased effort tolerance, pica, cold intolerance, altered immunity or cerebral vein thrombosis is known.

Deficiency status and Risk factors: Irrespective of age, geography and socioeconomic status, iron deficiency is still high, underestimated, under-treated and the commonest cause of disability in children [2].

Dietary factors which may result in decreased iron absorption and ultimately iron-deficiency include high phytates (cereals-legumes, roots-tubers, maize, beans, whole wheat flour and sorghum), low ascorbic acid (fruits-vegetables); high animal milk intake; regular tea-coffee with major meals; low consumption of iron supplementation; and low consumption of animal origin foods (meat, fish and poultry) [6]. Cow's whole milk is a risk factor due to low iron content, poor bioavailability due to high casein and calcium; and increased loss of blood in intestines [7]. However, authors do not suggest exclusion of these from the diet for improving iron status. Parental dietary history per se does not qualify as first stage screening tool for iron deficiency state [8].

Additional risk factors are poor maternal stores; prematurity or low birth weight; exclusive breastfeeding beyond 6 month without iron rich/fortified foods or supplements; worm infestations (hookworm, ascaris and schistosomiasis); low socioeconomic status; migrant worker parents; bottle feeding; and a mother who is currently pregnant [9,10]. Greater than 95th percentile weight and height, and obesity are emerging risk factors for iron-deficiency [11].

Screening

Iron-deficiency and iron deficiency anemia (IDA) are incorrectly used synonyms. In healthy appearing infants, anemia is neither a sensitive nor a specific screen for iron-deficiency [12], except for severe cases. In view of high prevalence of iron deficiency, we should have a high index of suspicion even in presence of normal hemoglobin level. Hemoglobin levels as surrogate marker of IDA underestimates iron-deficiency in up to 12-27% [13]. Red cell distribution width should be seen as it is the earliest marker of iron deficiency. It is recommended to treat children with subclinical iron deficiency even in absence of anemia. Serum ferritin <12 ng/mL is sensitive, with high false negative rates being common as it is a acute phase reactant. Transferrin receptor1 and Total iron binding capacity (TIBC) are good to establish iron-deficiency, especially in cases without anemia [14]. Usually a combination of tests is used to diagnose iron deficiency for certain. A cost-effective strategy is a therapeutic trial [15].

For asymptomatic and not at risk children aged 6 to 24 months undergoing primary preventive actions, the current evidence is insufficient to recommend routine screening for IDA [16]. Hemoglobin is the only practical screening test in field settings. All 6 to 36 month children without primary preventive actions should be screened at 9 to 12 months, 6 months later and at 2 year age [17]. At prevalence of anemia <5%, screening is not fruitful as majority of cases are unrelated to iron-deficiency. Screening for programatic purposes should be considered where anemia prevalence is between 5-20% and whole of India comes under this category [18].

Recommended Interventions

Diet: Beyond the age of 6 months, more than 90% of the iron requirements of a breast-fed infant must be met by complementary food rich in bio-available iron [6]. Dietary diversification must be encouraged. Vxegans should be monitored closely and treated early. It is advisable to avoid consumption of beverages like tea and coffee with food as tannin contained in these may interfere with iron absorption. Foods containing ascorbic acid may enhance iron absorption.

Infants with IDA should be screened for cow's milk protein allergy. Data from Western countries suggest that early introduction of cow's milk is associated with increased gastrointestinal blood loss [7]. In the absence of Indian data, we do not recommend avoiding whole cow's milk after 6 months age.

Cooking in cast iron vessels: Cooking of soups containing vegetables of low pH by simmering (heating for a long below boiling point) in cast iron vessels helps in increasing iron intake. This practice should be encouraged. Frying in iron vessels does not usually have similar effect [19].

Food fortification: It is difficult to meet full iron requirements in young children through diet without fortifying complementary feeds or iron supplements. As a public health measure, food fortification can play a major role in decreasing the prevalence of iron deficiency. Except for wheat flour or rice in some state government distribution systems, iron fortified foods are uncommon in India. Multiple Micronutrient Powder (MMNP) fortification should be considered in high-risk settings where above interventions are difficult to implement [20]. Fortification and supplementation together might breach the tolerable upper limit (TUL) for iron intake [21]; though, the clinical significance of this is not clear.

Iron supplementation

a) *In infants >6 month age (Public health measure guidelines):* Iron supplementation should be given to children aged 6-60 months in the dose of 10-30 mg /day, three months a year wherever prevalence of anemia is >20% [Strong recommendation, moderate quality evidence]. This comes to about 1-2 mg/kg/day [22] as most of India has >40% prevalence of anemia.

National Health Mission (NHM) guidelines [23] recommend bi-weekly 100 doses/year of 20 mg Fe + 100 mcg FA supplementation in 6-60 months age as syrup. Iron should be withheld in acute illness (fever, acute diarrhea, pneumonia, etc.), severe acute malnutrition (SAM) and hemoglobinopathy or history of repeated blood transfusion. In malaria endemic areas, public health measures to manage malaria must be in place [Strong recommendation]. Folic acid should not be used in malaria endemic areas where antifolate malaria medications are used [24].

b) In infants <6 month age (Individual case based supplementation): Low birth weight [LBW] babies should be supplemented with iron 2-3 mg/kg/day, beginning from 2 weeks for babies with birthweight <1500 g, and 6-8 weeks for babies with birthweight >1500 g [25], till toddlerhood when diet meets the iron requirements. Babies who received multiple transfusions during neonatal care should be clinically and biochemically assessed for need of supplementation at 6-8 weeks [14].

Since large number of term babies (21.4% at 4 months and 36.4% at 5 months) [26] suffer from iron deficiency, it is recommended that iron supplementation be started at 4 months in exclusively breastfed babies, especially where there is high risk of low iron transfer from mothers suffering from malnutrition, anemia, hypertension (with growth retardation) and diabetes.

Ancillary measures

- Promote delayed cord clamping as it helps to improve iron store in newborns [27].
- Since the prevalence of worm infestation in various parts of our country high, we recommend twice a year universal deworming in the dose of 400 mg for children above two years and 200 mg for children 1-2 years [23,28].
- *Behavior change:* Hand washing, prompt malaria treatment, diarrhea management and nutrition education also have an important role to play. WASH (Water, sanitation and hygiene) program, implemented well, will go a long way to achieve the required behavioral change.

Zinc Deficiency

Clinical presentations: There are no pathognomonic features for zinc deficiency except in acrodermatitis enteropathica. Zinc deficiency presents as growth failure, hypogonadism, skin lesions, impaired olfactory and gustatory sense, and impaired resistance to infection. It is pronounced in protein-energy malnutrition, Crohn's disease, sickle cell anemia and nephrotic syndrome [29].

Deficiency status and risk factors: About 43.8% under-five children in five Indian states have significant zinc deficiency [30]. Zinc is not well conserved in body as there is no conventional tissue reserve. Its status depends on regular dietary zinc intake. Low intake of zinc rich foods (animal or sea products); high intake of inhibitors (phytates) and losses in diarrhea contribute to widespread zinc deficiency.

Screening

Laboratory markers are inadequate for practical use due to cost and methodological obstacles even in developed countries, so indirect method of estimating zinc status of diets in various geographic areas are used [31].

Prevalence >20% is indication for public health intervention. Low height for age >20% is a surrogate indicator [32].

Recommended Interventions

Diet: In view of high prevalence of zinc deficiency in population, we recommend food rich in zinc (additional milk, eggs, grains, legumes, nuts and seeds).

Food fortification: Fortification with zinc only has shown to improve serum zinc status [33]. We recommend it to control/eliminate zinc deficiency despite lack of unequivocal evidence on benefits. It is recommended that zinc fortified foods be available.

Adjunct in therapy: Zinc should be prescribed as adjunct therapy for diarrhea as India has high prevalence of zinc deficiency and also malnourished children [34]. The dose recommendation is 10 mg/day for babies below six months and 20 mg/day for babies above six months, using any water soluble zinc salt [35]. In view of vomiting seen with zinc administration we recommend that it can be administered in two divided doses. Zinc should be prescribed as adjunct therapy for conditions like sickle cell disease, preterm babies, protein energy malnutrition, chronic diarrhea, Wilson disease, Thalassemia major.

Supplementation: Zinc supplementation does not have significant impact on all-cause mortality but prevents pneumonia and diarrhea significantly [36-38]. Zinc supplementation in deficient pre-pubertal children shows a significant increment in height and weight, but not weight-for-height, when there was low weight for age and height-for-age [39]. Preterm babies are recommended 2 mg/kg/day supplemental zinc till 3 months corrected age [40]. We recommend co-administration of zinc and iron as it is equally effective [39], contrary to popular belief, for ease of administration.

Iodine Deficiency

Clinical presentations: Iodine deficiency disorders (IDD) presents as goiter, cretinism, hypothyroidism, brain damage, abortion, still birth, mental retardation, psychomotor defects, hearing-speech impairment or neuropsychological deficits as subclinical manifestation. It constitutes the largest cause of preventable brain damage worldwide. Children from iodine-deficient areas have lower intelligence quotient by average 10-15 points. Majority of consequences of IDD are invisible and irreversible, but preventable.

Deficiency status and risk factors: WHO estimates a worldwide 37% prevalence of iodine deficiency in school-aged children. The IDD control goal was prevalence <10% in India by 2012 but 325 districts surveyed revealed 263 as endemic [41]. Smoking reduces iodine in breastmilk and needs consideration for supplementation [29].

Screening

Median urinary iodine >100 µg/L indicates sufficiency. Ultrasound measurements of thyroid volume are better than clinical assessment. Filter paper TSH test is recommended for neonatal screening. However, its role and cost effectiveness in screening for community iodine deficiency is not established. Filter paper Thyroglobulin (Tg) test is a promising method [42].

Recommended Interventions

Universal Salt Iodization: USI is the most cost effective and sustainable method of iodine supplementation for controlling IDD; we support a ban on availability of non-iodized salt. Iodine is a volatile compound hence the iodized salt should be stored in air-tight containers. Since method of cooking and duration of cooking affect iodine salt content of cooked food, it is advisable to sprinkle salt after cooking or towards end of cooking, wherever possible [43].

In areas of moderate and severe iodine deficiency (median urinary iodine <50 µg/L or total goiter rate >20%) approaches for iodine supplements are described in ***Table* I** [44].

A high iodine intake with urinary levels >300 µg/L is to be discouraged, especially in previously deficient populations as it can have an adverse effect of iodine induced hyperthyroidism [42].

Vitamin A Deficiency

Vitamin A deficiency (VAD) is most important preventable cause of blindness in low- and middle-income countries.

Deficiency status and risk factors: Zinc, Iron and protein-calorie deficiencies; recurrent clinical and

TABLE I WHO-RECOMMENDED DOSAGES OF DAILY AND ANNUAL IODINE SUPPLEMENTATION

Population group	*Daily dose of iodine (µg/d)*	*Single dose of iodized oil (mg/y)*
Pregnant women	250	400
Lactating women	250	400
Women 15-49 year	150	400
Children <2 year*#	90	200

**For exclusively breastfed 0-6 month child, lactating mother receives supplementation as above; #For situations where complementary food fortified with iodine is not available.*

subclinical infections; and parasitic infestations, adversely affect vitamin A absorption, transport and utilization. Habitual low dietary intake of vitamin A rich animal food or beta carotene-rich vegetables-fruits is the major factor for the poor vitamin A status among the South East Asia Region (SEAR) population. >0.5% Bitot's spots, >1% night blindness and >0.01% keratomalacia prevalence among under-five children indicates a public health issue.

The NNMB 2006 data from rural India shows 62% prevalence of VAD Disorders (Serum retinol <20 µg/L) in preschoolers [45]. Despite non-significant improvement in dietary intake and vitamin A program coverage, there is decline in clinical VAD in under-5 children in most/ countries of SEAR. Keratomalacia is no longer a major public health problem with improved health care, nutrition and measles vaccination, although cases are reported from remote areas [46]. Reappraisal of the prevalence of VAD is warranted at present [47].

Screening

Serum retinol, dark adaptometry and Rose-Bengal eye test are useful in detecting VAD. Serum retinol measurements alone, if not adjusted by C-reactive protein (CRP) levels for subclinical infections, can overestimate VAD burden. It may not be an operationally feasible indicator for community use [47].

Recommended Interventions

Diet: Pediatricians should provide guidance to promote vitamin A rich foods routinely (milk products like butter, ghee, yogurt, curd, cheese, eggs, liver and yellow/ orange colored vegetables and fruits); more so during diarrhea, measles and respiratory infections [48].

Supplementation: Vitamin A prophylaxis program was started with a view to control blindness due to keratomalacia. Later in 2006, age group was extended to 5 years from initial 3 years. Every six months a mega dose of 2 lakh units (1 lakh for < 8 kg or < 1 year age) of oil based vitamin A is given [48]. Mega dose vitamin A supplementation is not recommended in infants below 6 months age.

Recent data suggests a sharp reduction in the incidence of overt vitamin A deficiency across the country [47], and therefore there is a need for a relook at this program. On basis of possible adverse effects, overdosing, increase in acute respiratory infections, vitamin D and zinc antagonism, and reducing prevalence of deficiency, there is an opinion to adopt a targeted rather than universal mega dose vitamin A supplementation in preschool children [47]. Indian Academy of Pediatrics recommends supplementation till 3 years of age to all, and to older children only in severe malnutrition and measles [49].

Mega dose vitamin A supplementation is recommended in children with severe acute malnutrition (SAM), measles and cholestasis; in addition to those with signs of deficiency like xerotic conjunctiva, Bitot's spots and keratomalacia. When water soluble injectable preparation is given, oral fat soluble preparation is recommended to replenish stores.

Vitamin A supplementation coverage rate has increased from 16% (NFHS-3, 2005) to 27%-90% in different states (NFHS-4, 2015-16) with a national average of 60.2%. The large dose is well absorbed and stored in the liver, and mobilized over 4-6 months depending on dietary content and utilization rate [50].

Transient side-effects usually disappearing within 24 hours, like headache, nausea, vomiting and diarrhea are reported in 3%-7%, with no long term consequences [50]. There are no known deaths. As overdosing can lead to hypervitaminosis A, special training of field staff is recommended. The supply is irregular and oral syrup is available only to the Government sector, hence many children do not get regular supplementation.

Effects of supplementation: A cochrane review in 2017 found that VA supplementation at 0-6 month does not significantly reduce overall, diarrheal or pneumonia related mortality and morbidity, but increases benign raised fontanel cases [51,52]. VA supplementation in 6-59 month children in low-and middle-income countries with a high prevalence of VAD has shown [50]:

(*a*) Reduced all-cause mortality by 12%-24% and diarrhea-related mortality risk by 12% but no difference in cause-specific mortality of measles, respiratory disease or meningitis.

(*b*) Reduced diarrhea and measles risk, but no effect on respiratory disease or hospitalizations for diarrhea or pneumonia.

(*c*) Significantly increased vomiting in 48 hours.

(*d*) Supplementation at 6 or 9 months did not affect measles vaccine seroconversion.

(*e*) No significant effect when the data were stratified by National child mortality rates.

Vitamin D Defeciency

Clinical presentations: Role of vitamin D in bone mineralization and calcium-phosphorus homeostasis is well established with deficiency manifesting as infantile

hypocalcemia, rickets, delayed growth and dentition. Lower levels of vitamin D and its binding proteins were seen in children with severe sepsis.

Deficiency status and risk factors: Based on serum levels, vitamin D deficiency is prevalent, largely subclinical, across the country from 70-100% at various times in life cycle, irrespective of gender, region or dietary habits [53]. In infants aged three months and their mothers, prevalence of vitamin D insufficiency was found in 92.6% and two third infants were exclusively breastfed [54]. Prevalence of VDD (serum 25-hydroxyvitamin D <25-30 nmol/L) >20% in whole population or in at-risk population subgroups constitutes a public health issue warranting intervention.

Infants depend upon the vitamin D transferred from mother prenatally. In every deficient mother, evaluate child for calcium and vitamin D and also *vice versa*. Most infants are born with low vitamin D stores and are dependent on breast milk (containing <25 IU/L), sunlight or supplements as vitamin D sources in initial months of life. Sun exposure may be restricted due to many reasons. Vitamin D deficiency is also prevalent among infants in countries with food fortification and year-round sun exposure.

Screening

Laboratories use different methods for assessment. Wide variation in reports on same sample are noted [55]. Based on the observations of relation with calcium absorption and parathormone levels with vitamin D levels most authorities consider >30 ng/ml as sufficient, 20-30 ng/ml as relative insufficiency and <20 ng/ml as deficiency.

Recommended Interventions

Supplementation: Vitamin D supplementation is recommended for children at risk of vitamin D deficiency, especially where sun exposure is not available or is avoided for some reasons.

Recent Cochrane reviews [56] and WHO [57] do not recommend routine supplementation of vitamin D to term infants for preventing rickets or respiratory and diarrheal infections. Cochrane has some evidences for vitamin D supplementation for asthma prevention [58].

There is no national program for prevention of VDD in India. FSSAI has issued recommendation [5] for fortification of vegetable oil with 25 IU/g vitamin A and 4.5 IU/g vitamin D, which cannot meet daily requirements. European Food Safety Authority has set the upper limit of safety at 1000 IU/day for infants and 2000 IU/day for children ages 1 to 10 years [59]. Considering variable concentrations of available preparations, it is imperative to monitor the supplementation to avoid hypervitaminosis D.

Sun exposure: Encourage the socially accepted practice of oil massage under sunlight and promote outdoor activities under sun for older children and adolescents. Skin is a more efficient source for providing vitamin D than ingested form despite slow initial rise in plasma levels [60]. There are no defined recommendations on sunlight exposure. Generally, face, arms, hand and legs should be exposed twice or thrice a week, for the duration causing minimal sunburn [61].

Diet: Encourage children to consume more vitamin D rich or fortified foods. Dietary sources are scarce like fatty fish (wild salmon, mackerel, eel, anchovy, sardines, swordfish, tuna), and lesser extent in egg yolk and fortified foods, milk and dairy products, margarine, *etc*. [59].

Vitamin B-Complex Deficiency

Clinical presentations: Deficiency of B-vitamins can lead to glossitis, angular stomatitis, dermatitis, anemia, hyper-pigmentation or brain dysfunction. Cobalamin stores are so large that clinical deficiency is uncommon without predisposing factors like malabsorptive states; it takes years of inadequate intake or absorption before clinical symptoms. Niacin deficiency is encountered only as epidemic during emergencies in population with maize as staple food and high incidence of infectious diseases and malnutrition; and children may not present with skin changes although diarrhea can occur [62].

Deficiency status and risk factors: Folic acid and cobalamin are relatively more studied than rest. Significant deficiency of vitamin B_{12} has been reported in exclusively breastfed <6 month infants and their mothers [63] and infants and preschoolers [64]. Vegan diets are risk factors as there are no plant sources of cobalamin. Maternal deficiency is the strongest predictor of low cobalamin in neonates. Continued low intake because of low content in mother's milk; delayed introduction of animal based complementary foods; prolonged breastfeeding in populations with food insecurity; and cultural and economic factors play determinant roles in promoting a deficient state in childhood [65].

In a biochemical study from Hyderabad in residential school with students from middle income families [66], Folic acid deficiency was present in almost all children, while deficiencies of B_2 and B_6, vitamin C, vitamin A and B_{12} were reported in 44%-66% of the children. B_1 and Zinc deficiency was less.

KEY MESSAGES

1. Improve nutritional status of pregnant women using supplements.
2. Practice delayed cord clamping.
3. Encourage breastfeeding including colostrum feeding.
4. Supplement lactating women.
5. Supplement children by programatic (vitamin A, iron-folic acid, and zinc), and Case-based (vitamins K, B12, D, B_1, B_3, B_6, E, and Multiple micronutrient powder) approaches
6. Control infectious disease (De-worming, malaria control)
7. Dietary strategies [70]**:**
   a. *Dietary diversification:* Enhance food with ascorbic acid (for iron), other organic acids, cellular animal protein (for iron and zinc), Fat (for retinol and provitamin-A carotenoids) by encouraging inclusion of fresh fruits (e.g., citrus fruits), vegetables (e.g., tomatoes, green leaves), legumes (e.g., ground nut flour) or small amounts of flesh foods (animal muscle, fatty fish, fish flesh with bones and fish flour) in food
   b. *Mild heat treatment* (like preparation of porridges) to releases bound carotenoids
   c. Home Food processing*:*
      i. Soaking (reduces phosphates and phytates)
      ii. Fermentation (improves B12, improves phytase activity)
      iii. Germination (increase endogenous phytase, reduce polyphenols and tannins in some legumes)
   d. *Use Staple food fortification* (iodized salt, flour, sugar, oils) & fortified complementary foods
   e. *Cook food in cast iron vessels* by simmering

Screening

Low serum B_{12} and folate levels are not final evidence of deficiency. True B_{12} tissue deficiency is present if serum methylmalonic acid (MMA) is high and for folic acid deficiency if homocysteine levels are high. However, B_{12} deficiency also causes rise of serum homocysteine. Levels of B_{12} between 200-300 pg/mL and folate between 3-4 ng/mL indicate deficiency [67]. In selected cases non nutritional deficiency should be ruled out, *e.g*. genetic metabolic pathways defects.

Recommended Interventions

Diet: Family education on balanced diet *i.e.* inclusion of food from vegetable and animal source should be provided. Promote consumption of foods rich in B-vitamins. A useful source is National Institute of Nutrition, Hyderabad manual, which lists the dietary sources and RDA of dietary components [68].

Supplementation: B_{12} deficiency is not considered as a public health problem in India. The current policy is folic acid supplementation as Fe-FA supplementation program. It is yet unresolved whether folic acid supplementation can be harmful in population groups with a high prevalence of B_{12} deficiency.

Current evidence supports use of B_{12} supplements in pregnant and lactating women in low socio-economic strata, and in vegetarian population with poor intake of animal source food [69,70] with 50 µg/day B_{12}, in addition to Iron and Folic acid presently practiced.

All children suffering from nutritional anemia should be prescribed iron, folic acid and vitamin B_{12}. Routine prescription of vitamin B complex with antibiotics is not recommended.

Fortification: WHO recommends point-of-use fortification of foods with MMNP consumed by 6-23 months children [20], and we suggest adding one RDA of vitamin B_{12} to it. It is suggested to prioritize research to add to the evidence of impact of multiple micronutrient (including B-vitamins) supplementation or fortification strategies on morbidity, developmental outcomes and mortality in Indian children.

Other Micronutrients

There is very little data on other micronutrients from India. It is felt that the research activity in this area be encouraged to collect evidence for recommendation.

Intramuscular vitamin K administration at birth is recommended.

Routine use of vitamin E for preterm babies is not recommended.

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Disclaimer: These consensus statements are prepared for assisting pediatricians in accordance with current scientific evidence and guidelines for prevention of micronutrient deficiencies in young children; however, many areas are still not clearly defined. These statements cannot establish a standard of care, and decisions about treatment should be based on the judgment of the Pediatricians on merits of individual cases dealt by them.
Revision and Updating: These guidelines were drafted in 2017 and updated in May- June 2018 through email suggestions by the EB members of Central IAP and the writing committee. The recommendations shall be revised after three years i.e. in 2020- 21.

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ANNEXURE I

Members of the National Consultative Meet

Dr RK Agrawal (President IYCF chapter), Dr Rajkishor Maheshwari, Dr Satish Tiwari, Dr Balraj Singh Yadav, Dr Ketan Bharadva, Dr Sanjay Prabhu, Dr KE Elizabeth, Dr K Raghunath, Dr. Nimisha Goel (From MoHFW Govt of India), Dr Urmila Deshmukh, Dr BR Thapa, Dr Sushma Malik, Dr Hima Bindu Singh, Dr Sudhir Mishra, Dr CM Chhajer, Dr Jayant Shah, Dr Rajinder Gulati, Dr Mallikarjuna, Dr Somasekara, Dr S. Laishram, Dr Kritika Malhotra.

Invited but could not attend: Dr Promod Jog (President IAP 2016), Dr Bhavneet Bharati, Dr Ajay Khera (From MoHFW Govt of India), Ms. Raji Nair (UNICEF), Dr. Radhika (NIN), Dr Kanya Mukhopadhyay, Dr Vishesh Kumar (WHO).