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# Role of Diet in Hair Growth

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## Introduction

Patients with hair loss frequently ask if nutritional supplements might help them regrow their hair or keep it from falling out. Many people will start taking dietary supplements without consulting a doctor in the hopes that they will assist. This population's vulnerability is being exploited by the unregulated supplement sector. While hair follicles are among the most metabolically active in the body, and calorie and protein deprivation, as well as vitamin deficit, can affect hair growth, the relationships are complicated.

Hair structure and hair development can both be affected by nutritional deficiencies. Acute Telogen Effluvium (TE), a wellknown result of abrupt weight loss or decreased protein intake, as well as the diffuse alopecia seen in niacin insufficiency, can both affect hair development. Nutritional inadequacy has also been linked to chronic TE, Androgenetic Alopecia (AGA), Female Pattern Hair Loss (FPHL), and Alopecia Areata (AA). Because of this well-established association, many patients seeking hair loss treatment inquire about dietary suggestions. Is it necessary to check for vitamin insufficiency in a patient who is experiencing hair loss? Is there anything that should make you want to get tested? Is there any evidence to justify the use of micronutrient supplementation in the absence of such risk factors?

Physicians must be ready to respond to these inquiries. Hair loss is prevalent, with about half of men and women experiencing pattern hair loss by the age of 50. Hair loss remedies are sold as several dietary supplements. Given consumer marketing activities, physicians must be able to reply with an evaluation of the available data. It's worth noting that such supplements are not without risk. Supplementation may be damaging to hair in the absence of a deficiency. Hair loss has been linked to excessive intake of some minerals, such as selenium, Vitamin A, and Vitamin E. As a result, it's unexpected that some best-selling hair supplement contains both vitamin A and vitamin E, while the next combines selenium, vitamin A, and vitamin E. Despite the fact that such products include a variety of nutrients, a review of the medical literature reveals that there is little evidence to support their usage. Much of what we know about the effect of nutrients on hair loss comes from illness conditions that cause deficiencies. There is currently a scarcity of research on the effects of supplementing in people who are not deficient in any nutrients. In this work, we review the available literature

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on nutrient deficiencies that cause hair loss, discuss the risk factors for these deficiencies, and examine the evidence for the favourable and detrimental effects of supplementing on hair loss.

## Various Factors Important for Hairs Health

### Vitamin E

Tocotrienols and tocopherols are antioxidants that belong to the vitamin E family. Hemolytic anaemias, neurologic abnormalities, and skin dryness are all symptoms of deficiency. Vitamin E insufficiency is uncommon, however, it can occur as a result of fat malabsorption problems.

There is very little evidence in the literature about the effects of vitamin E supplementation on hair loss. When compared to a placebo group, 21 volunteers who got tocotrienol supplementation (100 mg of mixed tocotrienols daily) showed a substantial increase in hair number. Excessive supplementation, on the other hand, might cause hypervitaminosis E, which can raise the risk of bleeding and reduce thyroid hormone production. Furthermore, there is some evidence of a negative influence on hair development, as demonstrated in volunteers who took 600 IU per day for 28 days, which is around 30 times the daily recommended dose.

### **Biotin**

Biotin (also known as vitamin H) is a cofactor for carboxylation enzymes. Incubation in biotin-containing solutions boosted DNA concentration and protein synthesis in isolated sheep hair follicles. Eczematous skin rash, baldness, and conjunctivitis are all signs of insufficiency. In one study, a newborn who was fed a formula deficient in biotin developed periorificial dermatitis and patchy baldness, both of which improved with daily oral biotin supplementation. Biotin shortage is uncommon since intestinal microorganisms can usually manufacture enough biotin. Biotinidase or carboxylase insufficiency, antibiotic use upsetting the gut flora, and antiepileptic use are all examples of deficiency. Excessive intake of raw egg whites might cause a deficiency due to avidin binding. In the absence of a deficiency, no scientific research has proven that biotin supplementation can help with hair loss.

#### **Antioxidants**

Antioxidants are substances that can neutralise Reactive Oxygen Species (ROS) and so protect cells from oxidative damage. Antioxidants include zinc, selenium, and vitamins A and E, as well as vitamin C and polyphenols, all of which have been discussed before in this article. Hair loss has been linked to oxidative stress. *In vitro* investigations of male AGA patients' dermal papilla cells revealed that oxidative stress may have a role in the balding phenotype and progression of AGA. Furthermore, increased free radical formation was found in the scalps of patients with AA, along with high levels of antioxidant enzymes that we're unable to protect against the ROS, in a study of endogenous antioxidant enzymes and lipid peroxidation in the scalps of patients with AA.

### **Droplet drying**

The most common dietary deficiency in the world, Iron Insufficiency (ID), is a well-known cause of hair loss. What is unknown is the extent to which ID may play a role in hair loss.

While the method by which iron affects hair development is unknown, hair follicle matrix cells are among the fastest dividing cells in the body, and ID's role as a cofactor for ribonucleotide reductase, the rate-limiting enzyme for DNA synthesis, may contribute to hair loss. Furthermore, several genes have been discovered in the human hair follicle, some of which may be influenced by iron. The reversal of ID resulted in the restoration of hair development in a mouse model.