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Biotin

Technical Background

- Biotin (also known as vitamin B7) is a sulfur-containing member of the B vitamin complex. It is obtained both by synthesis by intestinal microorganisms and from the diet (though the proportions derived from each is unknown).
- Biotin is an essential cofactor in four human enzyme systems (all carboxylases) that catalyze critical steps in carbohydrate, amino acid, and lipid metabolism within our cells. As such, it is fundamental to cellular function and, ultimately, to human health. Secondary roles in cell growth, glucose homeostasis, and DNA synthesis have been reported.1
- Dietary biotin is often bound to proteins like avidin, a glycoprotein found in egg whites, and must be released by an enzyme (biotinidase) to create free biotin prior to uptake.
- While clinical biotin deficiencies are relatively infrequent, high and prolonged consumption of raw egg whites, prolonged use of antibiotics, short gut syndrome, and parenteral nutrition without biotin supplementation can lead to deficits.1 Symptoms of biotin deficiency include skin rashes, hair loss, nervous disorders, depression, and fatigue.2,3
- Biotin supplementation has recently proven effective in the prevention and maintenance of type 2 diabetes.4 Supplementation is also used to strengthen and re-grow hair, skin, and nails.

Sources and Recommended Intake

- Biotin is widely distributed in natural foodstuffs, but absolute levels are low when compared to other B vitamins. Brewer’s yeast, egg yolk, liver, soybeans, cauliflower, and unpolished whole grains are the best dietary sources of biotin. Biotin is also found in many hair and skin conditioning products.5
- The average American eats 35-60 mcg of biotin per day. The Food and Nutrition Board advises consuming at least 30 mcg/day to avoid symptoms of deficiency.6
- There have been no reports of toxic reactions, even in people taking doses up to 200 mg orally and 20 mg intravenously.1

Abstracts


Glucokinase (GK), expressed in hepatocyte and pancreatic beta cells, has a central regulatory role in glucose metabolism. Efficient GK activity is required for normal glucose-stimulated insulin secretion, postprandial hepatic glucose uptake, and the appropriate suppression of hepatic glucose output and gluconeogenesis by elevated plasma glucose. Hepatic GK activity is subnormal in diabetes, and GK may also be decreased in the beta cells of type II diabetics. In supraphysiological concentrations, biotin promotes the transcription and translation of the GK gene in hepatocytes; this effect appears to be mediated by activation of soluble guanylate cyclase. More recent evidence...
indicates that biotin likewise increases GK activity in islet cells. On the other hand, high-dose biotin suppresses hepatocyte transcription of phosphoenolpyruvate carboxykinase, the rate-limiting enzyme for gluconeogenesis. Administration of high-dose biotin has improved glycemic control in several diabetic animals models, and a recent Japanese clinical study concludes that biotin (3 mg t.i.d. orally) can substantially lower fasting glucose in type II diabetics, without side-effects. The recently demonstrated utility of chromium picolinate in type II diabetes appears to reflect improved peripheral insulin sensitivity—a parameter which is unlikely to be directly influenced by biotin. Thus, the joint administration of supra-nutritional doses of biotin and chromium picolinate is likely to combat insulin resistance, improve beta-cell function, enhance postprandial glucose uptake by both liver and skeletal muscle, and inhibit excessive hepatic glucose production. Conceivably, this safe, convenient, nutritional regimen will constitute a definitive therapy for many type II diabetics, and may likewise be useful in the prevention and management of gestational diabetes. Biotin should also aid glycemic control in type I patients.

References