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Anaemia and Iron Nutrition in India: insights from recent research studies

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Introduction

Anaemia, characterized by red blood cell count and haemoglobin below the normal range, results in impaired oxygen carrying capacity[1]. Anaemia is negatively associated with physical work capacity of adolescents and adults, cognitive performance and physical growth in infants and children, immune status and morbidity from infections in all age groups[2-4]. Iron deficiency anaemia during pregnancy is associated with higher perinatal risks for mothers and overall infant mortality[5]. National Family Health Surveys (NFHS) 2 to 5 have shown the persistence of high prevalence of anaemia across all age groups in India (NFHS 2 1998-99; NFHS 3 2005-6; NFHS 4 2015-16 and NFHS 5 2019-21 Fig 1). The recent NFHS 5 data show that 52% of pregnant women, 57% of women of reproductive age (WRA), 23% of male adults (15-49 years age), and 67% of children under 5 years of age were anaemic[6].

Anaemia prevalence and anaemia control programs in India

Considering the adverse effects of anaemia on maternal and child health, the Government of India first initiated the National Nutritional Anaemia Prophylaxis Program (NNAPP) in 1970. This program focused mainly on providing iron and folic acid (IFA) tablets to the high-risk groups including pregnant women (after the first trimester), lactating women (100mg iron and 500 µg folic acid per day) and preschool children (1-5 years, 20mg iron and 100µg of folic acid per day) for 100 days[7]. This program was executed via paramedical staff in Primary Health Centres and sub-centres. This program was revised in 1991 and renamed as National Nutritional Anaemia Control Programme (NNACP), and the emphasis was shifted from anaemia prevention to screening women for anaemia and treating them with appropriate dose of iron and folic acid tablets and nutrition education on consumption of iron rich foods. In 2013, this programme was further revised to adopt a life cycle approach under the National Iron Plus Initiative (NIPI) of the Ministry of Health and Family Welfare (MoHFW), Government of India[8]. The interventions under NIPI included therapeutic and prophylactic supplementation with age-appropriate iron and folic acid (IFA) dosages for all age groups and facility-based treatment of severe anaemia. Apart from NIPI, there are other complementary programmes that address other causes of anaemia, such as National Deworming Day (NDD) for deworming, National Vector Borne Disease Control Program (NVBDCP) for malaria control, and special efforts to reach out to populations affected with haemoglobinopathies. The more recent National Health Policy (2017), as well as the National Nutrition Strategy (2017) of India are focused on intensifying efforts to address all causes of anaemia in order to accelerate the rate of decline in anaemia prevalence among all age groups in a mission mode, using a structured multi-pronged strategy rather than

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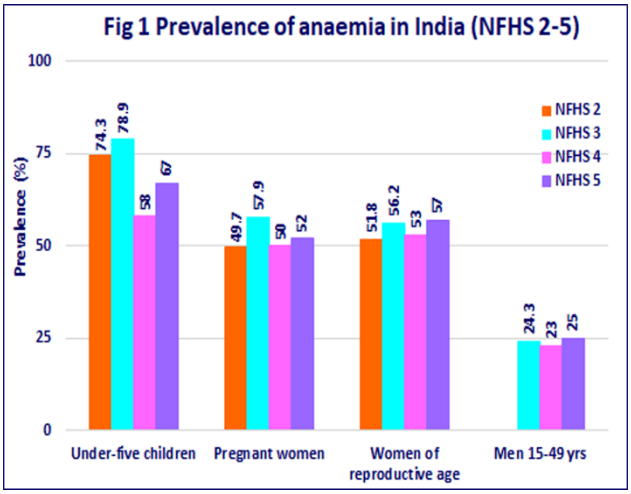
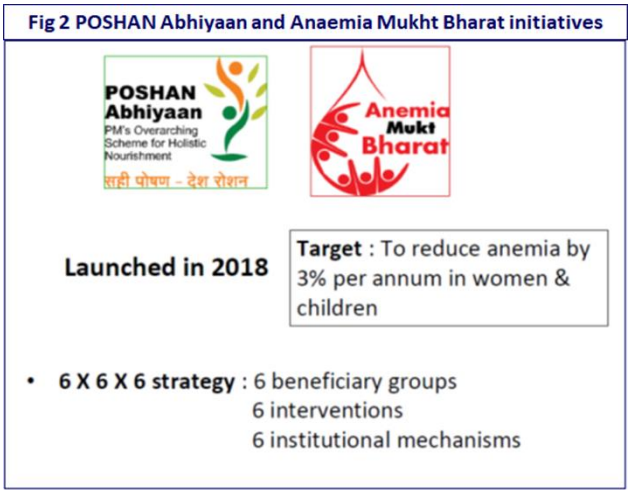
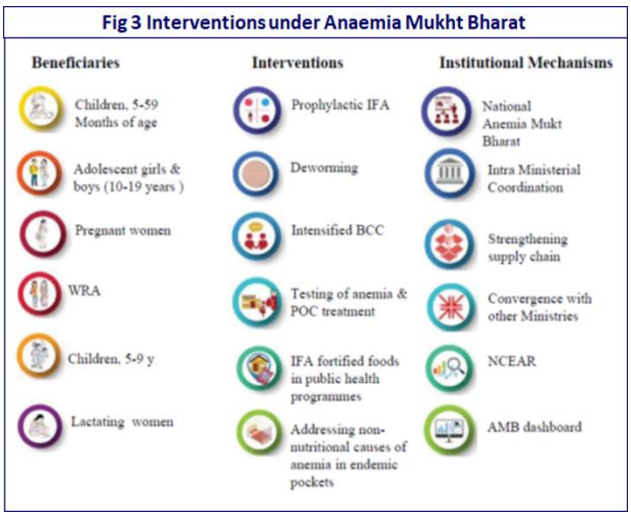




scattered programmes. The Government of India is also committed to the World Health Assembly target of 50% reduction in anaemia among WRA by 2025. In line with this, the POSHAN Abhiyaan launched in 2018 has set a target to reduce the prevalence of anaemia among children (6-59 months), adolescents and WRA (15-49 years) by 3% per year under the Anaemia Mukt Bharat (AMB) strategy with a multi-pronged approach and a more robust operational and accountability framework (Fig 2)[9,10]. The AMB programme launched in 2018 envisages implementation of a '6X6X6 strategy' wherein 6 population groups will be targeted with 6 interventions through 6 institutional mechanisms (Fig 3). Importantly, in addition to IFA supplementation, the AMB program also suggests screening for anaemia using point of care (POC) devices to aid diagnosis and treatment.

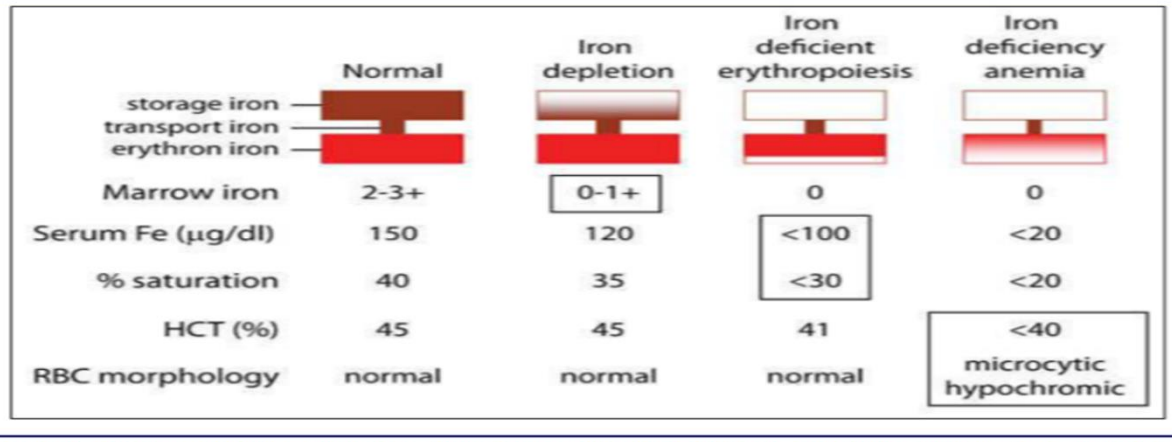
Despite the operationalization of anaemia control programmes over five decades and their intensification over the past few years, the prevalence of anaemia has remained largely static over the past two decades in India (Fig 1). In fact, the prevalence increased during the period between NFHS 4 and NFHS 5 by ≈9% among children 6-59 months of age and ≈5% in adolescent girls. The anaemia prevalence among WRA (15-49 years) increased in West Bengal, Assam, Gujarat, Odisha and Chhattisgarh while the prevalence for children (6-59 months) increased in all states except Haryana, Jharkhand and Uttarakhand[11]. The lack of effective outcomes from the ongoing anaemia control programmes is thought to be to be due to multiple operational limitations such as supply-chain related problems and poor compliance with IFA supplements by the target groups. Fortunately, there appears to be an improvement in compliance in recent years; for example, the numbers of mothers who reported consumption of >100 and >180 tablets of IFA during pregnancy were 13.8% and 11.6% higher, respectively, in NFHS 5 as compared to NFHS 4[12]. What is puzzling, however, is that despite the reported improvements in compliance with IFA supplement intake, there has been no decline in the prevalence of anaemia over this time period. There is a need to review the etiological factors and pathophysiology of anaemia and assess whether there is a requirement for midcourse corrections in the ongoing anaemia control programme in the country.



Prevalence of iron deficiency and contribution of iron deficiency to anaemia

Physiologically, iron is a type 1 nutrient, and its deficiency leads to depletion of stores in the body, followed by a reduction in functional pools; i.e. a reduction in serum ferritin (iron storage protein) followed by a decline in haemoglobin[13]. Longitudinal studies in pregnant women found reduction in iron stores prior to the fall of haemoglobin levels (Fig 4)[14]. This should translate to a higher prevalence of iron deficiency (ID) compared to anaemia in a given population, particularly when the major cause of

Fig 4 Concept about relationship between iron deficiency and anaemia



Iron deficiency precedes anaemia. Prevalence of iron deficiency should be higher than anaemia. Anaemia is the most severe form of iron deficiency.

anaemia is iron deficiency. The fundamental basis of universal iron supplementation programs to treat anaemia, is based on the assumption that most of the anaemia is due to iron deficiency[15]. Early modelling calculations based on National Health and Nutrition Examination Survey (NHANES) data in the US indicated almost 100% iron deficiency (as assessed by low serum ferritin levels) in population groups with anaemia prevalence of ≈40%[15,16]. Based on this data and possibly due to limitations in assessing iron status at population level, WHO recommended universal iron supplementation in countries where the prevalence of anaemia is >40%, indicative of a severe public health problem. This recommendation was adopted by all countries including India. However, research studies over the past decade and data from the Comprehensive National Nutrition Survey (CNNS) in children, have shown that the prevalence of iron deficiency anaemia (IDA) is much lower, only ≈50% of the total anaemia prevalence[17,18] (Figs 5-8). Thus, currently it is recognized that only about 50% of anaemia may be due to iron deficiency (IDA), which can be potentially treated with iron supplementation[19]. Data from supervised IFA treatment studies (studies wherein compliance with supplementation was high) in Indian women and children have shown that there was only about 50-60% reduction in anaemia from base line after intervention[18,20,21]. If only 50% of cases of anaemia are due to iron deficiency and the other half due to other factors such as concurrent deficiency of other haemopoietic nutrients, chronic inflammation, infections or unknown causes, only about half the anaemic population of the cases of anaemia may respond to IFA treatment. The CNNS conducted across all the states of India during 2016-18 measured the prevalence of anaemia and the biomarkers of multiple nutrients, including those of iron and provided information that filled some important gaps in the understanding of the aetiology of anaemia[22]. As expected, the prevalence of both anaemia and ID was found to be higher in adolescent girls and in under-five children (Figs 5, 6). However, anaemia due to ID was found to be much lower than anticipated; 36.5% in the 1-4 years age group, 15.6% in the 5-9 years age group and 25.6% in the 10-19 years age group. Other factors such as folic acid and B12 deficiencies, dimorphic anaemia or anaemia of

Fig 5 Prevalence of grades of anaemia (CNNS 2016-18)

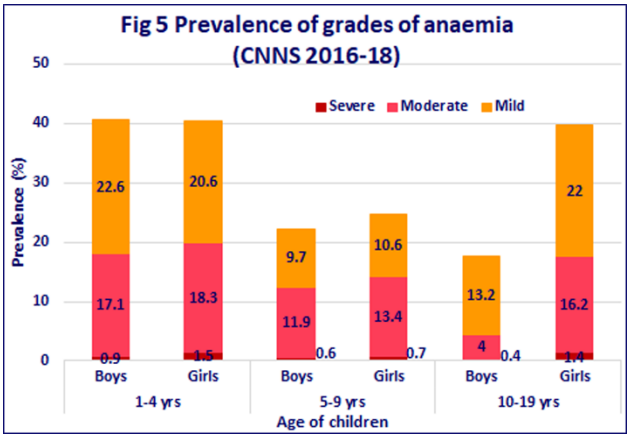
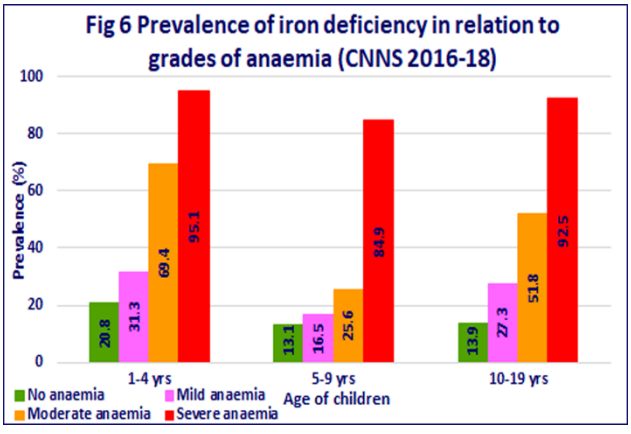
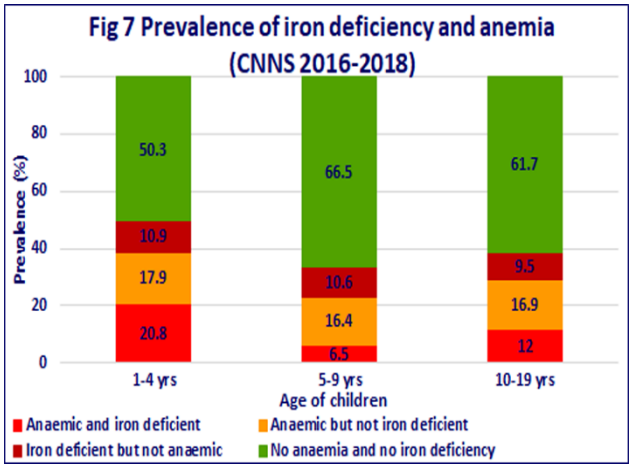
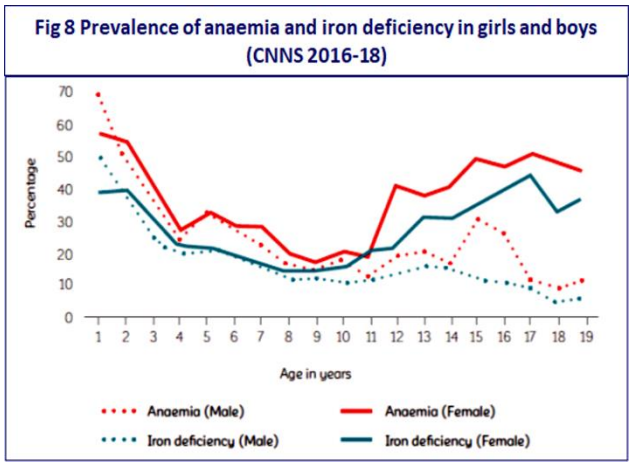


Fig 6 Prevalence of iron deficiency in relation to grades of anaemia (CNNS 2016-18)

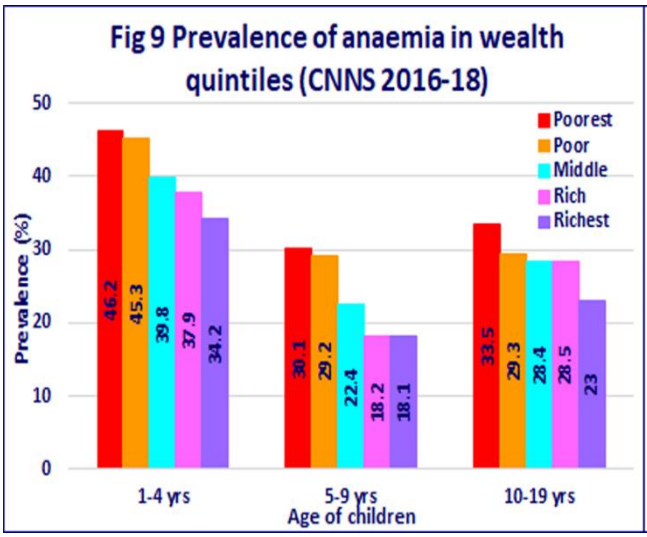


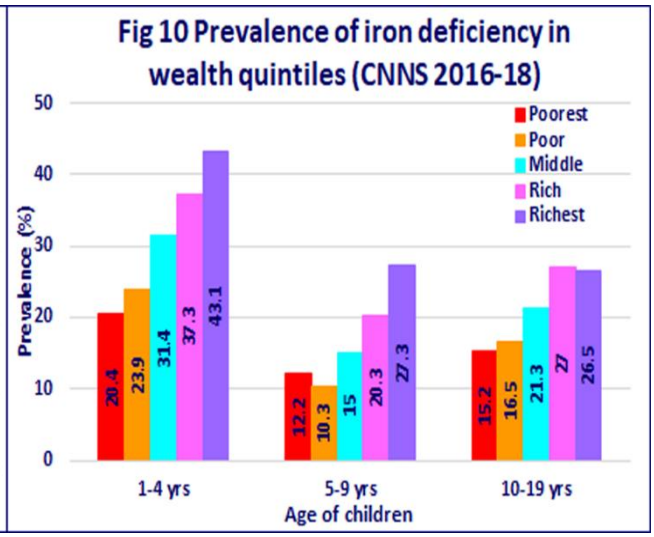




inflammation[23] were also responsible for anaemia in children. Around 44% and 31% anaemia were due to other causes in the 5-9 year and the 10-19 years age groups, respectively. The results of this survey showed that the prevalence of ID was lower than that of anaemia in all the age groups including preschool children, school-age children and adolescents[24] (Figs 5, 6). Further, it was found that the prevalence of anaemia was higher among children from the lower wealth quintiles and in rural children as compared to urban children, while the ID prevalence showed an inverse pattern with a higher prevalence in urban participants and in those from higher wealth quintiles (Figs 9, 10). The significant negative urban/rural differences between anaemia and ID persisted even after adjusting for underlying inflammation, and multiple relevant confounders. One of the possible reasons for this observation could be that underlying residual inflammation remained a significant factor despite adjustment for it by multiple methods. This may have resulted in higher serum ferritin levels independent of iron status, leading to low estimates of ID among poor or rural children who have a relatively higher risk of infections and inflammation[25]. Also, the findings were consistent when alternative markers of iron status, such as serum transferrin receptor (sTfR) and body iron stores (BIS) were considered. Analyses to examine the interaction between serum ferritin and wealth index with haemoglobin as a dependent variable to assess iron utilization in different wealth quintiles provided interesting insights. It was observed that serum ferritin significantly interacted with the wealth index, with declining trends in the strength of association between haemoglobin and serum ferritin from the richest to the poorest groups. After adjusting for relevant confounders, the change in haemoglobin with per unit change in serum ferritin (SF) was three times higher in the richest quintile as compared to the poorest quintile, suggesting impaired iron utilization for haemoglobin synthesis in poorer wealth quintiles.

These observations indicate that, although iron deficiency is the leading cause of anaemia in some age groups, there appears to be a significant contribution from other nutritional and non-nutritional causes primarily related to low socio-economic status that hamper utilization of body iron





for haemoglobin synthesis. These findings have been validated by studies in other settings. For example, multiple studies have shown lower haemoglobin despite higher ferritin concentrations in black populations than in white populations in USA and the authors of these studies have attributed this to lower iron mobilization from stores for haemoglobin synthesis due to poor nutrition and overall health[26,27]. It appears that the high prevalence of anaemia despite available iron stores in low-income participants may be indicative of 'functional' rather than 'absolute' iron deficiency. Therefore, focusing exclusively on iron supplementation programmes (IFA supplementation and iron fortification of multiple foods) may not result in the intended beneficial outcomes. Comprehensive investigations into other causes of anaemia are required in the Indian context in order to address the problem holistically.

Dietary iron intake and anaemia

Dietary iron exists in two forms, heme iron (40% of iron in animal foods) and non-heme iron (60% of iron in animal foods and ≈100% in plant foods). The bioavailability of heme iron is much higher than that of non-heme iron (around 25% vs 5-8%) due to differences in relative solubility and efficient intestinal absorption. But non-heme iron contributes about 90-95% of the total daily iron intake in typical vegetarian diets consumed in India[28]. Phytic acid and polyphenols that are abundant in plant foods, chelate the dietary non-heme iron and limit its bioavailability, while; vitamin C present in fresh fruits and vegetables increases the bioavailability of iron[29]. An analysis of dietary iron intake in nine states in India showed that cereals and millets contribute 30-80% of the iron intake from diets across different states. The high levels of phytic acid present in cereals and millets inhibits iron absorption. This fact, coupled with the overall poor intake of pulses, fresh vegetables and fruits rich in vitamins (contributing to 5-10% and 2.5% of daily iron intake, respectively) results in overall poor density and bioavailability of iron; this is thought to be the major cause of nutritional anaemia in India[28]. However, an analysis comparing dietary iron intakes with prevalence of anaemia among WRA indicated that neither total iron intakes nor the bioavailability of the iron in the diet could explain interstate differences in the prevalence of anaemia. This finding implies that there may be other factors leading to anaemia[28]. A recent study that triangulated the data from the National Sample Survey Organization (NSSO) surveys and NFHS data, showed only a weak association of dietary iron intake with anaemia (OR: 0.992; 95% CI: 0.991, 0.994) among WRA. In this analysis also, interstate differences in prevalence of anaemia could not be explained on the basis of dietary iron intake alone[30]. In a modelling analysis, an increase in the dietary intake of iron by 10mg/day substantially filled the gap between dietary intakes and the estimated average requirement (dietary iron inadequacy across the states shifted from 24-94% to 9-39%), but resulted in only an estimated 6% decrease in anaemia prevalence. Moreover, the benefits of iron supplementation appeared to be state-specific; that is, the states with high prevalence rates of anaemia and low iron intakes (north eastern states) were likely to benefit the most[30]. A comprehensive assessment of dietary intakes and estimates of haemoglobin and other nutritional biomarkers may help in providing better insights into the potential relationships between the various factors, and thereby help in formulating a more effective nutrition policy in the country.

The persistent high prevalence of anaemia and the lack of expected benefits from anaemia control programmes, had led to efforts to further increase iron intakes through simultaneously implemented multiple programmes such as IFA supplementation and iron fortification of foods (salt and rice) in India, with the hope to accelerate the decline in anaemia[10]. Fortification of rice and salt (distributed through the Public Distribution System) with iron is suggested in AMB guidelines, and distribution of iron-fortified rice has been initiated across the country[9]. However, clinical trials in India showed that although the consumption of iron-fortified rice, wheat or salt did result in improvement in iron stores as measured by serum ferritin levels, their effect on raising the blood haemoglobin levels remained marginal, if any[31-36]. Interestingly, two randomized controlled trials on supervised supplementation of iron-fortified rice to school children through midday meals reported significant reductions in anaemia prevalence, even in control groups[32,36]. This finding might possibly be attributable to the deworming programme taken up concurrently, and possibly better overall food intake through Mid-Day Meal due to assured delivery and supervised intake of mid-day meals by the children. The findings from these trials and the dietary intake analyses outlined above indicate that increasing the iron intake alone may not achieve the targeted 3% per annum reduction in anaemia prevalence envisaged in the AMB programme. This is not surprising considering that the contribution of iron

deficiency to the overall prevalence of anaemia in school age children appears to be only ≈30%[23].

Adverse effects of excess and unabsorbed iron

Emerging evidence points to deleterious effects of unabsorbed iron on the gut microbiome, and an association between high iron stores and metabolic disease[37-40] suggesting that in the long run, unintended metabolic complications may result from multi-pronged efforts to increase iron intake. Indeed, dietary intake analyses showed that concurrent IFA supplementation along with food fortification (rice and salt) could result in habitual intakes of iron beyond the tolerable upper limit (TUL)[41-43] a level of intake that is associated with an increased risk of adverse effects. It is essential to undertake in-depth studies on the aetiology of anaemia. Folic acid and vitamin B_{12} are considered major limiting haemopoietic nutrients, and the co-existence of anaemia with concurrent deficiencies of both these nutrients is common in India[23]. While folic acid is always supplemented along with iron, studies in India and elsewhere have shown that additional supplementation of vitamin B_{12} had no additional benefits on Hb levels when compared to IFA supplementation in Indian adolescent girls[18,44]. Systematic reviews also suggested that the impact of multiple micronutrient supplementation on haemoglobin were modest[45]. It is clear that there is no magic bullet to treat population level anaemia. The interaction between iron and inflammation in the body throws up multiple complex challenges which need to be addressed comprehensively.

Studies conducted at ICMR-National Institute of Nutrition showed that the consumption of Guava, a fruit rich in vitamin C, doubles the bioavailability of iron in adolescents[46]. Further, in a cluster randomized trial, the inclusion of Guava as a part of the supplementary meal in Anganwadis enhanced the iron stores and reduced the prevalence of acute respiratory infections among preschool children[47]. There is a need to expand the dietary interventions to address anaemia holistically.

Iron biology and interaction with inflammation

In mammals, iron is highly conserved and there are no obligatory pathways for its excretion. The basal losses of iron through shedding of intestinal cells, sweat and urine, and increased demands during infant and adolescent growth spurts and in pregnancy are compensated by the concurrent modulation of intestinal iron absorption. Intestinal iron absorption entails a reduction in luminal iron (Fe^{+3} to Fe^{+2}), uptake and subsequent release by intestinal cells, re-oxidation (Fe^{+3} to Fe^{+2}) and transport to the systemic circulation, all of which are mediated by specific proteins[48]. The intestinal absorption of iron is regulated by a variety of systemic factors. For instance, iron deficiency increases the rate of intestinal iron uptake and transport while its sufficiency reduces both absorption and transport to the serosal side. Within the body, iron is stored in tissues bound to ferritin or utilized for synthesis of new red blood cells. In addition, recycled iron from aged RBCs in the macrophages also forms an important source of iron. It is estimated that about 20 mg/day of iron is required for erythropoiesis per day, while the typical absorbable dietary iron is in the range of 1.2-2 mg/day, thereby rendering recycled iron critical in maintaining erythropoietic requirements[49]. Since the sites of iron storage (i.e. liver and other tissues) are different from those of its entry (intestine and macrophages), these tissues need to cross-talk to regulate iron absorption and mobilization. Hepcidin, a cysteine-rich 25- amino-acid cationic peptide synthesized and secreted by the liver, has been identified as the key regulator of mammalian iron homeostasis[48-50]. Hepcidin, secreted in response to high iron stores, inhibits both intestinal iron absorption and mobilization of iron from tissues[50].

In addition to iron status, hepcidin expression is also modulated by inflammation, ineffective erythropoiesis and hypoxia[50,51]. While both erythropoietin and hypoxia reduce hepcidin secretion (thereby making iron available for new RBC synthesis), inflammation does the opposite. Many studies showed elevated levels of hepcidin during inflammation, and hepcidin appears to be the mediator of anaemia associated with inflammation[51]. The measurement of hepcidin levels will be of value in studies evaluating anaemia and iron deficiency and will throw light on the mechanisms resulting in the observed lower prevalence of ID than of anaemia.

Unique iron biology in pregnancy

Management of anaemia in pregnant women is further complicated by the unique iron biology during pregnancy. Recent evidence suggests that, unlike other physiological groups, hepcidin response to iron status is suppressed during pregnancy[52]. The maternal hepcidin levels remain relatively unaltered by iron supplementation, potentially resulting in elevated iron absorption even in iron-replete pregnant women. The molecular evidence suggests

that placental derived molecules are involved in the process. Since maternal hepcidin, apart from its role in reducing iron absorption, blocks placental iron transfer, this suppression of hepcidin could be a physiological requirement for optimal iron transfer to the foetus[53]. However, supplementation of iron in this context of hepcidin suppression might increase iron stores and lead to associated adverse health consequences, particularly in those women with adequate iron stores. There had been reports that, iron supplementation to non-anaemic pregnant women resulted in increased oxidative stress, increased risk of gestational diabetes and increase in low birth weight (LBW), preterm birth[39,54]. In a cohort of non-anaemic pregnant women from South India (n=1196) who were prescribed 45 mg of elemental iron per day, women with higher supplemental iron intake (>39.2 mg/day) had a higher risk of delivering term LBW babies as compared to those with lower supplemental iron intake (⩽36.6 mg/day) (adjusted risk ratio: 1.89; 95% confidence interval: 1.26, 2.83)[55]. Given this background, the simultaneous implementation of multiple iron interventions in an attempt to accelerate the reduction in anaemia prevalence may prove to be counterproductive.

Currently WHO recommends supplementation with 60 mg/day iron during pregnancy in regions where anaemia is a severe public health problem (prevalence >40%). This is based on a systematic review in 2012 which showed that iron supplementation reduced the risk of LBW by 19% as well as maternal anaemia and ID at term[56]. The updated evidence, however, showed lower benefits (with borderline statistical significance) of iron supplementation on reducing the incidence of LBW, with no benefit for other birth outcomes[57]. In the light of these findings, some researchers are questioning the wisdom of routine iron supplementation in non-anaemic, well-nourished pregnant women[58]. In depth studies to provide a comprehensive understanding of the iron biology, particularly during pregnancy, is critical in designing effective anaemia management strategies.

Are we measuring anaemia correctly?

In addition to the above dietary and physiological uncertainties, there also appear to be methodological reasons that explain the stubborn resistance of anaemia prevalence despite iron supplementation interventions in India. The persistent findings of high prevalence of anaemia may also be linked with over-estimation of the problem due to:

- errors in haemoglobin estimation in epidemiological surveys, and
- the haemoglobin threshold levels set for diagnosing anaemia.

Typically, an anaemia diagnosis requires measurement of haemoglobin in a blood sample. In hospital/laboratory settings, venous blood haemoglobin is measured using automated coulter counters or by a direct cyanmethaemoglobin method, which is considered the gold standard. However, in epidemiological surveys, haemoglobin estimation in capillary blood samples with a point of care device (POC), often Hemocue, is preferred due to logistical simplicity in sample collection and measurement. Several comparative studies found significant over- or under-estimation of haemoglobin in capillary blood as compared to venous blood[56-64]. A study in Uttar Pradesh, India, found that haemoglobin measurements in capillary blood with Hemocue were ≈0.9 g/dL lower compared to measurements taken using the gold standard, resulting in an overall ≈25% higher prevalence of anaemia[59]. This study also reported inconsistent errors across the range of haemoglobin concentrations, with higher errors at higher ranges of haemoglobin estimates. Similar findings have been reported in a study which compared four nationally representative surveys matched by time, measuring haemoglobin by Hemocue with capillary blood samples [Demographic Health Surveys (DHS)] or venous blood samples[65]. This study showed substantial differences in anaemia prevalence estimates (ranging from 2 to 31% in young children and 2 to 16% in WRA) in three out of four countries with consistently lower prevalence in the BRINDA surveys. Another study reported a mean difference of 0.5g/dL between capillary and venous blood haemoglobin measurements, which, when applied to NFHS 4 data, resulted in 20-25% lower prevalence of anaemia[66]. Further, there appears to be a 20-30% variation in haemoglobin content in consecutive drops of capillary blood when measured with Hemocue, making it difficult to arrive at a possible correction factor for this random error[60]. Further, in addition to individual human errors in measurement, inconsistencies arise due to other factors such as lancet dimension, skin thickness, humidity and temperature, and storage conditions of cuvettes. In this context, most investigators now suggest using gold standard methods for assessment of population level anaemia prevalence[59,60,66].

One of the important findings of the CNNS (2016-18) is that the prevalence of anaemia among 1-19 year

old children and adolescents was ≈20-25% lower as compared to the data from the NFHS 5 (2019-21) survey, although both these surveys were conducted around the same time[12,22]. The apparent differences in anaemia prevalence between the CNNS and NFHS could be due to differences in haemoglobin estimation methods (venous blood measurement using automated coulter counter in CNNS *vs* capillary blood measurement using Hemocue in the NFHS). Added to these methodological uncertainties, the distribution of population haemoglobin levels is generally centred around the diagnostic haemoglobin cut-off for anaemia in India, and therefore small analytical errors in haemoglobin estimation have a large impact on the anaemia prevalence estimates. For instance, an upward correction of 0.5g/dL haemoglobin in NFHS 4 data resulted in findings of prevalence levels that were lower by 16% (preschool children, 61 to 45%), and 23% (WRA, 55.8 to 32.8), essentially downgrading the condition from 'severe' to 'moderate' public health problem in WRA[66]. An error of this magnitude is likely to have policy implications for anaemia control strategies (universal versus targeted approach). Therefore, precise and accurate measurements of haemoglobin using gold standard methods in population surveys is essential for assessing anaemia prevalence in order to guide future policy.

Yet another important aspect of anaemia assessment relates to the diagnostic cut-offs of haemoglobin levels. The current haemoglobin cut-offs of anaemia, recommended by WHO in 1968, were derived from 5 studies based on predominantly western populations[68]. These were subsequently revised with adjustments for latitude, smoking, and young children[1,68]. These cut-off levels to diagnose anaemia have been adopted by all countries, including India. However, several subsequent studies have reported that lower haemoglobin cut-offs may be appropriate in infants, young children, pre-menopausal women, and the elderly as compared to WHO recommendations[69]. Blood haemoglobin levels have also been reported to vary with ethnicity[67,69]. Based on these emerging global data, WHO is in the process of re-examining these cut-offs[1,68]. The current haemoglobin cut-offs were statistically derived from the lower 2.5th centile of haemoglobin distribution in healthy populations stratified by age and physiological groups[70]. Using a similar methodology, haemoglobin cut-offs were recently derived for 1-19 years old healthy Indian children and adolescents from CNNS data, following stringent criteria to exclude participants with multiple micronutrient deficiencies and abnormal metabolic markers. Importantly, the haemoglobin cut-offs (lower 5th centile) reported in this study were lower by 1-2 g/dL for different ages as compared to current WHO cut-offs[70]. The anaemia prevalence assessed by these new cut-offs across all age groups was found to be ≈20% lower as compared to prevalence levels arrived at by applying the WHO cut-offs (10.8% *vs* 30%). Other recent studies that reviewed the global data from 25 countries (including India) in 6-59 months children and 15-49 years WRA collected between 2005-2016, also reported that haemoglobin cut-offs could be lower than the current WHO cut-offs by about 1.35 and 1.2g/dL, respectively[71]. The INTERGROWTH-21st study conducted in pregnant women from geographically diverse urban areas in eight different countries (Brazil, China, India, Italy, Kenya, Oman, United Kingdom and United States) showed that haemoglobin thresholds compatible with good maternal and perinatal outcomes are gestation-specific, and that haemoglobin cut-offs could be lower by 1g/dL compared to current WHO cut-offs[72]. Evidence from these large data sets and from multiple countries provides strong evidence that current WHO haemoglobin cut-offs might be higher than optimal, resulting in potential over-estimation of anaemia prevalence in populations.

The way forward

Concerns have been expressed regarding persistent high prevalence of anaemia in India despite ongoing anaemia control programmes for five decades. The relatively lower prevalence of ID than of anaemia is counter-intuitive and indicates the need for reassessing causes of anaemia. Concurrent deficiencies of other nutrients are important, but emerging data suggests that a substantial proportion of high anaemia prevalence could be explained by cumulative contributions from: (i) under-estimation of haemoglobin (due to use of capillary blood samples) and (ii) over diagnosis of anaemia (due to higher than optimal haemoglobin cut-offs).

When these are accounted for, there might be a substantial reduction in the severity and magnitude of anaemia as a public health problem in India. Adoption of gold standard methods of haemoglobin estimation and appropriate cut-offs are important for accurate estimation of the magnitude of the problem. This will help in redirecting the efforts to increase iron intakes targeted at specific vulnerable groups in the population with existing institutional

mechanisms for effective anaemia reduction.

Multiple programmes of iron intervention should be carefully evaluated for potential harmful effects and it would be prudent to provide iron fortification through only one food item. Also, it is important to note that adverse health consequences are reported both with iron deficiency and excessive iron intake.

Therefore, screening for anaemia, and possibly iron status, is necessary to improve the treatment modalities while reducing the associated risks; some of the risks associated with excess intake may not be apparent or measurable in the short term. Future research should be directed towards the development of field-friendly methods of estimation of iron and biomarkers of other nutrients and also of inflammation, so as to make progress towards precision in nutrition strategies.

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FOUNDATION NEWS

Dr. Kamala Krishnaswamy, **Chairperson Governing Body** of Nutrition Foundation of India was conferred the **'Living Legend'** award by **the International Union of Nutrition Sciences** in December 2022

FOUNDATION DAY of the Nutrition Foundation of India was celebrated on 29.11.2022.

Dr. D. Prabhakaran delivered the C Ramachandran Memorial Lecture on 29.11.2022 at 3.00 PM on 29.11.2022. The theme of his lecture was **"New insights into the causes, consequences and control of CVD in India".** The webinar was well attended and his lecture was highly appreciated.

THE THIRD Dr. C GOPALAN MEMORIAL WEBINAR was held on 11.10.2022 between 10.00 AM and 1.00 PM. The theme of the Webinar was **"Action plan to mitigate health and nutrition challenges associated with climate change"**. There were four presentations:

Health action plan for combating climate change related problems Dr A Shrivastava
Action Plan to cope with heat stress Dr D Mavalankar
Climate change-and nutrition impact options for action Dr A Laxmaiah
Climate change & food security Dr P Ramachandran

The webinar was well attended; in addition to the nutrition and health professionals from academic and research institutions, state level consultants and nodal officers on climate changes and health from various state governments participated in the webinar. There were very good interactions between the speakers and the participants.

NUTRITION NEWS

The **54TH ANNUAL CONFERENCE OF THE NUTRITION SOCIETY OF INDIA** was held in physical mode at the National Institute of Nutrition, Hyderabad on 22nd and 23rd December, 2022. The Theme of the conference was "Sustainable Healthy Diets Health for All".

Professor Caroline Fall, Emeritus Professor of International Paediatric Epidemiology, MRC Lifecourse Epidemiology Centre, University of Southampton, Southampton General Hospital, UK received the **46th C GOPALAN ORATION AWARD**. The theme of her oration on 22.12.2022 was "Mothers, babies and health in later life".

Dr. R. Hemalatha, Director, ICMR-NIN received the **34th S.G. SRIKANTIA MEMORIAL LECTURE AWARD**. The topic of her oration on 22.12.2022 was "Is India eating right?"

Prof. Anupa Sidhu, Director, Lady Irwin College, New Delhi received the **13th Dr. RAJAMMAL P DEVADAS MEMORIAL AWARD**. The topic of her lecture on 23.12.2022 was "Maternal Nutrition; Issues and Initiatives".

Dr. H P S Sachdev, Senior Consultant, Paediatrics and Clinical Epidemiology, Sitaram Bhartia Institute of Science & Research, New Delhi received the 9th **Dr. B.K. ANAND MEMORIAL AWARD** on 23.12.2022.