# Nutrition and Hair
## Deficiencies and Supplements

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**KEYWORDS**

• Nutrition • Hair • Hair loss • Alopecia • Malnutrition • Deficiency • Diet • Supplements

**KEY POINTS**

- A caloric deprivation or deficiency of several components, such as proteins, minerals, essential fatty acids, and vitamins, caused by inborn errors or reduced uptake, can lead to structural abnormalities, pigmentation changes, or hair loss, although exact data are often lacking.
- Acquired reasons for nutrition-related hair growth disorders are combined or specific deficiencies due to malnutrition, inadequate diets, or insufficient parenteral alimentation or malabsorption in gastrotintestinal disease.
- The evidence on dietary supplements in hair disorders is limited, combinations containing l-cystine are studied best.

### INTRODUCTION

Hair follicle cells have a high turnover. Their active metabolism requires a good supply of nutrients and energy. A caloric deprivation or deficiency of several components, such as proteins, minerals, essential fatty acids, and vitamins, caused by inborn errors or reduced uptake can lead to structural abnormalities, pigmentation changes, or hair loss, although exact data are often lacking.[1] Combined deficiencies are not uncommon, especially in malnutrition.

In developed countries, hair growth disorders caused by nutritional deficiencies in healthy individuals are rare and tend to be overestimated by patients and physicians, especially concerning vitamins. National and International Institutions have established recommended daily allowances of many nutritional components. In the United States, the National Institutes of Health has published recommendations for the daily reference intake of micronutrients and macronutrients and the maximum daily intake that will likely not cause adverse effects.

Dietary supplements have traditionally been used unspecifically to improve hair growth, a few of which have been studied systematically in animals and humans.

### CLINICAL DIAGNOSIS

The diagnosis is based on a careful history of nutritional habits including 3 to 4 months before the hair problem. Clinical signs include a diffusely positive pull test, hair diameter, color, and quality changes or fragility as well as skin and nail changes. The latter changes are easier to recognize by holding a contrasting white or black paper (hair card) behind the tips of the hairs and by using trichoscopy and videotrichoscopy. A trichogram or digital phototrichogram may reveal increased rates of telogen. Laboratory blood tests should be targeted based on the suspected deficiency.

Hair analysis is often marketed as a tool to diagnose deficiencies and intoxications as causes of hair loss and structural hair changes. However, the use of hair analysis in a hair clinic is very limited, because there are no laboratory standards and no clear correlation between hair components and the nutritional status has been established. The hair content can be influenced by polluted air and other external factors. Concentrations can also be influenced by changes in hair growth speed because of variations in nutritional status.

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## SPECIFIC DEFICIENCIES

### *Malnutrition and Weight Loss Diets*

Marasmus is a diet low in calories, whereby amino acids are used to provide energy and are not available for tissue and plasma protein synthesis and other functions. The glycogen content of the follicular sheath is reduced, providing less energy for cell mitosis. The hair is thin, sparse, fragile, and even more easily shed than in kwashiorkor (see later discussion), whereas lanugo body hair may be increased.[2] Organs show atrophy. Hair morphology in children and their mothers has been used to detect malnutrition in developing countries.

Diets for weight loss can also lead to hair loss, especially if the daily calorie intake is less than 1000 kcal and if protein intake is inadequate. This cause should be suspected especially in young obese women.[3] The hair loss may be more profound in diets with a negative nitrogen balance (loss of lean body mass) and be partly due to reduced thyroid activity.

### *Proteins*

Protein is the major constituent of hair fibers. Therefore, a reduced protein uptake can impair hair growth, even before serum albumin levels are decreased.[4]

Kwashiorkor is a result of a low protein intake in a calorically normal diet. It is characterized by reddish, short, dull hair and a telogen and dystrophic effluvium. Hairs can be plucked easily.[5]

In early stages, anagen bulbs are atrophic,[6] and hairs are already reduced in diameter, often intermittently. Hair elasticity is reduced, and hairs feel weaker and softer. Hair color changes may also vary along the hair shaft depending on the nutritional situation,[7] leading to red or light bands in dark hair. Brown hair may become blonde. The skin is hypopigmented and partly dry. Edema and anemia are typical.

Other situations of low protein uptake are infants on special diets, such as in urea-cycle disorders, milk-free diets, gastrointestinal disease, blood loss and blood donations, anorexia nervosa,[8] depression, drug addiction, or malignancy.

### *Vitamin C*

Ascorbic acid is essential for collagen synthesis and cross-linkage of keratin fibers. The reference daily intake for men is 90 mg and for women is 75 mg. A deficiency is called scurvy and often occurs in elderly patients, alcoholics, and patients with chronic disease. Hair changes are corkscrew hairs, perifollicular hyperkeratosis and hemorrhage, follicular plugging, and curling caused by changes in the perifollicular fibrous tissue. Other symptoms include ecchymoses, bleeding gums, chronic wounds, and infections.

### *Biotin*

Vitamin H is a crucial cofactor of carboxylases in the mitochondria.[9] Biotin deficiency is rare because it is also produced by intestinal bacteria. It has been seen in congenital or acquired biotinidase or carboxylase deficiency,[10] parenteral alimentation, impaired gastrointestinal flora caused by antibiotics, and after excessive ingestion of raw white eggs due to binding by avidin. Symptoms include structural changes of the hair and nails, perioral dermatitis, conjunctivitis, and infections. Alopecia is not a typical symptom of biotin deficiency but trichorrhexis nodosa and other structural anomalies can occur. The reference daily intake for adults is 30 μg. Antiepilileptic drugs can reduce biotin levels; a prophylactic supplementation can therefore be recommended.

It has not been sufficiently shown that additional supplementation of biotin in patients with normal blood levels can improve hair loss, although an effect on hair and nail structure is possible.

### *Vitamin B12*

A deficiency of cyanocobalamin is seen in vegetarians, fish bandworm infestation, and various gastrointestinal disorders, including atrophic gastritis with antibodies to intrinsic factors leading to pernicious anemia. It can cause gray hair, megaloblastic anemia, peripheral neuropathy, Hunter glossitis, and angular cheilitis. The reference daily intake for adults is 2.4 μg.

### *Zinc*

Zinc is an essential cofactor of several metaloenzymes and transcription factors. The required daily zinc uptake of 8 to 10 mg per day is usually supplied through a normal diet, but deficiencies are still common in developing countries.[11]

Zinc deficiency can lead to telogen effluvium, thin white and brittle hair, as well as nail dystrophy, a seborrhoic and later psoriasiform acral and perioral dermatitis, cheilitis, blepharoconjunctivitis, infection, and skin superinfection with *Candida albicans* and *Staphylococcus aureus*.[12] Other symptoms are diarrhea, neurologic disturbances, and growth retardation. Histology shows pale superficial epidermal cells and single-cell necrosis.

The deficiency can be genetic, presenting after weaning because of an autosomal-recessive absorption disorder called acrodermatitis enteropathica, with an additional reduced uptake of

desaturated fatty acids and inadequate desaturation of linoleic acid and alpha-linoleic acid to their long-chain metabolites.[13] The hair shows diameter narrowing,[14] and polarized microscopy may show irregular bands, such as in trichothiodystophy.[15] Occasionally, serum zinc levels are normal. A clinical response to zinc supplementation confirms the diagnosis.

Acquired zinc deficiency occurs in elderly persons, in persons with alcoholism, anorexia nervosa, nephropathy, pancreatitis, after prolonged breast feeding without supplementation, following gastrointestinal bypass surgery, from cereals containing phytate, because of excessive intake of iron, and because of drugs that chelate zinc, such asangiotensin-converting enzyme inhibitors.

During treatment, zinc levels should be monitored because overdose can lead to copper or calcium deficiency, drowsiness, and headache. The daily reference intake for men and pregnant women is 11 mg and for women is 8 mg. In deficiency, the recommended dose for adults is 25 to 50 mg of elemental zinc and 0.5 to 1 mg/kg for children.

Although traditionally used in unspecific hair treatments, an effect of zinc supplementation on hair growth in patients with normal serum zinc levels has not been sufficiently proved.[16]

### Niacin

Vitamin B3 is an essential component of amide adenine dinucleotide connecting the citric acid cycle to the oxidative phosphorylation required for adenosine triphosphate production and thus cell energy supply. The recommended daily intake is at least 13 mg. A deficiency is called pellagra, meaning “rough skin.” The major symptoms are a photosensitive dermatitis with hyperpigmentation, diarrhea, dementia, and finally, death (the 4 Ds). Early signs are diffuse hair loss, weakness, irritability, glossitis, and stomatitis.

Pellagra occurs in areas where maize and millet are the main food, such as parts of Asia, Africa, and India. Other reasons are impaired food intake, Crohn disease, tumors that impair niacin metabolism such as carcinoid, and drugs such as isonicotine hydrazide. The reference daily intake for male adults is 16 mg and for women is 14 mg.

### Essential Fatty Acids

Linoleic acid and alpha-linoleic acid are required for normal human metabolism. They are an important component of cell membranes and lamellar bodies of the stratum corneum. A deficiency was seen in inappropriate parenteral nutrition and in children with impaired uptake, such as in children with biliar atresia, who were put on a diet rich in triglycerides, and in cystic fibrosis patients. Hair loss of the scalp and eyebrows and depigmentation are symptoms, among other complaints.[17]

### Iron

Iron deficiency is ranked as the world’s most common deficiency by the World Health Organization, affecting up to 80% of humankind.

Iron works as a catalyst in oxidation and reduction reactions, and it may control DNA synthesis through the enzyme ribonuclease in dividing cells. Its deficiency causes microcytic and hypochromic anemia. Even in the absence of anemia, diffuse hair loss and other skin symptoms, such as glossitis, cheilitis, and kolonychia, can occur. The impaired keratin production can lead to thinner anagen hairs. In African hair, band-like color changes have been reported.

Severe deficiency can cause fatigue, weakness, pale conjunctivae, and tachycardia because of anemia.

Iron is functional in hemoglobin within erythrocytes and in myoglobin and enzymes, stored in ferritin and transported in transferrin. For laboratory tests, sufficient ferritin levels are essential, reflecting the iron reserve.

In premenopausal women, iron deficiency is often due to menorrhagia or pregnancy, whereas, especially in older patients, gastrointestinal bleeding should be excluded. Other reasons may include a vegetarian or vegan diet, hookworms, nephropathy and dialysis, frequent blood donations, surgery, or chronic inflammatory bowel disease.

In at-risk groups, sufficient intake of red meat is important; other heme iron sources include clams and fish. Non-heme iron sources, such as beans, peas, and cereals, should be eaten together with sources of vitamin C. Excessive consumption of coffee and tea should be avoided.

The daily reference intake is 8 mg for men and 18 mg for women between 19 and 50 years of age; different values apply for children, seniors, and pregnant or lactating women.

The required iron levels and their significance for hair loss are still an object of scientific discussion, with several studies coming to different conclusions.[18–22]

Most authors consider a ferritin level of at least 40 mg/L as adequate in their female patients; others only require 10 mg/L, and some require 70 mg/L. To correct iron deficiency, ferrous fumarate, ferrous lactate, ferrous gluconate, or ferrous sulfate should be taken for several weeks in 2 to

3 daily doses, because absorption is lower in high doses. The latter 2 doses may be better tolerated. For the treatment of low iron deficiency anemia, the Centers for Disease Control and Prevention recommends 50 to 60 mg of oral elemental iron twice daily for 3 months, which corresponds to 325 mg ferrous sulfate twice a day. Gradually increasing the dose or intake with food may minimize gastrointestinal side effects. Iron overload is more common in adult men and postmenopausal women. It should be avoided, because it can cause tissue damage and fibrosis and exacerbate hemochromatosis.

### *Copper*

Copper is crucial for aminoxydases required for oxidation of thiol groups to dithio- crosslinks, which are essential for keratin fiber strength. Some enzymes also depend on copper, such as ascorbic acid oxidase and tyrosinase.

In children, a rare autosomal-recessive malabsorption disorder may be present. Hypopigmented hair and pili torti are typical in Menkes kinky hair syndrome,[23] as well as a degeneration of brain, bones, and connective tissue. including arterial occlusion, and pale and lax skin. The treatment consists of infusions with copper salts.

An acquired deficiency is also seen in premature babies, inadequate cow milk, or parental alimentation and after a longer zinc therapy.[24] It presents with hypopigmented hair, microcytic anemia, leukopenia, and myelopathy. The reference daily intake for adults is 900 μg.

### *Selenium*

Selenium is an important component of glutathione peroxidase, an antioxidant system. A deficiency has been reported in areas of soil with low selenium content and in parenteral nutrition. Although symptoms are mostly muscular and cardiac, hypopigmentation of the hair and skin can occur. The reference daily intake for adults is 55 μg. Selenium intoxicants from overdosed supplements have been reported.

### *Vitamin A*

Although vitamin A deficiency is not an established cause of hair loss, an excessive intake can lead to general hair loss and dry skin. The recommended maximum daily intake is 10,000 IU.

### *Vitamin D*

The role of vitamin D for hair growth is still under investigation.[25] Several studies in animals in vitro and in vitamin D–resistant rickets suggest a role for vitamin D in hair growth,[26,27] although no relation to male baldness or alopecia areata could be shown.[28,29] Therefore, obtaining a Vitamin D3 level in patients with telogen effluvium can be helpful. The reference daily intake for adults is 5 to 10 μg (1 μg calciferol = 40 IU vitamin D).

## TOXINS

The ingestion of toxins such as thallium, arsen, and mimosin in a plant called *Leucenia glauca* and others can cause hair loss and/or hair breakage. Acrodynia after mercurium intoxication has become rare. It is characterized by scalp hair alopecia and hypertrichosis on the arms, legs, and sometimes the trunk.

## NUTRITIONAL SUPPLEMENTS

Although many nutritional supplements have been used traditionally to treat hair disorders, there is limited evidence of their use in non deficient patients.

Long-term effects of antioxidants on hair aging may be possible, but are difficult to assess. The amino acid taurin has been shown to promote follicle cell survival in vitro[30]; it is combined with the polyphenol katechin and other ingredients. L-carnitine has been shown to stimulate hair follicle cells in vitro.[31] Components derived from soybeans may also have an effect on hair growth through anti-inflammatory and estrogen-dependent mechanisms,[32,33] but studies in vivo are lacking and an increase of angioma rubi has been reported.[34]

Orthosilicic acid increased hair tensile strength and thickness in a controlled study after 6 months[35] and decreased hair brittleness in a study after 20 weeks.[36]

From studies with sheep, it has been snown that additional intake of L-cysteine can improve wool production.[37] An antioxidant effect of L-cysteine is also suspected. Later, studies in humans have shown a significant effect in the treatment of diffuse telogen effluvium. In a randomized, placebo-controlled study in 30 women, a supplement of L-cysteine in combination with medicinal yeast and pantothenic acid led to a normalization of the rate of anagen after 6 months, whereas a placebo did not.[38] Clinically, this supplement of L-cysteine would correspond to avoiding a temporary loss of several thousands of long hairs. Further studies are needed to increase the evidence on nutritional supplements on hair.

## SUMMARY

Various genetic or acquired malabsorption deficiencies or insufficient uptake of nutrients can

influence hair growth. The diagnosis is established through a careful history and clinical examination. Typical clues are a diffusely positive pull test confirmed by a trichogram or digital phototrichogram, changes in hair diameter, structure and strength including broken or brittle hairs, and pigment changes seen in trichoscopy.

Laboratory blood tests confirm the specific deficiency. Relevant iron deficiency can be assumed at ferritin levels less than 40 mg/L. Specific treatment of the deficiency will lead to improved hair parameters within 3 to 6 months. Unspecific treatment of hair loss without confirmed deficiencies has only been effective in telogen effluvium with a supplement containing L-cysteine, but otherwise can not generally be recommended.

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