

# **Nutritional Anemia**

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# Nutritional Anaemia

- Deficiency of
  - Iron
  - Folate
  - B<sup>12</sup>
  - Protein
- corrected by supplementation

# Iron def anemia

# Introduction

- Iron deficiency (ID) is one of the most frequent nutrition deficiency all round the world. In India - 50%
- Its prevalence is higher in children and childbearing age women.
- Iron deficiency anemia (IDA) mainly affects child behavior and development, work performance and immunity.

# Prevalence of ID and IDA in the World

<b>Regions</b>	<b>Population with Iron Deficiency or Anemia (Millions)</b>	<b>Prevalence of Anemia in Pregnant Women (%)</b>
Africa	206	52
The Americas	94	40
Europe	27	18
Eastern Mediterranean	149	50
Southeast Pacific	616	74
Western Pacific	1058	40
Developed Countries		18
Developing Countries		56
<b>Total</b>	<b>2150</b>	<b>51</b>

# Iron physiology and metabolism

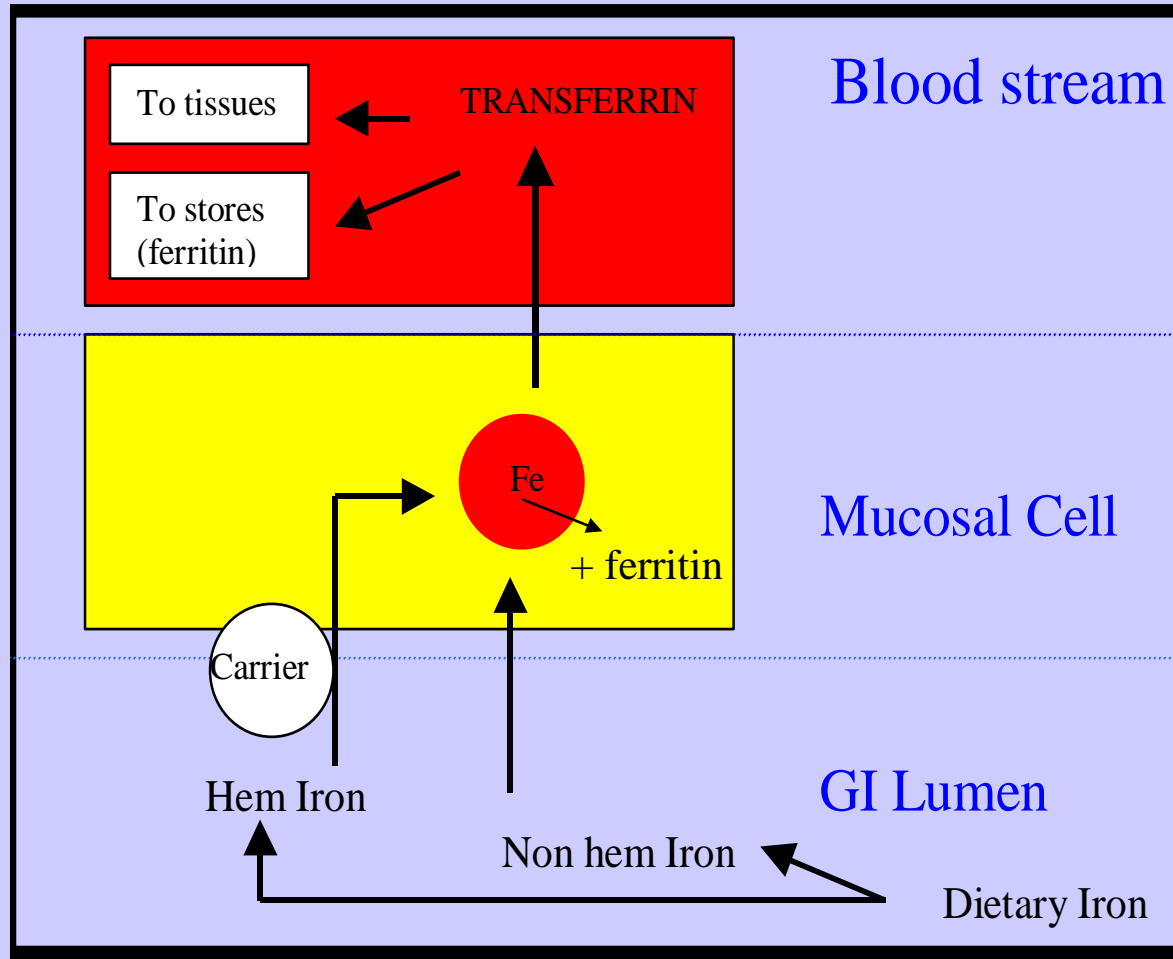
- Dietary sources of Iron can be classified as food sources and fortified foods.
- The amount of Iron varies widely between foods.
- Iron exists in food under two forms, heme and non heme iron.
- They are not only different in terms of their sources, but also in terms of bioavailability.

# Sources

- Animal- meat, liver, kidney, egg yolk.
- Veg.- pulses, beans, peas, green vegetables and fruits
- Milk- Human milk -0.29- 0.45mg/dl

(Cow's milk –poor source with 0.01 – 0.38mg/dl)

# Iron physiology and metabolism





# **Transfer of Iron to the circulation and transport**

- Transferrin is the major protein responsible for transporting Iron in the body.
- Transferrin receptors, located on the surface of nearly all cells in the body, can bind two molecules of transferrin.
- Transferrin saturation is important in assessing ID.

- Tissues with higher requirements of Iron (erythroid precursors, placenta and liver) contain higher concentration of transferrin receptors.
- Once in tissues, Iron is stored as ferritin and hemosiderin compounds, which are present primarily in the liver, RE cells and bone marrow.
- The amount of ferritin in storage compartment depends on Iron status which ranges from depleted to replete iron status
- Ferritin concentration expresses Body Iron Stores when assessing ID.

# Regulatory mechanisms of Iron absorption and cellular uptake

## Dietary Factors

- physico –chemical form (ferrous form better absorbed),
- other dietary constituents (phosphates, phytates, calcium, tannic acid, etc.),
- Iron dose

## Host-related conditions

Facilitators		Inhibitors	
Beef, lamb, pork, liver, chicken, fish	+++	Wheat bran	+++
Orange, pear, apple, pineapple juices	+++/++	Tea	+++
Plum, banana, mango	++/+	Nuts	+++
Carrot, potato, pumpkin, broccoli, cauliflower, tomato	++/+	Legumes	+++
Salad (lettuce, tomato, green pepper, cucumber)	+	Leafy vegetables	+++
		Coffee	+++/++
		Maize	+++/++
		Rice	++/+
		Eggs	+
		Spinach	+

# Host related factors

- Iron stores and the amount of iron to which intestinal cells have been exposed constitute the main factors regulating iron absorption.

# **Other factors influencing Iron absorption**

- Rate of erythropoiesis
- Physiological state
- Gastric juice

# Causes of iron deficiency

- Diminished stores
- Diminished intake
- Diminished absorption
- Increased demands
- Defective metabolism

# Diminished stores

- Preterm & small for date babies
- Twins
- Early cord clamping (100ml of blood)
- APH
- Feto-fetal or feto-maternal transfusion



# Diminished intake

- Not breast feeding
- Cow's milk feeding
- Iron poor diet

# Diminished absorption

- Malabsorption
- Low level of enhancers
- High level of inhibitors

# Excessive losses

- Occult bleeding (erosive gastritis, drug induced gastritis)
- Recurrent diarrhea
- Hookworm
- Polyposis
- Prolapse rectum
- Portal hypertension
- Dysentery
- Meckel's diverticulum
- Hiatus hernia
- Cephalhematoma

# Increased demand

- Rapid catch up growth in preterm and SFD
- Infancy & puberty
- Preg & lact.

# Errors of iron metabolism

- Idiopathic pulm. Hemosiderosis
- Sideroblastic anemia
- Congenital transferrin deficiency

# Iron requirements (RDA)

Category	Age (years)	RDA – Iron (mg)
Infants	0 – 0.5	6
	0.5 – 1	10
Children	1 – 3	10
	4 – 6	10
	7 – 10	10
	11 – 14	12
Males	15 – 18	12
	19 – 24	10
	25 – 50	10
	51 +	10

Females	11 – 14	15
	15 – 18	15
	19 – 24	15
	25 – 50	15
	51 +	10
Pregnant		30
Lactating	1 <sup>st</sup> 6 months	15
	2 <sup>nd</sup> 6 months	15

# Iron in body

- Infant-250-300mg (65-95 mg/kg)
  - 65% in heam
  - 20% in ferritin & hemosiderin
  - 10% in myoglobin
  - Rest in cytochrome, catalase
- Adult – 4gm

# Stages of Iron Status

Overload    Normal    Depleted  
Stores    ID    IDA

Serum Ferritin	↑	N	↓	↓	↓	↓
Transferin Satur.	↑ ↑	N	N	↓	↓	
Erythrocyte Protoporph.	N	N	N	↑	↑	↑
MCV	N	N	N	N		↓
Hemoglobin	N	N	N	N		↓



# Clinical features

- Pallor, pica, dull, irritable, poor appetite
- Failure to thrive, easily fatigued
- Frequent infections
- Splenomegaly in 15%
- Tongue papillae are atrophied
- Malabsorption and protein losing enteropathy
- Nails-flat, thin, brittle, spoon shaped (koilonychia)
- Decreased attention span, poor school performance, cognitive impairment
- Severe – cardiomegaly & CCF

# Assessment of IDA

- Clinical and Laboratory indices.
- Laboratory indices are the most common methods used to assess iron nutrition status.

# Laboratory Indices

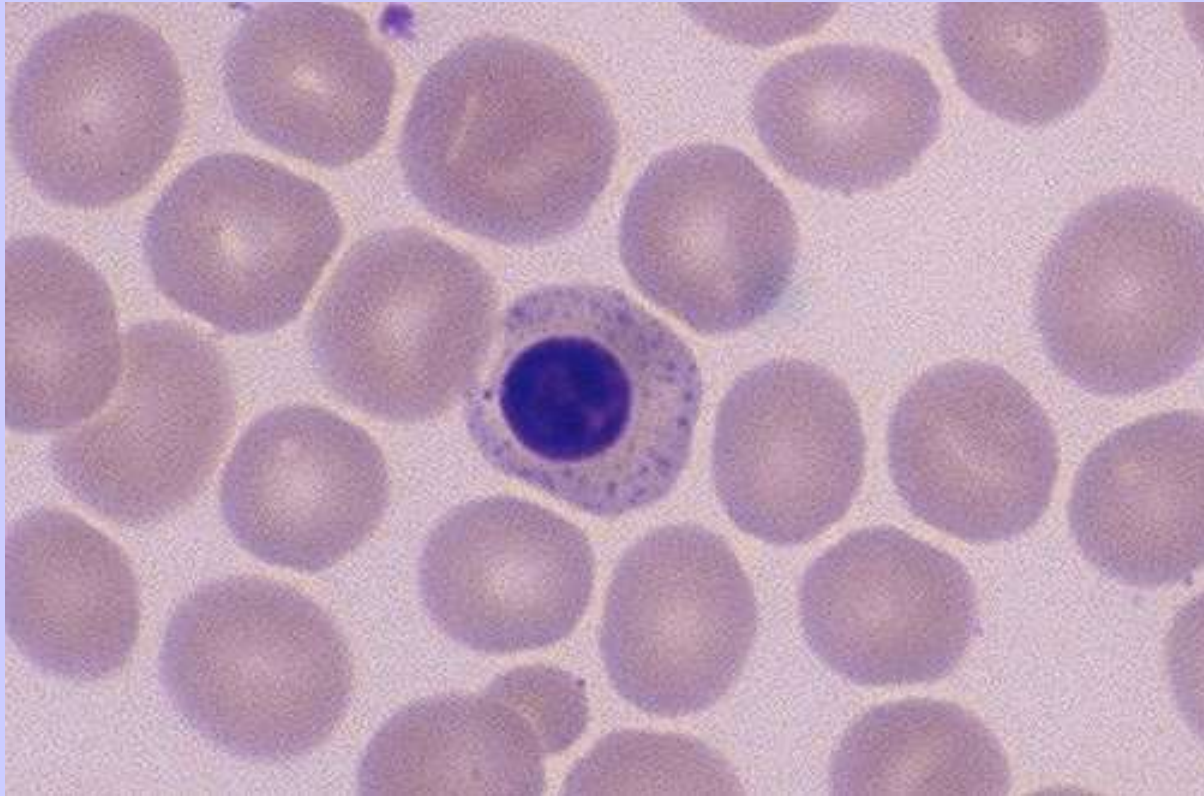
- Low Hemoglobin
- Low Hematocrit
- Low Mean Corpuscular Volume
- Serum Ferritin <10ng/ml
- Transferrin Saturation<15%
- TIBC>350μg/dl
- Increased free erythrocyte protoporphyrin

# Peripheral smear

- Microcytic hypochromic anemia, anisocytosis and poikilocytosis
- Low MCV, MCHC, MCH
- Low reticulocyte count

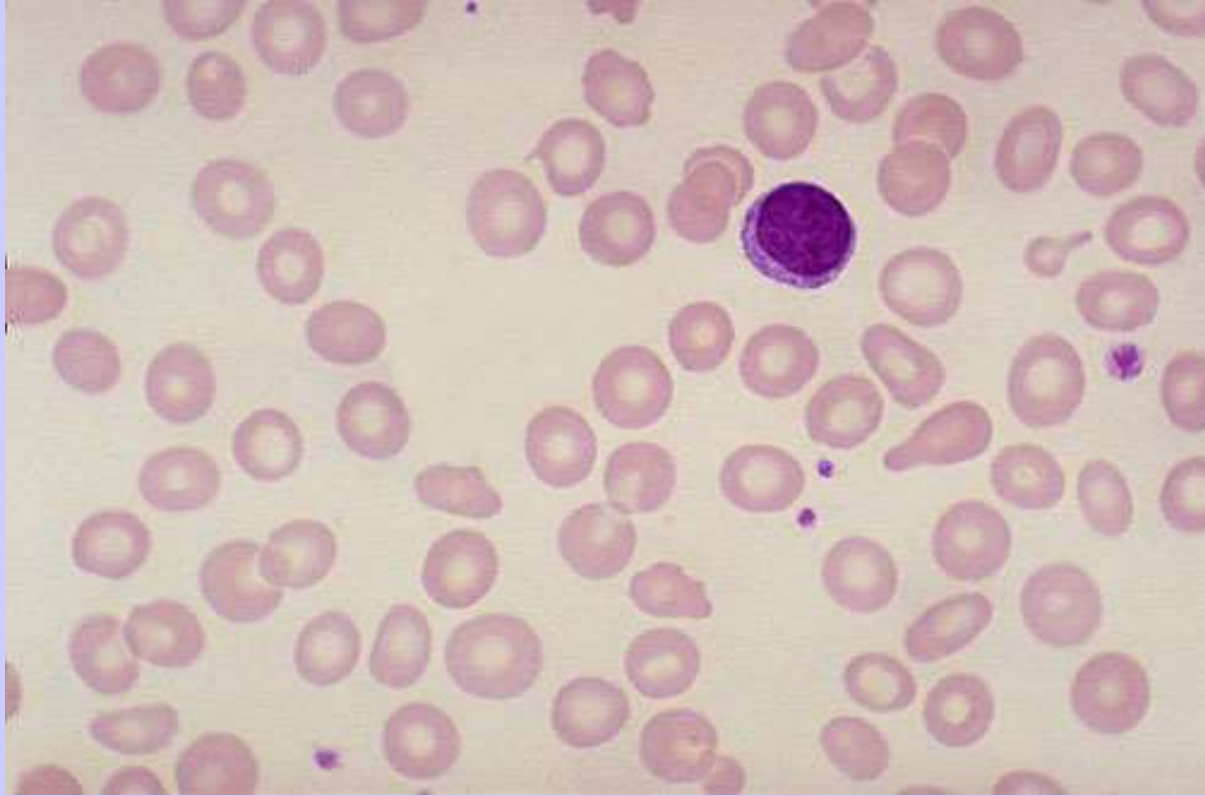


The red blood cells here are normal, happy RBC's. They have a zone of central pallor about 1/3 the size of the RBC. The RBC's demonstrate minimal variation in size (anisocytosis) and shape (poikilocytosis). A few small fuzzy blue platelets are seen. In the center of the field are a band neutrophil on the left and a segmented neutrophil on the right.



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The nucleated RBC in the center contains basophilic stippling of the cytoplasm. This suggests a toxic injury to the bone marrow, such as with lead poisoning. Such stippling may also appear with severe anemia, such as a megaloblastic anemia.



- The most common cause for a hypochromic microcytic anemia is iron deficiency. The most common nutritional deficiency is lack of dietary iron. Thus, iron deficiency anemia is common. Persons most at risk are children and women in reproductive years (from menstrual blood loss and from pregnancy)

# Clinical Indices

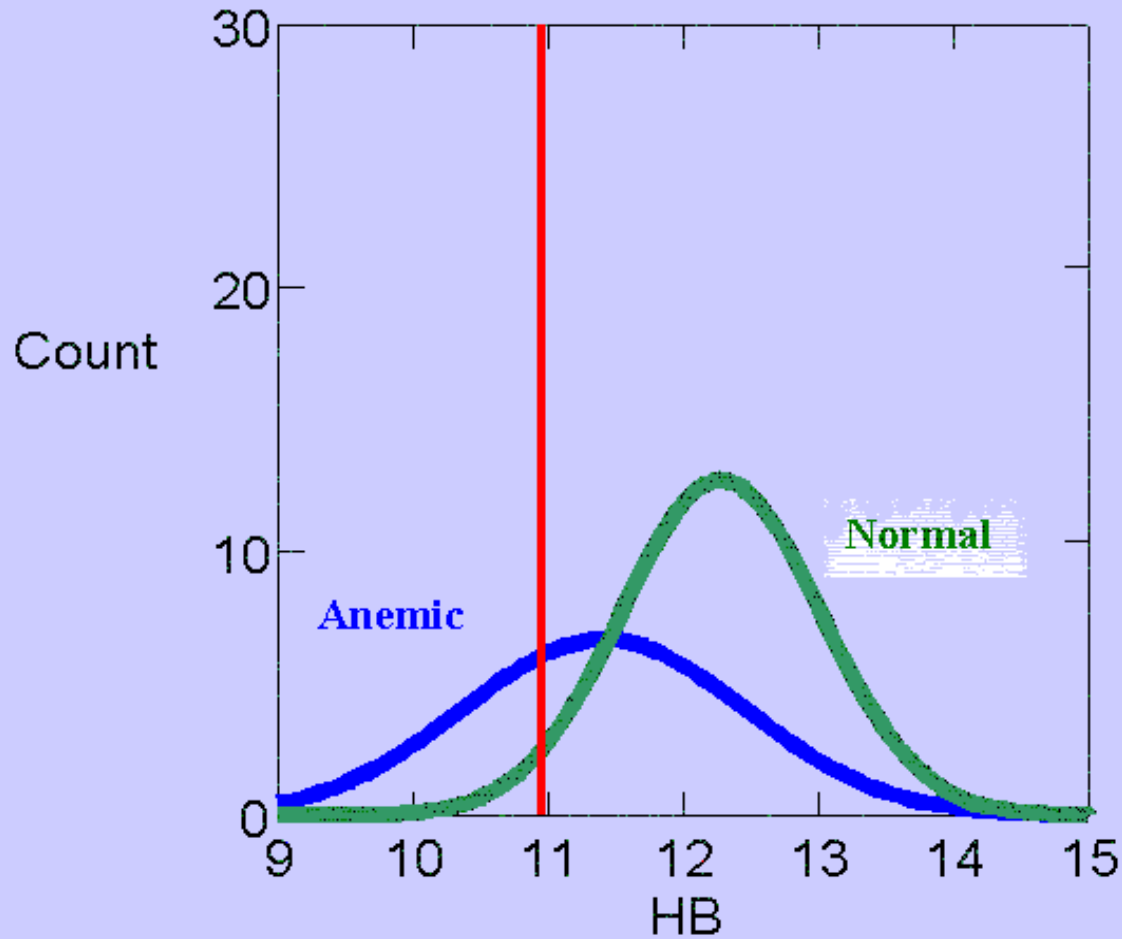
- Pallor of the conjunctiva,
- tongue,
- nail bed and palm



# Which is the best indicator?

- Several methods have been applied in order to assess iron deficiency in populations
- Hemoglobin cut off points have been one of the most frequently used criteria
- Mixed-distribution analysis is another methodology been used with the same purpose

# Mixed Distribution Model



- Response to iron supplementation has also been used to assess iron deficiency anemia.
- Response to iron supplementation is the best strategy to assess iron deficiency in rural areas.

# Stages of Iron deficiency

- Anemia is defined as hemoglobin concentration or hematocrit below 90% or 95% of range for healthy persons.
- Iron status can range from iron deficiency to iron deficiency anemia

# Stages of Iron Status

Overload    Normal    Depleted  
Stores    ID    IDA

	Overload	Normal	Depleted Stores	ID	IDA
Serum Ferritin	↑	N	↓	↓	↓ ↓
Transferin Satur.	↑ ↑	N	N	↓	↓
Erythrocyte Protoporph.	N	N	N	↑	↑ ↑
MCV	N	N	N	N	↓
Hemoglobin	N	N	N	N	↓

# Consequences of IDA

- During infancy studies have shown that IDA is related to decrease in responsiveness and activity, and tendency to fatigue.
- Studies have demonstrated increased lead absorption associated with ID.
- Lead poisoning is relevant especially during infancy because it also affects cognitive functions.

# Consequences of IDA

- IDA is related to decreased resistance to infections.
- Decreased work capacity is also described as another consequence of IDA.
- Anemia during pregnancy has been related to preterm delivery, low birth weight and fetal death.

# Treatment

- Treat underlying cause
- Oral iron therapy
  - 3-6mg/kg in 3 divided doses ( Hb rises by 0.4g/day)
  - Vit C, empty stomach or in between meals
  - For 6-8 wks after Hb is normal
- Parental iron therapy ( Iron in mg=wt in kg× Hb deficit×in gm/dl×4)
- Blood transfusion –rarely when Hb<4gm/dl, CCF, severe infection with poor iron utilisation



# Response to treatment

- Less irritable & increased appetite within 24 hrs
- Bone marrow response by 48 hrs
- Increased reti count by 3<sup>rd</sup> day
- Increased Hb level by 2 months
- Body iron store repletion

# Prevention of Iron Deficiency

- **Dietary modification**
  - Breast feeding and appropriate weaning diet
  - Iron rich food
  - Increase ascorbic acid
  - Decrease inhibitors
- Food fortification
- Iron supplementetation

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- Dietary modification
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- **Iron supplementation**
  - Preterm and LBW babies-10-15 mg/day iron
  - Iron supplementation during puberty

# Prevention

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- Iron rich food
- Increase ascorbic acid
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- Salt fortification by NIN
- Preterm and LBW babies-10-15 mg/day iron
- Iron supplementation during puberty
- *Deworming*
- *Foot wear use*
- *Safe drinking water*

# Megaloblastic Anemia

# Folic acid def

- Goat's milk is poor source
- Cooking destroys folic acid
- Chronic diarrhea, malabsorption and recurrent infections are prone
- In hemolytic anemias due to increased erythropoiesis
- Treatment with phenytoin / antimetabolites

# B<sup>12</sup> deficiency

- Mothers with B<sup>12</sup> deficiency – exclusively breast fed with delayed weaning





2006/01/01

# Clinical features

- Pale
- Very sick
- Irritable
- Severe anorexia
- Failure to thrive
- Knuckle pigmentation (hands and nose)
- Tremor and developmental regression

# Lab Investigations

- Macrocytic normochromic anemia
- Polymorphs have hypersegmented nuclei
- Bone marrow cellular with erythroid hyperplasia



# Treatment

- Folic acid 2-5 mg/day
- B<sup>12</sup> 1 μg/day

PEM