



# A REVIEW OF THE IRON DEFICIENCY ANAEMIA'S IMPACT ON CHILDREN'S CONSCIOUS DEVELOPMENT

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## ABSTRACT

A lack of iron progresses gradually through different stages. As evidenced by a decrease in the levels of hepatic non-heme iron in the newborns of iron deficient mothers, early iron shortage led to a depletion in iron reserves. The impact on the central nervous system, which results in deficiencies in human cognition and learning processes, is particularly significant. Strong evidence suggests that iron insufficiency is the primary cause of anaemia in many underdeveloped nations. Supplementation may, in some cases, but not always, prevent various iron deficiency-related abnormalities.

**Keywords:** Iron Deficiency, Anaemia, Heme-Free Iron.

## Introduction

With an estimated 2.5 to 5 billion people impacted, micronutrient deficiencies continue to be a serious public health issue, particularly in underdeveloped nations where infants and pregnant women are particularly at risk. Anaemia can be silent and symptom-free in its milder forms, but it can also cause weakness, weariness, dizziness, and sleepiness in more severe cases. Infants need additional attention since iron is actively transferred from the mother to the foetus during pregnancy, with the third trimester being the time when transfer is at its highest. As a result, depending on the gestational age, the premature newborn is delivered with comparatively decreased iron stores.

Medical research demonstrates that extremely severe anaemia is the root cause of maternal and infant death. The nutritional link between breastfeeding women and their newborns is particularly interesting in this context. The fact that brain iron intake peaks during periods of rapid neuronal growth is significant. There is significant evidence that iron deficiency is the primary cause of anaemia in many developing nations. Its effects range from mild iron reserve depletion to severe iron deficiency, and treatment in experimental circumstances may prevent some but not all deficits of iron deficiency anaemia.

## Iron deficiency diagnosed

The clinical examination, followed by laboratory confirmation using peripheral

blood results and serum iron studies, are the primary diagnostic tools for determining whether a patient has iron deficiency anaemia. Red cell indices, which measure the amount of haemoglobin in red blood cells, change depending on how severe and long-lasting anaemia is. Red blood cells that are iron deficient in the first stages may nevertheless show normal haemoglobin levels of 9 to 12 g/dl. Low MCV (55–74 fl), low MCH (25–30 g/dl), and an increased RDW (>16) are the characteristics of patients with hemoglobin levels < 9 g/dl. Examination of the morphology of peripheral blood smears alone is not fully reliable because (1) mild degrees of anaemia frequently result in normochromic and normocytic blood cells, and (2) when present, hypochromia and microcytosis may be caused by other conditions such as the anaemia of chronic diseases, sideroblastic anaemia, and thalassemia (11,12,13). The histochemical estimate of reticuloendothelial iron reserves in the aspirated bone marrow particles or biopsy specimens is the most accurate method for this goal.

In general, it has been discovered that the radioimmunometric assessment of serum levels of the iron storage protein, ferritin, correlates well with body iron stores (14). However, the lack of sophisticated infrastructure and the associated training prevents these tests from being widely used. The measurement of serum iron and iron binding capacity is an indirect technique that is more often employed, less expensive, and less uncomfortable for patients. However, in mild cases with hypoalbuminemia or an accompanying inflammatory condition combined with iron deficiency anaemia, a direct visual measurement of bone marrow iron

reserves is required for a definitive diagnosis.

### **Therapeutic presentation**

While menstruation, childbirth, or gastrointestinal illness are the most prevalent causes of iron deficiency in adults, nutrition is the main contributor in children. The introduction of cow's milk, exclusive breastfeeding for more than six months, and malnutrition are major contributing factors in youngsters (15).

If iron stores continue to be depleted, anaemia will inevitably get worse, causing tissue alterations as a result of steadily falling intracellular iron levels and iron-dependent enzymes brought on by protracted iron depletion (15).

The way iron deficiency manifests in children and adults, however, differs further. Both age groups share the characteristic that the illness will frequently be completely asymptomatic. In addition, children may exhibit small behavioural issues, recurring illnesses, or failure to thrive, all of which are all too simple to brush off as toddler-related issues (7, 10)

### **Development-related research**

There have been few studies done on older kids and teenagers, and those that have been done have inadequate methodological designs. It is yet unknown if anaemic and non-anaemic children and adolescents exhibit significantly different levels of intelligence on a statistical and clinical level (8). Reduced labor capacity and delayed psychomotor and cognitive development are the most dangerous possible effects of iron deficiency. Although this may be subtle in an individual child and therefore not truly a presenting symptom as such, there is

emerging evidence that .marked iron shortage can produce significant CNS impairments even in the absence of anaemia (13).

Children who are anaemic as infants have lower cognition, academic performance, and more behavioural issues as they become older, according to longitudinal research. Iron is involved in the synthesis, packaging, absorption, and breakdown of neurotransmitters into other iron-containing proteins, all of which may directly or indirectly affect how the brain functions. It most likely results from the brain's inability to receive iron during a certain stage of early brain development (12). This might be connected to human behaviour in young people as well as delayed motor deficiency. An amino acid known as GABA functions as a neurotransmitter. GABA is highly concentrated in the hypothalamus area of the brain, which indicates that it has a considerable impact on the function of the hypothalamus and pituitary (15). This implies that it aids in the body's overall hormonal synthesis and has a good impact on growth hormone levels.

The following are typical laboratory variables examined for the diagnosis of iron deficient anaemia:

- a) A trustworthy and sensitive criterion for determining the amount of iron reserves in healthy persons is serum ferritin. According to quantitative phlebotomy, 1 pg/I of serum ferritin equates to 8–10 mg of stored iron, and there is a strong correlation between serum ferritin concentration and mobilizable iron reserves. Widespread use of serum ferritin in clinical settings and population screening.

- b) Body iron stores are calculated by combining a number of laboratory markers, and they offer information on both the iron deficient and iron abundant populations. In relation to the storage compartment, body iron is expressed. The amount of iron that can be removed without causing a deficiency in the functional compartment is represented by a positive value. A negative number indicates an iron deficit and indicates how much iron must be reabsorbed by the body before iron stores can build up. Iron deficiency anaemia (abnormal haemoglobin and at least two other abnormal iron parameters) is identical to iron reserves of less than or equal to 300 mg. Subjects with sufficient iron storage have their iron stores quantitatively assessed using the serum ferritin level. The level of functional iron deficiency in people with iron deficiency anaemia is determined by the deficit in circulating haemoglobin. Estimating bodily iron storage has the primary benefit of defining the population's iron status.
- c) Iron deficiency anaemia is indicated by transferrin saturation levels below 15% and red cell protoporphyrin levels above 100 mug/100 ml packed red blood cells.
- d) Anaemia from an iron deficit is indicated by a haematocrit value less than 33%.
- e) If the result is less than 32 g/dl, the person has low iron status and is anaemic due to iron insufficiency. Children's age-specific reference ranges must be used to detect iron deficiency. Children who have serum ferritin levels below 10 or 12 pg/I are

likely to be iron deficient, and this test is frequently employed as the only indicator of iron status.

### **Infants' Cognitive and Motor Development**

The case for iron deficiency's detrimental effects on cognitive and psychomotor development is becoming more and more compelling. According to the motor, mental, and Denver development screening tests and the Nancey Bayley Scale of Development. Infants who had been anaemic for at least three months performed worse on tests than infants who had been anaemic for less than three months, according to studies. Infants with preanemic iron deficiency or moderate degrees of iron deficiency showed no substantial deficit. At haemoglobin values less than 10.5 g/dl, significant disparities in motor and mental development scores were seen (mildly anaemic).

### **Neurotransmitters and Iron**

Dopaminergic neurons require iron for proper growth and operation, and early alterations may cause lifelong harm.

There could be a number of pathways that connect anaemia to altered cognition. The most obvious one is the alteration of CNS structure and function (17). Without changes in haematocrit, there has been a considerable drop in non-heme iron levels in the liver and brain, which strongly suggests that the amount of iron in some tissues has decreased noticeably. Early iron shortage has a major impact on neurotransmitter receptors, which suggests that both excitatory and inhibitory central nervous system pathways are impaired (18). The regulation of the neurotransmitter receptors, which are in a state of dynamic equilibrium, rely on the

synthesis, metabolism, and several other elements of the signal transduction cascade.

The most serious functional limitations are brought on by iron deficiency anaemia, which involves tissue iron shortage as well as a decrease in circulating haemoglobin. Adolescents with iron-deficient erythropoiesis may have difficulty working or tolerating physical activity. Another clinical effect of iron shortage in adults has been proposed to be neurological impairment, but it is uncertain how severe this effect is (12). Iron status parameters and more exact psychometric assessments are needed. A more accurate indicator of iron status for use in this context may be the serum transferrin receptor. Low iron reserves have not been linked to any clinical consequences.

### **Conclusion**

Children at present and future risk of delayed development are identified by iron deficiency. Additionally, it has been determined that an iron deficit is typically accompanied by a number of negative psychosocial, economic, and biological outcomes. Studies have shown that even with iron supplementation, anaemic children under the age of two years have trouble catching up to non-anaemic youngsters. Children who were anaemic for longer than two years typically performed worse in school and had worse cognition than non-anaemic children. With repeated testing and therapy, they typically catch up in cognitive, but not in academic performance.

The GABA neurotransmitter system may experience irreversible disruptions and damage due to iron shortage throughout the fetus's brain development. The majority of early co-relational and

experimental investigations supported the idea that mild to severe iron insufficiency has a negative impact on cognitive development. Therefore, it may be reasonable to hypothesize that alterations in neurotransmitter receptors and the ensuing signal transduction process in the nervous system may be related to impairment of higher mental function such as cognition and learning in humans.

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