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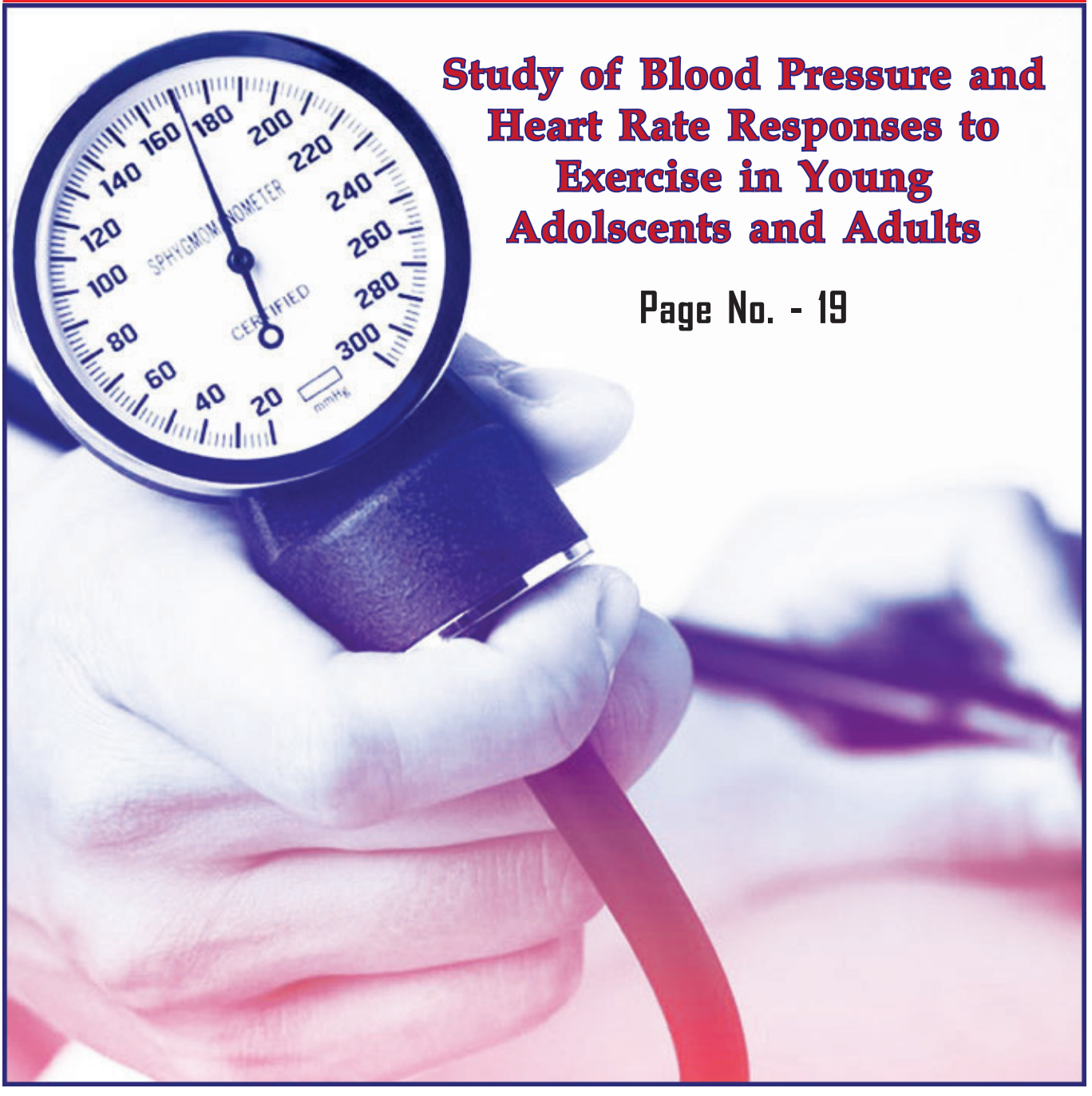

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GENERAL

Biotin (5 mg) and Benefits

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Biotin: Background

Biotin, also known as vitamin H (the H represents Haar and Haut, German words for "hair and skin") or vitamin B7, is a water-soluble vitamin of B-complex. It is composed of an ureido (tetrahydroimidizalone) ring fused with a tetrahydrothiophene ring. A valeric acid substituent is attached to one of the carbo atoms of the tetrahydrothiophene ring. Biotin is a coenzyme for carboxylase enzymes that assist various metabolic reactions involved in the transfer of carbon dioxide. Biotin is important in fatty acid synthesis, branched-chain amino acid catabolism, and gluconeogenesis.

Biotin is often recommended as a dietary supplement for strengthening hair and nails. In addition to being taken as a supplement, many health and beauty products include biotin as an alternative supplement. Ultimately, biotin is found in many cosmetics and health products for the hair and skin although scientific data supporting this outcome are weak. Moreover, it has been demonstrated that proliferation and differentiation of cultured human follicular keratinocytes are not influenced by biotin.

The average dietary biotin intake in Western populations has been estimated to be 35–70 µg/day. For that reason, statutory agencies in many countries do not prescribe a recommended daily intake of biotin. However, a number of metabolic disorders and conditions exist in which an individual's metabolism or uptake of biotin is abnormal. Biotin deficiency can arise due to rare inborn errors of metabolism. These include deficiencies in the enzymes holocarboxylase synthetase (MIM 253270) or biotinidase (MIM 253260).

The frequency of marginal biotin status is not known, but the incidence of low circulating biotin levels in alcoholics has been found to be greater than in the general population. Smoking may further accelerate biotin catabolism in women. Further, relatively low levels of biotin have been reported in patients on parenteral nutrition, who have had a partial gastrectomy or other causes of achlorhydria, patients on antiepileptics, patients on isotretinoin for acne treatment, elderly individuals, and athletes.

Pregnancy and lactation may be associated with an increased demand for biotin. In pregnancy, this may be due to a possible acceleration of biotin catabolism whereas in lactation, the higher demand has yet to be elucidated.

Symptoms of Biotin deficiency

Symptoms of biotin deficiency include hair loss (alopecia), conjunctivitis, dermatitis in the form of a scaly, red rash around the eyes, nose, and mouth (this has been termed as the "biotin-deficient face" by some experts), and neurological symptoms, such as depression, lethargy, hallucination, and numbness and tingling of the extremities. The neurological and psychological symptoms can occur with only mild deficiencies while dermatitis, conjunctivitis, and hair loss generally occur only when deficiency becomes more severe. Individuals with hereditary disorders of biotin deficiency additionally have evidence of impaired immune system function, including increased susceptibility to bacterial and fungal infections.

Biotin's role in skin health isn't well understood. However, it is known that deficiency may lead to red, scaly skin rashes.

Some studies also suggest that biotin deficiency may sometimes cause a skin disorder called seborrheic dermatitis, also known as cradle cap.[1-3]

Biotin: Skin and Hair

Biotin's role in skin health may be related to its effect on fat metabolism, which is important for the skin and may be impaired when biotin is lacking. Biotin is a water-soluble vitamin that acts as an essential cofactor for four carboxylases, each of which catalyzes an essential step in intermediary metabolism. For example, acetyl-CoA carboxylase catalyzes the rate-limiting step in fatty acid elongation. In infants, children, and adults, deficiency of biotin causes alopecia and a characteristic scaly, erythematous dermatitis distributed around body orifices. The rash closely resembles that of zinc deficiency. Candida albicans often can be cultured from the skin lesions. Biotinidase deficiency, an inborn error, causes biotin deficiency, probably as a consequence of unpaired intestinal absorption, cellular salvage, and

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renal reclamation of biotin; biotinidase deficiency causes dermatologic manifestations similar to biotin deficiency. There is evidence that impaired fatty acid metabolism secondary to reduced activities of the biotin-dependent carboxylases (especially acetyl-CoA carboxylase) plays an etiologic role in the dermatologic manifestations of biotin deficiency. Candida infections secondary to impaired immune function might also contribute to the dermatitis of biotin deficiency.

Due to its availability, affordability, and effective marketing for this purpose, biotin is a popular nutritional supplement for treatment of brittle nails and hair loss. Moreover (in contrast to other nutritional supplements, such as vitamin A, selenium, iron, and zinc), there is no known toxicity of biotin in an order of magnitude greater than of the nutritional requirements. There are no reported cases of adverse effects from receiving high doses of the vitamin, in particular, when used for treatment of the inborn errors of biotin metabolism in infants (10–30 mg daily).

While response of splitting brittle nails (onychoschizia, onychoschisis) to daily biotin supplementation, irrespective of serum biotin levels, has been demonstrated, there are little data on the frequency of biotin deficiency in the general population and in patients complaining of hair loss and on the value of oral biotin for treatment of hair loss that is not due to an inborn error of biotin metabolism or acquired biotin deficiency. Recognized risk factors for biotin deficiency from patients' personal history are gastrointestinal disease (inflammatory bowel disease) or intake of drugs interfering with biotin metabolism or uptake (antiepileptics, antibiotics, or isotretinoin). Cutaneous manifestations of biotin deficiency are hair loss and seborrheic-like dermatitis.[4-6]

Of the women complaining of hair loss assessed for serum biotin levels, 38% had values consistent with biotin deficiency (<100 ng/L) and 13% optimal (>400 ng/L). In the women in whom trichograms were performed, both groups (biotin deficiency and optimal levels) demonstrated diffuse telogen effluvium in 24%. Therefore, the trichogram is not helpful in detecting hair loss relating to biotin deficiency since it is neither sensitive nor specific, with telogen effluvium in women, usually being of multifactorial origin. Rather, associated seborrheic-like dermatitis found in 35% of women with serum biotin levels <100 ng/L and telogen effluvium versus 0% of women with optimal biotin levels and telogen effluvium in trichograms, may point to significance of biotin deficiency. Finally, the patient's history may be helpful in detecting biotin deficiency, though with low sensitivity, since in the biotin deficiency group 11% (eight inflammatory bowel disease, six isotretinoin treatment, six antibiotic treatments, and one antiepileptic treatment) versus 1.5% in the group with optimal biotin levels (one on antibiotic treatment) presented with a history of risk factors for biotin deficiency.[7-10]

Conclusion

The custom of treating women complaining of hair loss in an indiscriminate manner with oral biotin supplementation is to be rejected unless biotin deficiency and its significance for the complaint of hair loss in an individual has been demonstrated. It must be kept in mind that hair loss in women may be of multifactorial origin, including female androgenetic alopecia, other nutritional deficiencies (e.g., iron deficiency), and/or endocrine disorders (e.g., thyroid disorder). Treating the patient exclusively with oral biotin poses the risk of neglect or delay of appropriate treatment of hair loss in the particular case. Rather, a careful patient history and clinical examination with respect to risk factors for biotin deficiency (gastrointestinal disease, medication with isotretinoin, antibiotics, or antiepileptics) and associated symptoms (seborrheic-like dermatitis, neurological symptoms) must be performed. When biotin deficiency is suspected, the serum biotin level must be determined, and in case of biotin deficiency (<100 ng/L), the cause must be sought (unless obvious from the patient history) and treated. Regardless of the cause, the deficiency can usually be successfully addressed directly with nutritional supplementation with usually high bioavailability of oral biotin supplements usually in a dosage of 5 mg/day. At the same time, potential additional causes of hair loss, for example, androgenetic alopecia, other nutritional deficiencies, and endocrine disorders, must systematically be addressed and treated as needed. The high percentage of marginal biotin levels in this study may reflect that a number of other factors may contribute, such as anorexia/bulimia, alcoholism, smoking, pregnancy, lactation, athleticism, and old age. Finally, a wide variability in biotin bioavailability from foodstuff may be due to the ability of an organism to break various biotin-protein bonds from food.

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Hypothyroidism in diabetes

- The pathophysiological basis of association between thyroid dysfunction and diabetes depends on a complex interaction of common signaling pathways in type 2 diabetes and on a linked genetic susceptibility in type 1 diabetes and autoimmune thyroid disease.
- The pathophysiological mechanisms for the linked regulation is demonstrated by the regulation of 5' adenosine monophosphate activated protein kinase (AMPK), a central target not only for the modulation of insulin sensitivity but also for the feedback of thyroid hormones on appetite and energy expenditure.

The increased hepatic glucose output is a major factor in the induction of hyperinsulinemia, glucose intolerance and the development of peripheral insulin resistance. This along with glycogenolysis in thyrotoxicosis is a major factor for decrease in glucose tolerance. Despite increased insulin clearance, hyperinsulinemia is induced and peripheral insulin resistance develops. The clinical implication of this is hyperthyroidism which precipitates subclinical diabetes and worsens glycemic control in pre-existing type 2 diabetes.

- Insulin sensitivity, or drugs used to modulate it may affect thyroid growth and function. Hence, better definition of the interactions between diabetes and thyroid hormones can optimize treatment of diabetics.
- Insulin requirement in hypothyroidism is decreased due to impaired renal insulin clearance. This along with loss of appetite due to hypothyroidism may contribute to a decrease in insulin in underactive thyroid disease. Type 1 diabetic patients having hypothyroidism have a higher risk of hypoglycemic episode.

- Thyroid Life

According to a systematic review and metanalysis of trials of PCSK9 inhibitors. Alirocumab (Sano-fi-Regeneron) and evolocumab (Amgen), both fully human monoclonal antibodies are cholesterol-lowering agents.

Cardiology today

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