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IMPACT OF LIFESTYLE FACTORS, NUTRITIONAL CHOICES AND SUPPLEMENTS ON ALOPECIA AREATA: A LITERATURE REVIEW

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ABSTRACT

Alopecia areata (AA) is a chronic, immune-mediated condition characterized by non-scarring hair loss which can significantly impact patients' quality of life. While its precise etiology remains elusive, emerging evidence suggests that various lifestyle factors may contribute to disease onset, progression, and severity. This review aims to synthesize current findings on the potential impact of modifiable lifestyle elements including smoking, alcohol consumption, psychological stress, sleep disturbances, physical activity, obesity, and diet on the pathogenesis and clinical course of AA. The role of specific micronutrients such as iron, vitamin D, zinc, B vitamins, and biotin is also examined. Although several studies suggest associations between these factors and AA, the evidence remains inconsistent and largely observational. At present, no definitive lifestyle modification guidelines can be proposed for AA patients. However, this review underscores the need for increased clinical awareness and further high-quality research to elucidate the mechanisms by which lifestyle and dietary habits may influence autoimmune processes involved in AA, with the ultimate goal of integrating holistic approaches into patient care.

KEYWORDS

Alopecia Areata, Lifestyle, Diet, Supplements

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Introduction

Alopecia areata (AA) is an autoimmune disorder characterized by non-scarring hair loss resulting from immune-mediated damage to hair follicles. Clinically, it presents as a sudden onset of well-demarcated, smooth alopecic patches, most frequently on the scalp. In severe cases, it may progress to alopecia totalis or alopecia universalis [1,2]. The precise etiology remains unclear. However, current evidence suggests a multifactorial pathogenesis involving genetic predisposition, immune system dysregulation, and environmental triggers. AA is the second most common non-scarring alopecia. The disease affects approximately 2% of the general population over the course of a lifetime [3]. The majority of studies indicate no significant sex- or ethnicity-based differences in age of onset, disease duration, or clinical subtype. While AA can manifest at any age, it most commonly presents before the age of 40. Early-onset cases, typically with a mean age of onset between 5 and 10 years, are often associated with more severe forms of the disease, such as alopecia universalis [4]. The management of AA remains challenging, as numerous therapeutic approaches exist, yet high-quality evidence from randomized controlled trials supporting their efficacy is limited. Treatments such as corticosteroids, minoxidil, and light-based therapies may cause side effects like irritation or unwanted hair growth in other hair-bearing regions of the body. Moreover, there is no guarantee that regrown hair will be sustained after treatment is discontinued [5]. Newer approaches include JAK inhibitors, biologics, small molecules, antihistamines, and PRP injections [6]. AA can significantly reduce the quality of patients' life, with implications for psychological well-being and social functioning. Patients exhibit a significantly increased risk of developing depression and anxiety [7]. Psychological and environmental factors, such as smoking, alcohol consumption, sleep disturbances, obesity and inappropriate diet can trigger AA [8]. The following paper aims to provide a literature review regarding the importance of lifestyle modifications in AA.

Nicotinism

A meta-analysis by Khanimov identified a link between ever smoking and the occurrence of AA. A similar association was observed in a subgroup analysis focusing on current smokers. However, this finding was based on data from only two studies [9]. A study on the relationship between smoking and AA found that current smokers are at a higher risk of developing AA compared to those who have never smoked [10]. Moreover, the researchers observed a trend suggesting that prolonged smoking duration and higher cumulative pack-years further increase the risk of developing AA [10]. Smoking-induced free radicals and pro-inflammatory cytokines may play a role in the disruption of hair follicle integrity and the collapse of immune privilege [11]. Moreover, secondhand smoke exposure has been associated with the onset of inflammatory skin conditions [12]. As highlighted in the studies mentioned above, efforts should be made to encourage smoking cessation among AA patients, while also informing them about available support strategies to aid the process. Patients should additionally be advised to avoid exposure to secondhand smoke.

Alcohol

Curtis et al. demonstrated that patients with AA exhibited significantly higher odds of alcohol consumption than controls, potentially reflecting underlying psychiatric comorbidities and the psychological burden of the disease [13]. Alcohol may play a role in the pathogenesis of AA by promoting the production of pro-inflammatory cytokines (e.g., IL-6, TGF-α, IFN-γ), enhancing Th17-mediated immune responses, and modulating stress-related hormonal pathways involving ACTH and cortisol [11]. However, both social and regular alcohol consumers demonstrated a significantly reduced risk of developing AA compared to individuals who never drank [10]. Further research is needed to determine the long-term effects of alcohol consumption and its potential role in the pathogenesis of AA.

Psychological stress

Acute stress has been shown to induce immune system hyperactivation within both the central and peripheral nervous systems, potentially facilitating the initiation of autoimmune pathologies. An estimated 23% of individuals with AA report experiencing a major emotional crisis or traumatic event prior to the onset or worsening of the disease [14]. Psychological stress activates the hypothalamic-pituitary-adrenal axis, inducing the release of neuropeptides such as corticotropin-releasing hormone and substance P, which disrupt hair follicle immune privilege and thereby increase susceptibility to autoimmune attack [15]. Patients with AA demonstrated greater tendencies toward harm avoidance and reward dependence than healthy controls. Recurrent episodes of AA were significantly linked to elevated psychiatric symptomatology, with notably large effect sizes observed for obsessive-compulsive behavior and paranoid thinking [16]. Patients with AA reported a higher frequency and greater impact of stressful life events compared with their healthy siblings, suggesting that psychosocial stress may play a contributory role in the onset or exacerbation of the disease [17].

Sleep disorders

Studies have shown that patients with AA have an increased risk of sleep disturbances, including obstructive sleep apnea and non-apneic insomnia, both of which have been independently associated with greater susceptibility to AA [18]. A large retrospective cohort study demonstrated that sleep disorders, particularly in individuals under 45 years, were associated with an increased risk of AA and a higher prevalence of comorbidities such as autoimmune thyroiditis, vitiligo, rheumatoid arthritis, and solid organ neoplasms. [19]. Another study involving 105 AA patients assessed excessive daytime sleepiness (EDS) using the Epworth Sleepiness Scale (ESS), finding EDS in only 11.4% of cases and average ESS scores within normal limits, suggesting no clear association between AA and sleep quality [20]. Although current evidence linking AA and sleep disturbances remains limited, research on other autoimmune diseases indicates a potential relationship with sleep disorders.

Sport

Data regarding the impact of physical activity on the progression of AA remains limited. A cross-sectional study of 83 AA patients found that most did not meet physical activity guidelines, and those with greater hair loss and low activity levels reported significantly higher depression, anxiety, and stress, underscoring the need for interventions promoting physical activity in this population [21]. Jiang et al. reported that engaging in aerobic exercise or exercising for more than 60 minutes per session can slow the progression of androgenic alopecia and alleviate its symptoms [22].

Obesity

The prevalence of obesity is higher among individuals with AA compared to the general population (17.2% vs. 13.3%). Moreover, the prevalence of prediabetes increases significantly with age in AA patients, particularly among those aged 40 years and older [23]. Clinical and experimental evidence suggests that adipokines contribute to autoimmune pathogenesis, supporting the role of obesity as an environmental risk factor in the development and progression of AA [24,25]. Obesity has been shown to modulate immune responses relevant to AA by promoting Th1-, Th2-, and Th17-driven inflammation. Increased BMI is associated with enhanced IL-17-mediated skin inflammation, upregulated TSLP production and Th2 activity, and impaired skin barrier and lymphatic function, all of which contribute to sustained inflammatory responses and may play a role in the immunopathogenesis of AA [11, 26-27].

Diet

Despite the availability of FDA-approved treatments for severe AA, many patients adopt dietary interventions based on online information. In a national survey, nearly half sought dietary advice, most commonly using supplements or specific diets, though about half reported no clinical or psychosocial improvement. These findings highlight the need for clinician awareness of non-medical influences on patient behavior [28]. A gluten-free diet has been found to promote hair regrowth in AA patients with coexisting celiac disease, whereas a lactose-free diet showed no such effect [29]. Adequate protein intake is essential for maintaining healthy hair. Diets such as those based on human chorionic gonadotropin, hypocaloric restrictions, and increased consumption of fish, buckwheat, and millet groats have been identified as potential triggers for various types of alopecia [29]. Although evidence is limited, omega-3 fatty acids may exert beneficial effects in AA by modulating Th1-, Th2-, and Th17-related immune responses. DHA and EPA have been shown to reduce skin inflammation and cytokine production, suggesting a potential immunomodulatory role in AA pathogenesis [11, 30-31].

Adherence to anti-inflammatory diets such as the Mediterranean diet may protect against hair loss by reducing oxidative stress through bioactive compounds like carotenoids and polyphenols, which help maintain hair follicle function and promote growth [32]. Modifying the diet can also influence the gut microbiota composition, which regulates immune responses through metabolites such as short-chain fatty acids that enhance Treg activity. Observations of hair regrowth after fecal microbiota transplantation in AA patients further suggest that restoring microbial balance may contribute to disease modulation [33]. Research on the therapeutic potential of individual micronutrients in AA has primarily been limited to small-scale studies. The heterogeneity of dietary patterns among participants complicates the ability to accurately assess the isolated effects of specific micronutrients. Consequently, there is insufficient evidence to support routine micronutrient supplementation in the clinical management of AA at this time.

Iron

Several studies have explored the link between iron deficiency and hair loss, primarily focusing on women and noncicatricial forms of hair loss. Some findings suggest a potential association with conditions like alopecia areata, androgenetic alopecia, telogen effluvium, and diffuse hair loss [34,35]. Currently, there is insufficient evidence to support routine screening for iron deficiency or to recommend iron supplementation in patients with hair loss and iron deficiency, unless anemia is present [34,35].

Vitamin D

Patients with AA demonstrate significantly lower serum 25-hydroxyvitamin D [25(OH)D] levels compared to healthy controls, along with a higher prevalence of vitamin D deficiency. An inverse correlation has been observed between vitamin D status and disease severity in AA [36]. Although vitamin D receptor (VDR) gene polymorphisms do not differ between AA patients and controls, reduced VDR expression in hair follicles is linked to increased local inflammation, suggesting an immunomodulatory role of vitamin D signaling in AA pathogenesis [37,38].

Serum zinc

Multiple studies have reported significantly lower serum zinc levels in individuals with AA compared to healthy controls, with levels inversely correlated to disease severity [39]. For instance, one study found mean plasma zinc levels of 74.2 μg/100 mL in AA patients versus 95.5 μg/100 mL in controls [37]. Although some studies report no overall difference in zinc levels between groups, subgroup analyses often reveal lower zinc concentrations in patients with more severe disease [37]. These findings suggest a potential association between zinc deficiency and AA severity, warranting further investigation into its role in disease pathophysiology.

B vitamins

Several studies have reported links between AA and reduced red blood cell folate levels, as well as MTHFR gene polymorphisms, with some case reports describing co-occurrence of AA and pernicious anemia [40,41]. These findings indicate that folate or vitamin B12 status may influence the risk or progression of AA. However, conflicting evidence from other studies prevents the establishment of clinical guidelines regarding serum screening or supplementation of these vitamins [42,43].

Biotin

Biotin plays a critical role in protein metabolism, particularly in the synthesis of keratin, a key structural protein in hair. Its involvement in these biochemical processes supports cellular growth and differentiation within hair follicles, thereby contributing to healthy hair development and maintenance [44]. Only one study has evaluated biotin supplementation in AA, reporting complete hair regrowth in one-third of patients treated with biotin, zinc, and topical clobetasol [43,45]. However, the specific effect of biotin remains uncertain, highlighting the need for further targeted research [43,45]. Biotin supplementation is not supported by available data for the treatment of AA.

Conclusions

Our study highlights the role of various daily lifestyle factors and supplements in the pathogenesis of AA, though the precise molecular mechanisms remain unclear. Given the limited clinical evidence on the impact of lifestyle factors, specific recommendations for patients with AA cannot be made. Further clinical trials and epidemiological studies are necessary to better understand the molecular mechanisms and guide future lifestyle interventions for these patients.

Disclosures:

Author's contribution

Conceptualization, Martyna Różańska; methodology, Martyna Różańska; software, not applicable; check, Karolina Niewola; formal analysis, Karolina Niewola, Kacper Rozenberg; investigation, Martyna Różańska, Justyna Stryjecka; resources, not applicable; data curation, Justyna Stryjecka; writing - rough preparation, Martyna Różańska, Karolina Niewola, Kacper Rozenberg; writing - review and editing, Karolina Niewola, Justyna Stryjecka; visualization, Karolina Niewola; supervision, Martyna Różańska; project administration, Martyna Różańska; receiving funding, not applicable

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