A Review of the Use of Biotin for Hair Loss

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Introduction

Biotin (also known as vitamin B7 or vitamin H) is a water-soluble vitamin that serves as an essential cofactor for carboxylase enzymes in multiple metabolic pathways. Due to its relatively low cost and abundance of availability in cosmetic products, biotin has become the new trend for consumers wishing to have longer, healthier hair and nails. Current recommendations for biotin by the Institute of Medicine state that the daily adequate intake (AI) for adults is 30 μg/day\textsuperscript{[1]}.

Most healthy individuals meet these requirements through a well-balanced diet, though many still take up to 500–1,000 μg of biotin supplementation daily. Although no major toxicities of excess biotin have been reported, data on the actual benefit of biotin's effect on hair and nail growth is limited. Moreover, outside the setting of pregnancy, malnutrition, medication effects, and biotinidase deficiency in children, reports of low biotin levels have rarely been cited. Therefore, we propose that true biotin deficiency is uncommon and that there is lack of sufficient evidence for supplementation in healthy individuals.

Keywords

Biotin · Hair · Nails · Vitamin · Supplement

Abstract

**Background:** Biotin has gained commercial popularity for its claimed benefits on healthy hair and nail growth. Despite its reputation, there is limited research to support the utility of biotin in healthy individuals. **Objective:** To systematically review the literature on biotin efficacy in hair and nail growth. **Methods:** We conducted a PubMed search of all case reports and randomized clinical trials (RCTs) using the following terms: (biotin and hair); (biotin and supplementation and hair); (biotin supplementation); (biotin and alopecia); (biotin and nails); (biotin and dermatology), and (biotin recommendations). **Results:** We found 18 reported cases of biotin use for hair and nail changes. In all cases, patients receiving biotin supplementation had an underlying pathology for poor hair or nail growth. All cases showed evidence of clinical improvement after receiving biotin. **Conclusions:** Though its use as a hair and nail growth supplement is prevalent, research demonstrating the efficacy of biotin is limited. In cases of acquired and inherited causes of biotin deficiency as well as pathologies, such as brittle nail syndrome or uncombable hair, biotin supplementation may be of benefit. However, we propose these cases are uncommon and that there is lack of sufficient evidence for supplementation in healthy individuals.
Materials and Methods

We conducted a PubMed search of all case reports and randomized clinical trials (RCTs) published using the following terms: (biotin and hair); (biotin and supplementation and hair); (biotin supplementation); (biotin and alopecia); (biotin and nails); (biotin and dermatology), and (biotin recommendations). We identified additional sources through references contained in the original articles. We limited our search to studies discussing human subjects only. Through this search, we found 18 reported cases of biotin use for hair and nail changes (Table 1).

Table 1. Reported cases of patients categorized by age, dose of biotin, symptoms, and length of treatment until clinical improvement

<table>
<thead>
<tr>
<th>Study</th>
<th>Age</th>
<th>Reason for biotin deficiency identified</th>
<th>Alopecia reported</th>
<th>Nail changes reported</th>
<th>Dose of biotin</th>
<th>Reported time and degree of hair improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dakshinamurti and Triggs-Raine, 1997 [2]</td>
<td>newborn</td>
<td>yes, inherited enzyme deficiency</td>
<td>no</td>
<td>no</td>
<td>2,500 μg/day</td>
<td>clinical improvement by 6 months of age</td>
</tr>
<tr>
<td>Rajendiran and Sampath, 2011 [3]</td>
<td>2 months</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/ twice a day</td>
<td>total resolution in 8 months</td>
</tr>
<tr>
<td>Fujimoto et al., 2005 [4]</td>
<td>5 months</td>
<td>yes, dietary/only on amino acid formula for dyspepsia</td>
<td>yes</td>
<td>no</td>
<td>1,000 μg/day</td>
<td>hair regrowth in 2 months</td>
</tr>
<tr>
<td>Colamaria et al., 1989 [5]</td>
<td>4 months</td>
<td>yes, inherited enzyme deficiency</td>
<td>not true alopecia, but sparse scalp hair was reported</td>
<td>no</td>
<td>5,000 μg/ twice a day</td>
<td>clinical improvement of neurological symptoms seen after 10 days of starting biotin</td>
</tr>
<tr>
<td>Coulter et al., 1982 [6]</td>
<td>11 months</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/day</td>
<td>not specified</td>
</tr>
<tr>
<td>Coulter et al., 1982 [6]</td>
<td>14 months</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/ twice a day</td>
<td>not specified</td>
</tr>
<tr>
<td>Boccaletti et al., 2007 [7]</td>
<td>1 year</td>
<td>no</td>
<td>no (uncombable hair syndrome)</td>
<td>yes, onychoschizia of nail plates of hands and feet</td>
<td>5,000 μg/day</td>
<td>excellent results in 3 months</td>
</tr>
<tr>
<td>Shelley and Shelley, 1985 [8]</td>
<td>18 months</td>
<td>no</td>
<td>no (uncombable hair syndrome)</td>
<td>no</td>
<td>300 μg/3 times daily</td>
<td>significant improvement in 4 months</td>
</tr>
<tr>
<td>Boccaletti et al., 2007 [7]</td>
<td>2 years</td>
<td>no</td>
<td>no (uncombable hair syndrome)</td>
<td>yes, onychoschizia of nail plates of hands and feet</td>
<td>5,000 μg/day</td>
<td>excellent results in 3 months</td>
</tr>
<tr>
<td>Mukhopadhyay et al., 2014 [9]</td>
<td>3 years</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>30,000 μg/day</td>
<td>6-week follow-up showed dramatic improvement in scalp</td>
</tr>
<tr>
<td>Komur et al., 2011 [10]</td>
<td>3 years</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/ twice a day</td>
<td>marked improvement in 1 month, complete hair growth in 6 months</td>
</tr>
<tr>
<td>Gannavarapu et al., 2015 [11]</td>
<td>5 years</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/day</td>
<td>n/a</td>
</tr>
<tr>
<td>Rahman et al., 1997 [12]</td>
<td>5 years</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/day</td>
<td>marked clinical improvement of neurological symptoms was observed at 5 months</td>
</tr>
<tr>
<td>Roth et al., 1980 [13]</td>
<td>5 years</td>
<td>yes, inherited enzyme deficiency</td>
<td>yes</td>
<td>no</td>
<td>20,000 μg/day</td>
<td>n/a</td>
</tr>
<tr>
<td>Castro-Gago et al., 2011 [14]</td>
<td>n/a</td>
<td>valproic acid, but no significant decreases in biotin and biotinidase levels seen</td>
<td>yes</td>
<td>no</td>
<td>10,000 μg/day</td>
<td>3 months</td>
</tr>
<tr>
<td>Hochman et al., 1993 [15]</td>
<td>n/a</td>
<td>yes, brittle nail syndrome</td>
<td>no</td>
<td>yes</td>
<td>2,500 μg/day</td>
<td>6 months</td>
</tr>
<tr>
<td>Colombo et al., 1990 [16]</td>
<td>n/a</td>
<td>yes, brittle nail syndrome</td>
<td>no</td>
<td>yes</td>
<td>3,000 μg/day</td>
<td>2 months</td>
</tr>
<tr>
<td>Floersheim, 1989 [17]</td>
<td>n/a</td>
<td>yes, brittle nail syndrome</td>
<td>no</td>
<td>yes</td>
<td>2,500 μg/day</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Use of Biotin for Hair Loss

Of the reported cases in the literature, all patients receiving biotin supplementation had some underlying pathology for either poor hair or nail growth. Moreover, all cases showed evidence of clinical improvement after receiving biotin. Time to improvement as well as dosage administered varied for each case. Ten of the 18 cases were reports of patients with inherited enzyme deficiency in either biotinidase or holocarboxylase synthetase. Of these 10, 8 cases reported alopecia that subsequently resolved after varying months of biotin supplementation. Additionally, there were 3 reported cases of uncombable hair syndrome that all showed improvement in hair quality after a few months of treatment. Fujimoto et al. [4] reported a case of biotin deficiency secondary to diet...
in an infant who was consuming a special amino acid formula. This patient had low serum and urine levels of biotin as well as perioral dermatitis and alopecia. Hair regrowth in this patient occurred after 2 months of biotin supplementation. Only 1 study conducted by Castro-Gago et al. [14] showed decreased levels of both biotin and biotinidase secondary to medication usage (valproic acid) that improved after 3 months of supplementation with biotin. Finally, 3 cases of brittle nail syndrome treated with biotin were found in the literature and each case showed improvement of nail strength as well as growth on either 2,500 or 3,000 μg of biotin/day.

**Discussion**

Biotin is a required cofactor for carboxylase enzymes that become activated once they are joined together by holocarboxylase synthase [18]. These enzyme complexes play an important role in multiple metabolic processes including gluconeogenesis, fatty acid synthesis, and amino acid catabolism [19]. Biotin’s function in protein synthesis and more specifically, in keratin production, explains its contribution to healthy nail and hair growth. Biotin is readily found in many foods and is also produced by normal gut flora. Foods found to have high amounts of biotin include nuts, legumes, whole grains, unpolished rice, and egg yolk [20]. Recommended daily allowances of biotin have not been established due to a lack of sufficient evidence [21]. However, AI levels have been recommended by the Food and Nutrition Board of the Institute of Medicine (Table 2). It has been estimated that in Western populations, typical dietary intake of biotin is between 35 and 70 μg/day [22]. Though several animal models [23–25] demonstrating the effects of induced biotin deficiency can be found in the literature, there are currently no studies that show biotin deficiencies in healthy human individuals with balanced diets.

Biotin deficiency can be either acquired or congenital. Though an acquired biotin deficiency is possible, it is still rare. A commonly documented cause of acquired biotin deficiency is secondary to increased raw egg consumption. The protein avidin, found in raw egg whites, can be denatured through cooking, but when uncooked, this protein binds to biotin tightly preventing it from being used as an essential cofactor [26]. Patients taking anticonvulsant medications, such as valproic acid, can also become deficient, and therefore, are prophylactically administered biotin [21]. Additional causes of acquired biotin deficiency include states of alcoholism or pregnancy, other medications, such as isotretinoin [27], impaired intestinal absorption, or prolonged use of antibiotics interrupting normal gut flora [18, 23, 24]. Congenital or genetic biotin deficiency is due to an autosomal recessive trait leading to a lack of either holocarboxylase synthase or biotinidase. When it occurs within the first 6 weeks of life, this deficiency is defined as the neonatal type. In this type of biotin deficiency, the enzyme holocarboxylase synthetase is absent and patients typically have severe, life-threatening conditions [18, 28, 29]. Beyond 3 months of life, the infantile form predominates and is defined by a biotinidase deficiency which is involved in the absorption of free biotin following carboxylase degradation [18, 28, 30]. Whether acquired or congenital, typical symptoms of biotin deficiency include alopecia, eczematous skin rashes, seborrheic dermatitis, conjunctivitis, and multiple neurological symptoms, such as depression, lethargy, hypotonia, and seizures [3, 20]. While the neurological symptoms occur at more severe levels of biotin deficiency, the dermatological manifestations often appear first and are therefore an important indicator [31]. The normal biotin plasma concentration ranges from 400 to 1,200 ng/L [22]. Deficiency is technically considered to be a level of less than 200 ng/L. However, plasma biotin levels can fluctuate daily and thus are not considered to be a sensitive marker [22]. A more validated measure of biotin deficiency is an increased urinary excretion of the metabolite, 3-hydroxyisovaleric acid (normal level: 195 μmol/24 h) [22].

In our search, we found 18 reports in the literature that showed improvement of hair and nail growth on supplementation in patients with established biotin deficiency. For patients with inherited enzyme deficiency, larger doses of biotin supplementation are recommended (from 10,000 to 30,000 μg/day). Those with brittle nail syndrome and other underlying hair pathologies, such as uncombable hair syndrome, require much lower doses of biotin supplementation ranging from 300 to 3,000 μg/
day. Despite these data, there have been no randomized, controlled trials to prove the efficacy of supplementation with biotin in normal, healthy individuals. Moreover, only 1 case in the literature has measured the levels of biotin in normal individuals that had complaints of hair loss. In this study of 541 women (age range between 9 and 92 years), 38% had low biotin levels [20]. However, of those women, 11% were later found through patient history (use of antibiotics, antiepileptics, isotretinoin, or GI disease) to have a reason for the underlying deficiency and 35% had co-existing seborrheic dermatitis, suggesting a multifactorial cause for hair loss. Additionally, in vitro studies have shown that proliferation and differentiation of normal, nonpathologic follicular keratinocytes are not influenced by biotin [27].

Conclusions

Despite its popularity in the media and amongst consumers, biotin has no proven efficacy in hair and nail growth of healthy individuals. Only 1 study has shown decreased levels of biotin in healthy individuals, though this data was confounded by multiple factors, including patient history. Therefore, in the absence of additional studies, we have found no evidence to suggest benefit from biotin supplementation outside of known deficiencies secondary to congenital or acquired causes.

Disclosure Statement

The authors declare no conflicts of interest pertaining to the current publications.

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