Orentreich Foundation for the Advancement of Science, Inc.

VitaLongevity™

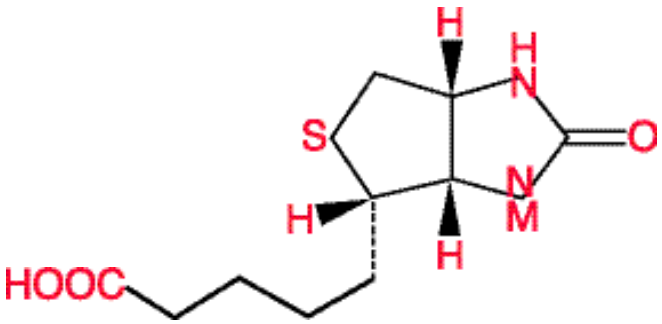
Logo: Life's blood flows through the hourglass; the stopcock represents the alteration of aging and disease as biomedical research progresses.

June 2004

BIOTIN

HISTORY

In 1916, W. G. Bateman discovered that adding raw egg white to an otherwise adequate diet produced toxicity and disease. Called egg-white injury, the symptoms included effects on the skin (scaly dermatitis and hair loss), the nervous system (depression, lassitude, and muscle pain), the gastrointestinal tract (anorexia and nausea), and even the cardiovascular system (abnormal heart action, i.e., changes in the electrocardiogram). Later research identified the culprit to be avidin, a protein in egg white which avidly binds biotin, preventing its absorption. Thus, the biological necessity of biotin became evident, and by 1935 scientists had isolated it in pure form. Biotin, originally called vitamin H after the German word *Haut* meaning "skin", belongs to the water soluble B vitamin class.



Biotin

ACTIVITY

Biotin plays a supporting role as a co-factor in a number of key reactions that help convert food to energy: glucose production, fatty acid metabolism, and amino acid catabolism (breakdown). It helps carry out carboxylation reactions, important steps in metabolism, giving biotin an essential role in maintaining metabolic homeostasis. So, it is not surprising that some reports suggest biotin supplementation might beneficially increase insulin sensitivity. However, not all biotin-responsive disorders can be explained based on the classical role of this vitamin in cell metabolism, suggesting that underlying mechanisms remain to be discovered.

REQUIREMENT

Food sources rich in biotin include yeast, liver, organ meats, egg yolks, grains, nuts, and fish. Diets providing a daily intake of 0.15 to 0.3 mg of biotin are considered adequate. Even absent a well-balanced diet, out-right biotin deficiencies rarely occur because gut microbes synthesize biotin. Subtle deficits can result from the use of broad spectrum antibiotics, which kill off these helpful gastrointestinal bacteria, as well as from diseases such as Crohn's, ulcerative colitis, or other ailments that interfere with nutrient absorption. Some individuals lack the enzyme biotinidase, which releases biotin from food proteins. A mild degree of biotin deficiency is not uncommon in normal pregnancy. Large doses of two other nutrients, pantothenic acid and lipoic acid, potentially could interfere with biotin uptake and distribution to cells. Surprisingly, urinary biotin excretion has proved to be a more reliable measure of biotin status than monitoring blood levels.

Unfortunately, biotin remains one of the most poorly understood vitamins in terms of nutritional requirement and responsiveness to physiological and pharmacological states. Because of this, sub-optimum biotin status may be more prevalent than is currently appreciated. For instance, a biotin deficit may be an overlooked cause of infantile seborrheic dermatitis or even of sudden infant death syndrome (SIDS). A biotin deficiency might only become apparent under conditions of stress; for example, liver cells deprived of biotin might not have the energy needed to detoxify chemicals and drugs. Other possible biotin-deficiency conditions include reduced post-natal growth, epileptic disorders, decreased testosterone production, and impaired immune function resulting in, among others, *Candida* skin infections.

SUPPLEMENTATION FOR BRITTLE AND SPLIT NAILS

This is not to suggest that supplementation with large, even pharmacological, doses of biotin might not be of benefit to individuals with adequate nutritional intake and healthy functioning GI tracts. In fact, our interest in biotin stems from years of clinical experience prescribing biotin for weak and brittle nails. This concept originated in

England decades ago when veterinarians began treating horses with biotin for split-hooves syndrome; British dermatologists picked up on the idea and started treating patients with brittle nails. Doses of 7.5 mg 2 to 3 times daily produce marked improvements in nail quality and hardness in most patients after 6 months of treatment; a 25% increase in nail plate thickness can also occur. Contrary to expectation, biotin, either taken orally or applied topically, shows no benefit in common hair loss conditions, such as male pattern baldness, although formal studies have not been conducted.

BIOTIN AND THE METABOLIC SYNDROME

In December 2002, OFAS discussed the Metabolic Syndrome, a condition resulting from insulin resistance (the inability of insulin to cause glucose uptake by cells and to stop the liver from producing glucose), and the importance of the insulin glucose tolerance stress test (IGTT) for its evaluation. Features of the syndrome include abdominal obesity, high blood pressure, elevated triglycerides, low HDL-cholesterol, and high blood levels of insulin. Metabolic Syndrome frequently leads to impaired glucose tolerance and, in some individuals, Type 2 diabetes. Therefore, recent reports about biotin's ability to substantially lower fasting glucose levels in Type 2 diabetics, and to do so without side effects, renewed our interest in this vitamin. After one month of treatment with oral doses of 3 mg 3 times per day (9 mg/day), fasting glucose levels plummeted by 45%. Type 1 diabetics receiving 16 mg/day for one week also experienced reduced blood glucose levels. Remarkably, biotin in high doses given to several Type 1 diabetic patients suffering severe peripheral neuropathy resulted in marked clinical improvement within 4 to 8 weeks.

Biotin affects the pancreatic cells that secrete insulin in response to changes in blood glucose levels by increasing the activity of a key enzyme, resulting in more insulin release. But biotin must do more than just increase insulin secretion since it also lowers glucose levels in Type 1 diabetics who typically have little-to-no insulin production. In fact, in diabetic rats, injected biotin improves glucose handling without increasing insulin secretion. Other studies show improvements in insulin sensitivity and lower body weights despite higher daily food intake in rats with impaired glucose tolerance when they are fed a high biotin diet. Exactly how biotin mitigates insulin resistance is under investigation but could involve the regulation of the same enzymes that insulin suppresses when it shuts down glucose production in the liver.

SPECIFIC BASIC DEFICIENCY SYMPTOMS

- Scaly dermatitis
- Inflamed sore tongue
- Loss of appetite
- Nausea
- Depression
- Muscle pain
- Sitophobia (morbid dread of food)
- Pallor
- Anemia
- Abnormalities of heart function
- Burning or prickling sensations; sensitive skin
- Insomnia
- Extreme lassitude
- Increased cholesterol
- Depression of immune system

TO BIOTIN OR NOT TO BIOTIN

Biotin is a very safe B Vitamin. No toxicity has been reported to date at doses of up to 200 mg; in fact, biotin is the only vitamin with no known toxicity. Small amounts are essential; large amounts seem very therapeutic, benefiting skin, nails, and pancreatic function.

No accomplishment of OFAS is possible without your encouragement and generous support of our goal: to prevent, halt, or reverse those disorders that decrease the quality or length of life. To this end, the OFAS VitaLongevity *newsletters are designed to alert you to those strategies that are still valid, those that are no longer valid, and new suggestions for making your life as long and healthy as possible.*

INFORMATION FOR DONORS

The Orentreich Foundation for the Advancement of Science, Inc., was founded in 1961. OFAS is a non-profit institution dedicated to biomedical research to prevent, halt, or reverse those disorders that decrease the quality or length of life. It is duly registered with the US Internal Revenue Service as an Operating Private Foundation under Section 4942(j)(3).

Your tax-deductible contribution should be mailed to:
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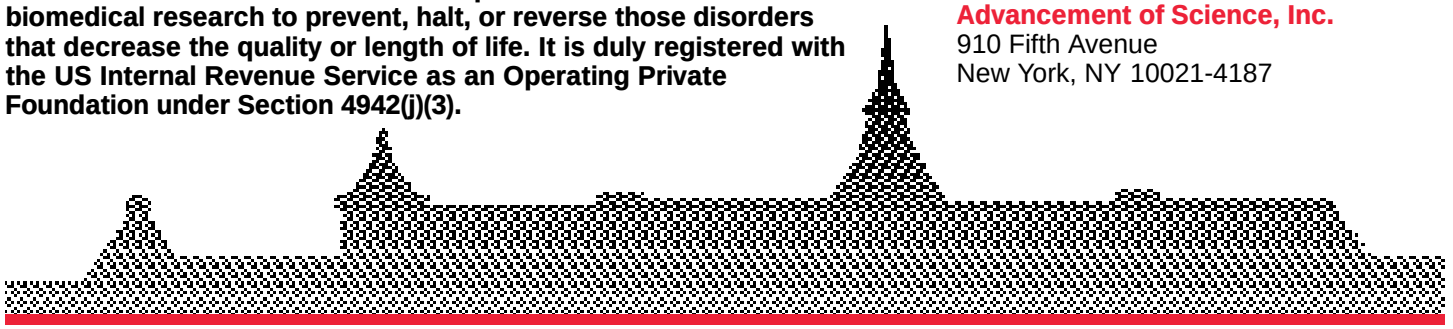

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