Iron deficiency in adolescent girls

Iron deficiency is the most widespread nutrient deficiency in Australia and is a common cause of anaemia in Western countries.[1,2] Adolescent girls are at risk. This article summarises current knowledge about iron metabolism, iron deficiency, and methods of investigating iron status, as well as prevention and treatment strategies for female adolescents.

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Iron is one of the most common metals on earth and an essential mineral. The role, location, absorption and loss of iron in the human body are described in the box on page 77.[3-6]

Iron deficiency is the most widespread single nutrient deficiency in both developed and developing countries. Adolescent girls are at risk for and have a high prevalence of iron deficiency with 9.2% of 15-year-olds in Australia found to have low serum ferritin and transferrin saturation levels. In comparison, the prevalence of iron deficiency in adolescent boys and premenarchal girls is approximately 0.5 to 2%.[7] Primary healthcare providers have a significant role in preventing, identifying and managing iron deficiency in adolescent females.

Causes of iron deficiency

Iron deficiency develops as a consequence of blood loss, dietary insufficiency and increased demands during growth and pregnancy. Rarely is diminished absorption, such as occurs in coeliac disease, a cause of iron deficiency.

Increased loss of iron

Menstrual disorders, such as menorrhagia or polymenorrhoea, are obvious causes of iron deficiency in females; however, the onset of menstruation as the main contributor to iron deficiency in adolescent girls is often underestimated. In girls, mean serum ferritin concentration has been shown to decline with the onset of menstruation rather than during the growth spurt, with

IN SUMMARY

- **Iron deficiency is common in adolescent girls.**
- **Risk factors include primarily blood losses with menstruation, followed by low dietary intake and increased demand due to growth.**
- **Laboratory measures to investigate iron deficiency include blood count and film, serum ferritin, and transferrin saturation; serum iron should not be used as a single parameter. For reliable interpretation, it is advisable to take blood when the patient is in a noninfectious state.**
- **In adolescent females, iron deficiency is considered present when the serum ferritin level is less than 12 µg/L and transferrin saturation is less than 16%. Additionally, in iron deficiency anaemia, the serum haemoglobin level is less than 120 g/L.**
- **The management of iron deficiency in adolescent girls involves menstrual regulation, a balanced diet and, in the case of anaemia, iron supplementation.**
- **The repletion of diminished iron stores requires months rather than weeks of iron therapy.**

the lowest levels occurring around three years after menarche.[8] Heavy menstrual loss (more than 80 mL per month) occurs in approximately 10% of teenage girls.

In the general population, chronic blood losses occur in the gastrointestinal tract – for example, in peptic ulcers, gastritis, or due to haemostatic defects. In infants, sensitivity to cow's milk is often a cause for gastrointestinal bleeding.

Iron deficiency occurs in athletes, especially in runners and swimmers. In runners, blood loss is explained by intravascular haemolysis due to foot strike and increased capillary flow. Additional iron losses result from sweating and gastrointestinal bleeding following training.[9,10]

Blood donation or venesection from self-harm should also be considered in teenagers with low iron stores.[11]

Inadequate dietary iron

On average, girls aged 13 to 16 years need 0.38 mg of extra iron per day to cover growth requirements.[12] The daily recommended dietary iron intake of 10 to 13 mg is met by only about a third of female teenagers, due to restrictive and purging eating behaviour and poor food choices.[6]

Levels of iron deficiency

The severity of iron deficiency may be classified into three stages:

- Stage 1: iron depletion – iron stores drop, while transport and functional iron are unaffected.
- Stage 2: iron deficient erythropoiesis – iron stores are exhausted, transport iron also decreases and the synthesis of haemoglobin and other functional compounds is compromised.
- Stage 3: iron deficiency anaemia – haem synthesis is reduced further and red blood cells become hypochromic and microcytic.

Figure 2 illustrates these stages, including typical laboratory findings. The risk factors for, symptoms, investigation and management of iron deficiency in adolescent girls are described below and summarised in the flowchart on page 79.

Signs of latent iron deficiency

Latent iron deficiency (which includes stages I and II, see above) occurs in approximately 10% of

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Figure 1. Iron deficiency occurs in athletes, especially runners and swimmers.

Iron in the body: how we use it, absorb it and lose it

The average total iron in females is 2.3 g (42 mg/kg body weight), and 3.8 g (50 mg/kg body weight) in males.[3]

Role

The main role of iron is as an oxygen carrier, but it is also involved in cell metabolism and thermoregulation.[4,5] During physical development, iron deficiency will adversely influence growth rate, body weight and cognition.

Location

Iron is located in three main compartments. It occurs as:

- Functional iron – predominantly in haemoglobin. A much smaller amount is in myoglobin, and even less is contained in respiratory enzymes. Functional iron is the major compartment.
- Storage iron – occurs as a soluble protein complex (ferritin) or as an insoluble complex (haemosiderin). Ferritin is found in virtually all body cells (particularly in hepatocytes) and in small concentrations in the plasma, where it reflects total body iron stores. Haemosiderin is mainly found in cells of the monocyte–macrophage system in the bone marrow, liver (as in Kupffer cells) and spleen.
- Transport iron – where iron is bound to transferrin. The third and smallest compartment, it has the highest turnover, with iron exchanged more than 10 times every 24 hours.

Absorption

Iron is absorbed mainly in the duodenum and upper jejunum. Mucosal surface, intestinal motility and intrinsic factors (such as hydrochloric acid, pancreatic proteases and bicarbonate) influence absorption.[6]

Loss

The body sheds approximately 1 mg of iron per day in faeces, cells shed from the gastrointestinal tract and skin, urine and sweat. Females of childbearing age lose an additional 0.3 to 0.5 mg of iron per day, due to menstruation.

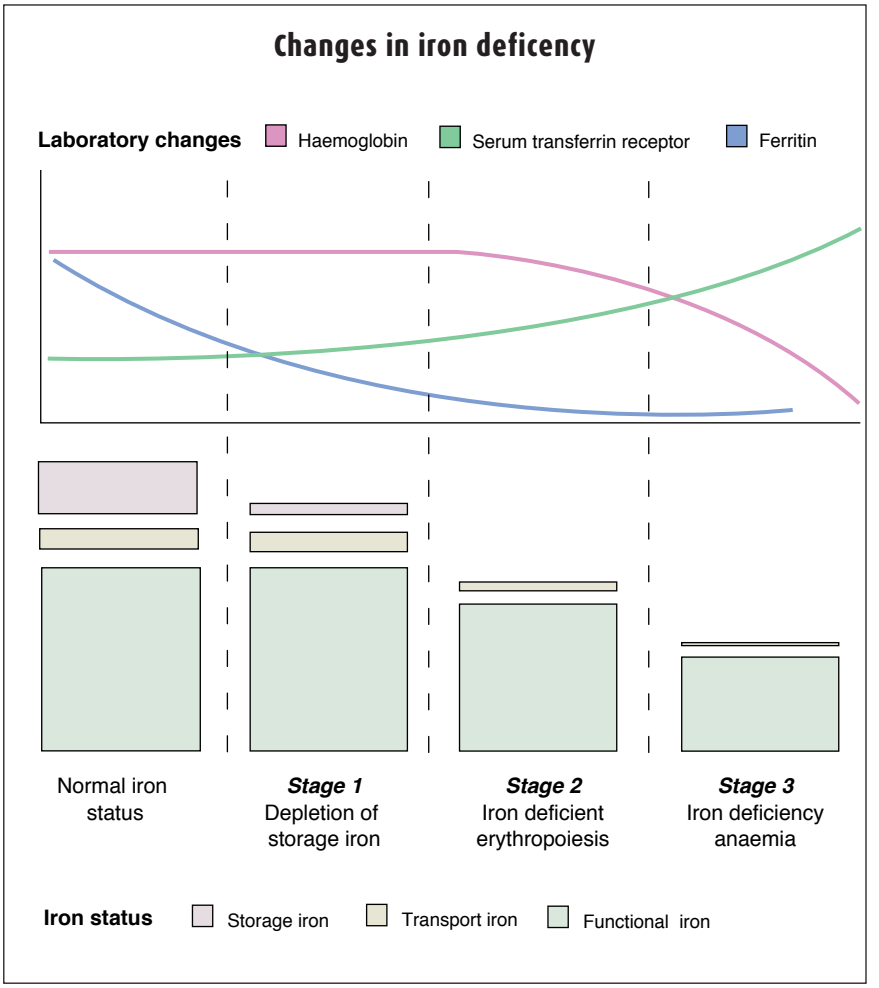

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Figure 2. Stages of iron deficiency with laboratory changes.

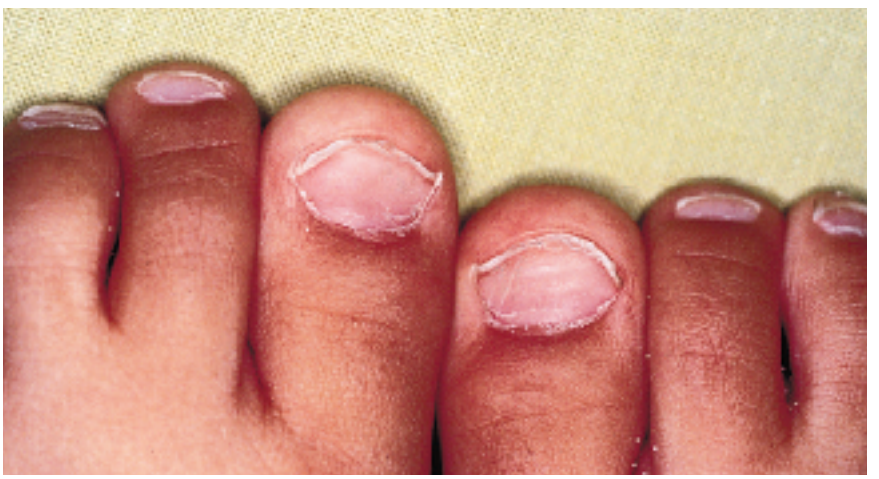

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Figure 3. Koilonychia (spoon nails). Concavity of the nail plate, seen in the toenails of a 10-year-old girl with iron deficiency.

adolescent girls. Symptoms related to latent iron deficiency include changes in cognitive functioning, declining academic performance, decreased concentration and memory, and fatigue.[13] Other symptoms reported in this context are headaches, paraesthesiae, taste disturbance, and diminished maximal exercise tolerance.[14] Iron deficiency also alters immunofunction.

Signs of iron deficiency anaemia

Common signs of iron deficiency anaemia such as pallor and tachycardia are well described. Tinnitus and taste disturbance are less common; abnormalities of the nails (koilonychia, Figure 3), hair (brittle, dry hair; diffuse hair loss) and skin (chronic inflammatory conditions, dermatitis herpetiformis, photodermatitis, pruritus and angular stomatitis) are rare.[15]

Assessment

Assessment of the adolescent girl with suspected iron deficiency should include a review of menstrual history, mental state, dietary history, sporting activity, physical examination, as well as a variety of laboratory tests that measure different aspects of iron metabolism.

Investigation

Initial investigations

Standard laboratory measures to evaluate iron status include a blood count, serum ferritin and transferrin saturation levels (see below). Table 1 gives the values of the primary measures that are indicative of iron deficiency in adolescent girls. Table 2 lists conditions that influence these and further laboratory parameters that can be measured in instances of suspected iron deficiency.

Red blood count and blood film

In iron deficiency, the first recognisable morphological change in the erythrocytes is mild ovalocytosis. Next, with a fall in haemoglobin and red cell count, comes microcytosis and hypochromia.

Managing iron deficiency in adolescent females

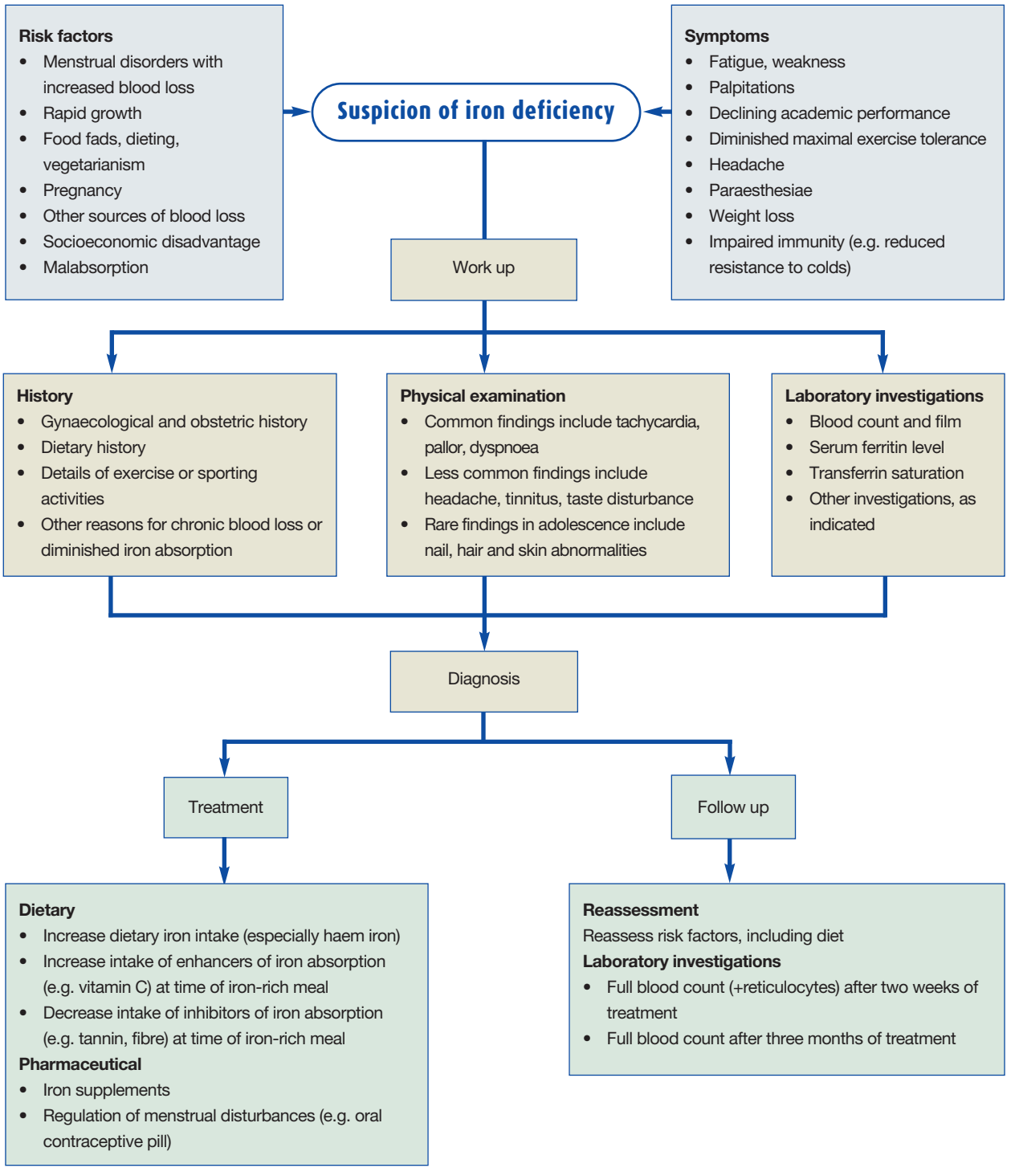

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Table 1. Laboratory measures indicating iron deficiency and iron deficiency anaemia in adolescent girls

Diagnosis	Laboratory measure	Level
Iron deficiency	Serum ferritin	< 12 μg/L
	Transferrin saturation	< 16%
	Haemoglobin	≥ 120 g/L
	Mean cell volume	≥ 78 fL
Iron deficiency anaemia	Serum ferritin	< 12 μg/L
	Transferrin saturation	< 16%
	Haemoglobin	< 120 g/L
	Mean cell volume	< 78 fL

Table 2. Disorders that influence laboratory parameters of iron deficiency

Measure	Influence	Condition
Serum ferritin level	Raised	Recent/current infection Chronic inflammation (e.g. arthritis) Chronic renal disease Malignancies Liver disease Gaucher's disease
Serum transferrin level	Raised	Oral contraceptive pill, pregnancy
	Reduced	Acute infection Chronic inflammation Malignancies Liver diseases Nephrotic syndrome Malnutrition
Serum iron level	Raised	During chemotherapy
	Reduced	Acute and chronic inflammation Malignancies Time of menstrual bleeding
EPC*	Raised	Infection, inflammation Lead poisoning
sTfR level†	Raised	Conditions with marked increased erythropoiesis (e.g. thalassaemia, megaloblastic anaemia)

*EPC = erythrocyte protoporphyrin concentration;
†sTfR = serum transferrin receptor.

In practice, a low or falling mean erythrocyte volume (mean cell volume [MCV]) is a commonly used indicator of iron deficiency; although thalassaemia should also be considered if the MCV is low. Figure 4 shows microcytic hypochromic red cells and ovalocytes.

Serum ferritin level

The serum ferritin level correlates with the total body iron stores; however, this correlation is not strictly linear. Ferritin is an acute phase reactant and values vary with acute or chronic inflammation, and may mask iron deficiency.

Serum iron level

Although the serum iron concentration is usually low in iron deficiency, it can be normal. Measurements are dependent on the laboratory methods used. Further, serum iron levels are also influenced by several physiological and pathological conditions. Moreover, concentration varies diurnally, with maxi-

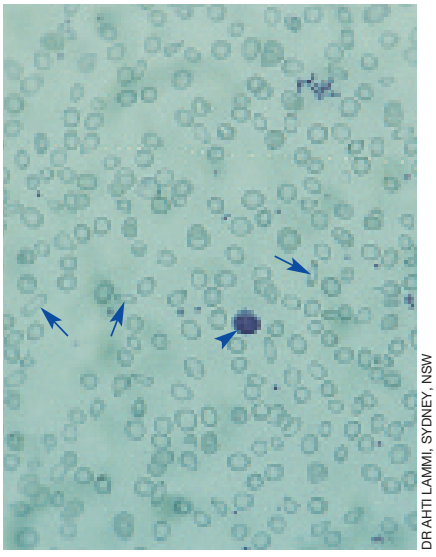

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Figure 4. Blood film showing microcytic hypochromic cells and ovalocytes (arrows) consistent with iron deficiency. The red cells are smaller than a lymphocyte nucleus (arrowhead), whereas a normochromic red cell would have the same size as the lymphocyte nucleus shown.

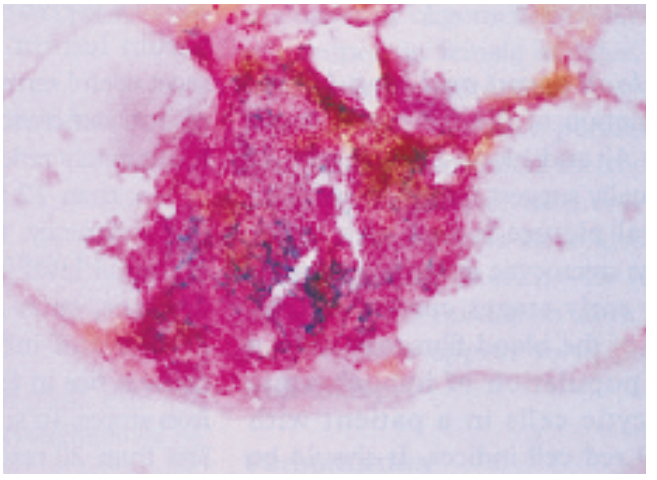

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Figure 5a. Normal bone marrow.

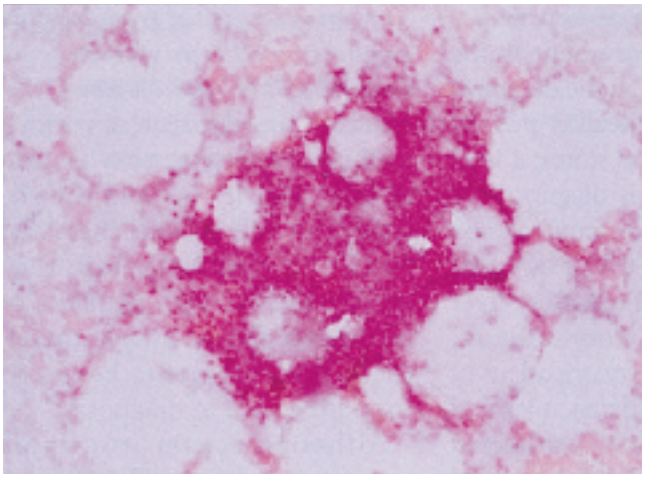

Figure 5b. Bone marrow aspirate from an iron deficient patient.

mal levels in the morning, and a nadir around 9 o'clock at night.

Serum iron concentration is reduced in acute infection, chronic inflammation or malignancy, whereas with chemotherapy it is increased. Higher levels are found after a meal, and oral iron medication may produce transient higher levels, even when iron deficiency anaemia is still present.

Transferrin saturation

The level of transferrin saturation indicates the extent to which iron-binding sites in the transport protein transferrin are saturated. Transferrin saturation alters after iron stores are depleted and thus is less sensitive to iron depletion than serum ferritin. Non-iron-related factors influencing transferrin saturation are listed in Table 2.

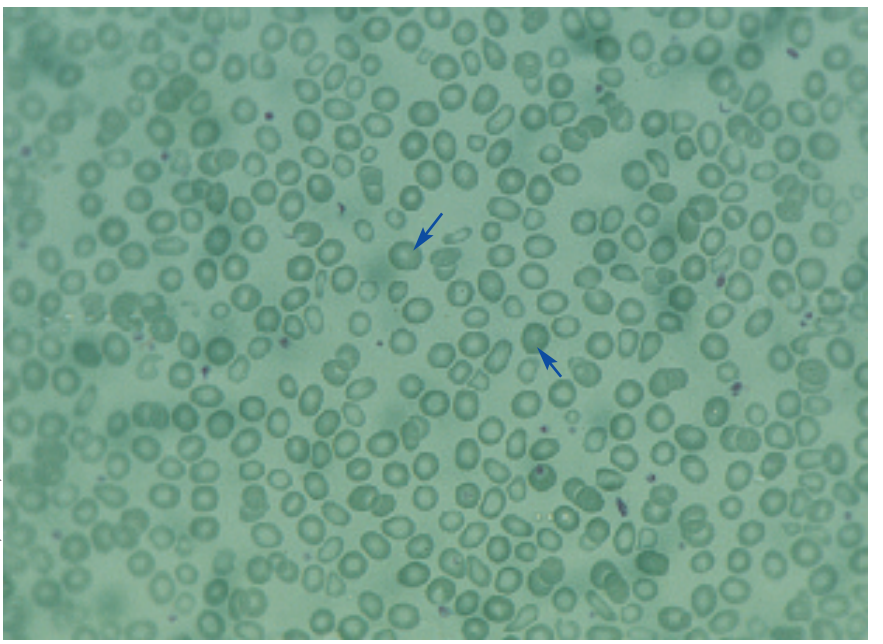

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Figure 6. Blood film showing early effect of treatment of iron deficiency. Normocytic, normochromic cells (arrows) are seen with residual iron-deficient erythrocytes.

Further investigations

Serum erythrocyte protoporphyrin level

Erythrocyte protoporphyrin is a precursor of haemoglobin. When iron depletion impacts on haemoglobin production the level of erythrocyte protoporphyrin increases. Lead toxicity is another cause of raised erythrocyte protoporphyrin. This test is used in some laboratories in Australia.

Serum transferrin receptor level

A more recent investigation to detect iron deficiency before the development of anaemia is the serum transferrin receptor. This is a transmembrane protein on the cell surface that is involved in cellular iron uptake. Iron deprivation leads to prompt induction of serum transferrin receptor synthesis. Small amounts are found in the serum and correlate with the amount of tissue iron. Soluble serum transferrin receptor has been shown to increase in iron deficiency but not in conditions associated with infection, anaemia of chronic disease and liver disease, and pregnancy, but at this time is not routinely used in Australia.[16]

Bone marrow aspirate and iron stain

The bone marrow aspirate sample (Figure 5) remains the gold standard

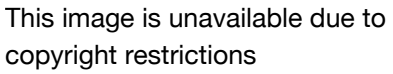

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Figure 7. About one-third of the daily iron intake should come from a mixed breakfast – milk, iron-fortified cereal and toast.

for the diagnosis of iron deficiency and is of value in the presence of inflammatory conditions and liver disease.[17]

Management

Dietary changes and limiting ongoing iron loss are paramount in the treatment of iron deficiency; however, pharmacological treatment may also be indicated in some circumstances.

Treatment of iron deficiency is effective in improving the physical, cognitive and laboratory indices associated with iron deficiency (see Figure 6).

Diet

Iron in food

There are two major types of iron found in food:

- haem iron (in the soluble ferrous form, Fe^{2+})
- non-haem iron (in the insoluble form, Fe^{3+}).

Good dietary sources for both types are listed in Table 3. Approximately 25% of haem iron is absorbed from a 'mixed' meal containing different foods from different food groups. Non-haem iron is not well absorbed and needs to be reduced to the Fe^{2+} form to promote absorption. Table 4 lists dietary enhancers and inhibitors of iron absorption.

Daily requirements

The recommended daily iron requirements of children, adolescents and adults are given in Table 5.[18]

Teenagers need to be encouraged to eat a variety of different foods from the five food groups. About one-third of the daily iron intake should come from a mixed breakfast (see Figure 7), consisting of milk, iron-fortified breakfast cereal and toast. Nearly one-fifth (17%) of adolescent girls regularly miss breakfast,[19] which is a major factor accounting for the low dietary iron and calcium levels reported in this group.[20] This proportion rises to 44% in girls of lower socio-economic status.[21]

To maximise the absorption of dietary iron, it is necessary to choose iron-rich foods and combine a variety of different foods at mealtimes. Table 6 illustrates how the dietary needs of adolescent females may be achieved through a combination of food group variety, attention to good dietary sources of iron and combining foods to ensure maximum iron absorption.

Table 3. Food sources of haem and non-haem iron

Foods	Iron (mg)
Containing haem iron	
Liver (100 g)	11.1
Kidney (50 g)	5.7
Beef (120 g)	3.6
Lamb (120 g)	3.0
Chicken (120 g)	0.8
Fish (120 g)	0.5
Pork (1 chop)	1.8
Salmon or tuna (150 g)	2.6
Containing non-haem iron	
Egg (1 whole)	0.9
Wholemeal bread (2 slices)	1.4
White bread (2 slices)	0.6
Breakfast wheat biscuits (2 biscuits)	2.6
Wholemeal pasta (1 cup, boiled)	3.1
White pasta (1 cup, boiled)	0.7
Bran breakfast cereal (30 g)	5.4
Cornflakes (30 g)	2.8
Baked beans, lentils (½ cup, cooked)	2.2
Spinach (½ cup, boiled)	2.2
Broccoli (⅔ cup, boiled)	1.0
Peanut butter (2 tablespoons)	1.0
Nuts (15 g)	0.6
Dried apricots (10 halves)	2.2
Milo or Ovaltine (2 tablespoons)	2.8
Brown rice (1 cup, boiled)	0.8
White rice (1 cup, boiled)	0.6

Eating a variety of foods will ensure an adequate intake of the nutrients required for haemopoiesis apart from iron, such as folic acid and vitamin B_{12}.

Achieving good food habits in adolescence will help to reduce the recurrence of iron deficiency in adulthood.

Groups with special needs

Vegetarians

Many teenagers believe that they can lose weight by following a vegetarian diet. Although a vegetarian diet can be nutri-

Table 4. Dietary factors which inhibit and enhance the absorption of dietary iron

Inhibiting factors
Polyphenols (e.g. red wine)
Tannins (e.g. tea, vegetables, coffee)
Phytates (e.g. cereals, legumes)
Phosphates (e.g. eggs)
Oxalates (e.g. leafy green vegetables)
Phosvitin (e.g. egg yolks)
Enhancing factors
Haem-iron containing foods (e.g. meat)
Muscle protein (e.g. meat, fish, chicken)
Acid pH
Vitamin C-rich foods (e.g. fruit juice, vegetables)
Citric acid
Lactic acid
Sugars

Table 5. Recommended dietary intake (RDI) of iron per day

Infants	RDI of iron (mg/day)
0 to 6 months	
Breast-fed*	0.5
Bottle-fed†	3
7 to 12 months	9
Children	
1 to 11 years	6–8
Adolescents	
12 to 18 years	10–13
Adults	
Men	7
Women	
Menstruating	12–16
Postmenopausal	5–7
Pregnant‡	22–26
Lactating	12–16

* 50% of the iron in breastmilk is absorbed.
† RDI based on 10% absorption of iron from infant formula and infant cereals.
‡ RDI in the second and third trimester

Table 6. Example of a healthy eating plan to ensure an adequate dietary iron intake in adolescent girls*

Meals	Iron (mg)
Breakfast	
Breakfast cereal (30 g)	2.8
Milk (½ cup)	0.0
Toast (wholemeal, 1 slice)	0.7
Butter/margarine (1 teaspoon)	0.0
Fruit juice	Neg†
Morning snack	
Banana	0.7
Water	0.0
Lunch	
Bread (wholemeal, 2 slices)	1.4
Chicken (40 g)	0.3
Salad (on sandwich)	0.5
Flavoured milk (300 mL)	0.1
Afternoon snack	
Toast (wholemeal, 1 slice)	0.7
Butter/margarine (1 teaspoon)	0.0
Soft drink (1 can)	Neg†
Evening meal	
Pasta (boiled, white, 1 cup)	0.7
Meat and tomato sauce (1 cup)	3.0
Grated cheese (2 tablespoons)	Neg†
Vegetables or salad	1.2
Ice cream (2 scoops)	0.5
Tinned fruit	0.7
Chocolate syrup	Neg†

* The recommended dietary intake (RDI) of iron for adolescent girls is 10 to 13 mg and the total iron intake of this example is 13.3 mg with breakfast accounting for 29% of intake.
† Neg = negligible, meaning less than 0. 1 mg of iron.

tious and well balanced, non-haem sources of dietary iron need to be carefully selected and combined, at the same meal, with foods that contain enhancing factors (see Tables 3, 4 and 6).

Athletes

Adolescent girls involved in sporting competitions and sports training should be monitored for iron deficiency, fad diets, eating disorders and dietary iron intake. Because of their high phytate content, high carbohydrate diets prescribed to enhance athletic performance may increase the inhibition of iron absorption.[22]

Pharmacological treatment

Iron supplementation

Iron supplements and iron rich foods are commonly prescribed.[23] The success of these interventions is dependent upon adherence to the prescribed therapy as well as ensuring that potential dietary inhibitors of iron absorption are avoided.

Daily doses of 10 to 15 mg of elemental iron are recommended for maintenance of iron status. Regular multivitamin preparations contain around 2 to 5 mg of iron. Iron tablets contain from 30 to over 100 mg iron.

Patients should be forewarned of the changes to their stool colour (blackening). The potential risk for abuse and overdose with iron supplements should also be considered prior to prescription. For adolescents, sequelae such as gastrointestinal upset are not commonly observed during oral iron replacement therapy.

Debate continues about the optimal dosing routine as well as optimal length of treatment. Iron supplementation has been prescribed in a number of differing regimes. There is no consensus as to whether daily or weekly therapy is best. Certain high risk populations benefit from supervised oral therapy, as has been shown with treatment for tuberculosis and other micronutrient deficient states.[24]

Continuing iron supplementation for

one or two months after normalisation of haematological indices has been suggested by several authors. This is the usual practice in children, in whom a dose of 6 mg of elemental iron per kg per day is given. Recent recommendations for 'high risk' populations, such as athletes and adolescent females, include continuing oral iron therapy for a prolonged period – for example, a dose of up to 200 mg of (ferrous) iron per day, best taken in combination with fruit juice, a vitamin C supplement or other enhancer of iron absorption.[25] The introduction of new measures such as the transferrin receptor assay will improve the ability to predict the point at which iron stores are replete.

Oral contraceptive pill

For a range of menstrual disturbances, prescription of the combined oral contraceptive pill may be needed to limit or suspend menstrual bleeding.

Reassessment

In assessing recovery for adolescents with iron deficiency anaemia it is recommended that a full blood count (including reticulocytes) be requested after two weeks of treatment, to document the rise in haemoglobin, particularly if compliance is an issue. Further testing is strongly recommended after three months of iron. A full blood count, film and serum ferritin level should be requested, to document the response to the treatment (see Figure 6). A persistent low serum ferritin level may indicate insufficient compliance, poor diet, ongoing chronic blood loss or other persisting risk factors. Should these initial measures not be effective, referral to a specialist unit is recommended.

Conclusion

Adolescent females have a high prevalence of iron deficiency, and approximately 10% of adolescent girls in Australia are classified as iron deficient. Iron deficiency in young girls occurs mainly as a result of blood loss due to menstruation, growth, poor diet and insufficient absorption of dietary iron. Symptoms of iron deficiency include fatigue, decreased academic performance, reduced physical fitness and impaired immune response. Investigation of iron deficiency involves monitoring adolescent females for fad diets, their involvement in sports and by reviewing the menstrual history, as well as physical and laboratory examination.

Some measures allow the detection of iron deficiency before anaemia is present. Providing sound nutritional advice to ensure adequate levels of well-absorbed dietary iron is essential in the prevention and treatment of iron deficiency as is pharmacological regulation of menstrual disturbances and, where needed, iron supplementation. MT

A list of references is available on request to the editorial office.

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