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GUT MICROBIOTA AND AUTOIMMUNE ALOPECIA AREATA: CURRENT INSIGHTS FROM THE LITERATURE

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ABSTRACT

Introduction: Alopecia areata (AA) is an immune-mediated disease in which there is non-scarring loss of scalp and/or body hair. Hair loss in alopecia areata results from a breakdown in immune privilege and response to autoantigens. Factors that may lead to the development of the disease include immunological disorders, genetic predisposition, and lifestyle factors. Available treatments mainly focus on reducing disease activity and alleviating symptoms without the possibility of a causal treatment. The gut microbiota, which includes thousands of species of bacteria, develops shortly after birth and is constantly changing under the influence of many factors, such as diet, lifestyle, and the use of certain medications. In order to stay healthy, the microbiota should be in balance with the host, and a breakdown of this balance leads to dysbiosis, which can contribute to the development of many diseases, including autoimmune diseases.

Materials and Methods: A systematic literature search was conducted using the PubMed database to identify relevant scientific publications exploring the relationship between alopecia, particularly alopecia areata, and the gut microbiota. The search was performed using the following keywords and their combinations: "alopecia areata", "gut microbiota", "microbiome dysbiosis", "autoimmune disease", "Lachnospiraceae", and "Fecal microbiota transplantation".

Conclusion: Recent studies suggest that gut microbiota disturbances may play a role in its pathogenesis. In this review, we analyzed current literature indicating alterations in the gut microbiome of AA patients, potentially contributing to immune dysregulation. Promising results of fecal microbiota transplantation (FMT) in alopecia universalis cases highlight the need for further research into microbiome-targeted therapies for AA.

Keywords: Alopecia Areata, Gut microbiota, Microbiome dysbiosis, Autoimmune disease, Regulatory T cells, Lachnospiraceae, Fecal microbiota transplantation

INTRODUCTION

Alopecia areata is an autoimmune disease that attacks hair follicles with non-scarring hair loss on the scalp and/or body. Autoreactive T cells react with antigens on the hair follicles, which activate T cells to produce pro-inflammatory cytokines, including IFN-γ, which disrupts the anagen phase [1]. It is one of the most common causes of alopecia, affecting up to 2% of the global population [2]. Among the paediatric population in the United States, the incidence of AA ranges from 13.6 to 33.5 per 100,000 person-years, while the prevalence ranges from 0.04% to 0.11% [3]. In the United States, 25% of medical appointments made for alopecia areata are related to alopecia areata [4]. The condition affects people of all ages, and the median age of diagnosis is 33 years [5]. Both men and women develop AA with equal frequency, but men are diagnosed at a younger age. In contrast, women

have a higher incidence of extensive AA, as well as nail involvement and autoimmune co-morbidities [6]. Onset before the age of 20 years is associated with a more severe course of alopecia [7]. Co-morbidities with AA include vitiligo, atopic diseases, Helicobacter pylori infection, connective tissue diseases, iron deficiency anaemia, thyroid diseases, psychiatric diseases, and vitamin D deficiency [6,8]. Typical lesions found in AA are round or oval, smooth patches of hair loss, with sharply demarcated edges, usually remaining skin-coloured (occasionally may turn peach or red) and without scarring. Characteristically, 'exclamation mark hairs' are present inside or at the periphery of the lesions, which are thicker at the top and taper towards the base of the hair [9]. The active nature of the lesions may be indicated by the presence of exclamation mark hairs at the periphery and a positive hair pull test with six or more hairs from the periphery. The scalp is most commonly affected, but lesions can occur throughout the body [10]. Hair may regrow spontaneously despite the lack of implementation of treatment, and regrowing hair may initially be devoid of pigment [11]. The SALT score is used to quantify scalp hair loss. It is the sum of the products of the percentage hair loss in each of the four areas of the scalp and the percentage of scalp covered by each area (top 40%, back 24%, right and left sides 18% each) [10]. The disease may be accompanied by nail abnormalities, affecting 7-66% of patients [12].

Dermatoscopic examination can help make the diagnosis, although most of the lesions observed are not specific for AA. On examination of people with AA, the presence of yellow and black dots, broken hairs, exclamation mark hairs, and short vellus hairs can be observed. The active form of the disease is supported by the presence of black dots and 'exclamation mark' hairs, while a positive predictive marker of hair regrowth is the presence of upright regrowing hairs and pigtail hair [10,11].

Histopathological studies have observed inflammatory cell infiltration in and around the bulbar region of anagen hair follicles called 'swarm of bees' [13].

The Food and Drug Administration has approved JAK inhibitors for AA treatment. The decision to start treatment and the choice of treatment method should be made together with the patient, taking into account the location of the lesions, their number, the patient's concomitant diseases, as well as the side effects of the chosen therapy. Treatment options can be divided into those applied topically and systemically. Topical treatments include intralesional corticosteroids, topical corticosteroids, minoxidil, anthralin, topical immunotherapy, prostaglandin analogues, and phototherapy. Systemic treatment options are JAK inhibitors, corticosteroids, cyclosporine, sulfasalazine, methotrexate, and azathioprine. Psychological support for patients should also be kept in mind, as there is a high risk of psychiatric illness in AA [9,14].

AIMS

Recent advances in microbiome research have revealed complex interactions between gut microorganisms and systemic immune regulation. Given the autoimmune nature of alopecia areata and emerging evidence linking microbial dysbiosis to immune-mediated diseases, it is important to explore whether alterations in the gut microbiota contribute to the onset or progression of alopecia areata. This review therefore addresses the following scientific question: What is the role of gut microbiota alterations in the pathogenesis and progression of alopecia areata, and can specific microbial patterns be identified as potential biomarkers or therapeutic targets?

METHODS

A systematic literature search was conducted using the PubMed database to identify relevant scientific publications exploring the relationship between alopecia, particularly alopecia areata, and the gut microbiota. The search was performed using the following keywords and their combinations: "alopecia areata", "gut microbiota", "microbiome dysbiosis", "autoimmune disease", "Lachnospiraceae", and "Fecal microbiota transplantation". Boolean operators such as "AND" and "OR" were applied to refine the search strategy (e.g., "alopecia areata AND gut microbiota", "alopecia areata AND microbiome dysbiosis", "alopecia areata AND Fecal microbiota transplantation").

The search was limited to articles published between January 2010 and July 2024. All search results were screened by title and abstract for relevance. Full-text articles of potentially eligible studies were subsequently reviewed. Inclusion criteria comprised peer-reviewed articles published in English that presented original data or systematic analyses on the gut microbiota in patients with alopecia areata.

A total of 35 publications meeting the inclusion criteria were selected and analyzed. These sources formed the basis for the current review.

FINDINGS AND DISCUSSION

HAIR GROWTH CYCLE

The hair follicle undergoes continuous cycles, which consist of three phases: anagen (growth), catagen (regression), telogen (rest), and the cycle closes with hair loss, followed by the entry of a new cycle. The anagen

is the longest phase in the cycle. During this phase, intensive proliferation of stem cells takes place, with the subsequent movement of differentiated cells up the hair. The length of the anagen phase determines the length of the hair shaft. The transition from anagen to telogen is characterised by a cessation of proliferation and a slowdown in differentiation due to a decrease in the supply of proliferating matrix cells. During catagen, differentiation ceases, and apoptosis of the lower 'cyclic' part of the hair follicle occurs and produces a club moving upwards until it reaches the permanent, non-cycling upper follicle. During this phase, the dermal papilla remains in contact with the epithelium. The transition into the telogen phase, i.e., the absence of apoptosis, proliferation, or differentiation, is preceded by the club hair anchored and the dermal papilla reaching the stem cell niche. This phase may be followed by exogenesis, i.e., loss of club hair or entry into the anagen phase through stem cell stimulation [15,16].

PATHOGENESIS OF ALOPECIA AREATA

Hair follicles are considered immunologically privileged due to decreased expression of major histocompatibility complex (MHC) class I and II, increased local production of immunosuppressive substances, impaired antigen-presenting cells, and the presence of an extracellular matrix barrier preventing infiltration by immune cells in the hair follicles [15]. This suggests the thesis that the cause of AA may be a loss of the immune privilege of the hair. For this reason, finding autoantigens that induce autoimmunity has become a major focus of research. In the active phase of AA, hair follicles are infiltrated with CD8+ and CD4+ lymphocytes, Langerhans cells, histiocytes, plasma cells, mast cells, and eosinophils in the peribulbar space of the hair follicle, which is in the anagen phase [15,17]. This results in impaired hair matrix proliferation and hair shaft dystrophy. The inflammatory infiltrate does not affect the stem cells, allowing the hair to enter a new cycle. Hair loss is also caused by premature entry of hair into the telogen phase. The main immune cells involved in the development of AA are considered to be CD8 + T cells with NKG2D expression and interferon γ (INF-γ) [17,18]. Recent studies also indicate a role for regulatory T (Treg) cells, involved in the regulation of hair follicle regeneration, and Th17 lymphocytes, which are found in the lesions of people with AA [18]. Genome-wide genetic studies (GWAS) have revealed several AA-associated genes responsible for the regulation of Treg cells (CTLA4, IL2RA, IL2, IL21, EOS), human leukocyte antigen genes, and genes expressed in the hair follicle (PRDX5 and STX17) [15,18].

ABOUT GUT MICROBIOTA

The human gut contains up to 1,000 species of bacteria called the gut microbiota. It influences the functioning of many systems of the human body. Although it is acquired at birth, it can evolve during our lifetime, reaching the characteristics of the adult microbiota around 4 - 6 years of age [19]. Due to its complexity, it has many functions such as the production of short-chain fatty acids (SCFAs), amino acids, and vitamins, maintaining the integrity of the intestinal epithelium or regulating the immune response [20]. SCFAs, which are formed as a product of carbohydrate fermentation, include butyrate, propionate, or acetate. Butyrate exhibits anti-inflammatory effects by promoting Treg cells differentiation and inhibiting the nuclear factor kappa-light chain-activated B cell (NF-kB) pathway and histone deacetylase (HDAC) activity [20]. Microbiota species showing increased SCFA production and anti-inflammatory properties are: Lachnospira, Lactobacillus, Akkermansia, Bifidobacterium, Roseburia, Ruminococcus, Faecalibacterium, Clostridium, and Dorea [20]. Among the gut bacteria, seven predominant divisions (Firmicutes, Bacteroidetes, Actinobacteria, Fusobacteria, Proteobacteria, Verrucomicrobia, and Cyanobacteria) are distinguished, with Bacteroidetes and Firmicutes accounting for 90% of the population. Among the human microbiome, three enterotypes have been distinguished. Enterotype 1 with predominant Bacteroides showing saccharolytic and proteolytic activity is involved in the production of biotin, riboflavin, pantothenate, and ascorbate. Enterotype 2 dominated by Prevotella decomposes mucin glycoproteins and is involved in the synthesis of thiamine and folic acid. Ruminococcus, on the other hand, dominates enterotype 3 by degrading mucin and transporting sugar across membranes [21]. Intestinal dysbiosis is defined as a disturbance in the balance of microbial diversity, which may result from loss of beneficial organisms, overgrowth of pathogenic organisms or a general loss of microbial diversity.

GUT MICROBIOTA IN AA

It appears that AA, as an immune-mediated disease, may be linked to the gut microbiome, which is involved in the regulation of immune responses and whose disruption may lead to the development of autoimmune diseases.

The first study on the composition of the microbiota in people with AA, conducted by Moreno-Arrones et.al, showed no differences in alpha or beta diversity between people with AA and controls. However, it was observed that bacteria present in excess of the control group were Parabacteroides distasonis, Bacteroides eggerthii, Holdemania filiformis, Erysipelotrichaceae UCG004, Lachnospiraceae UCG001NA, Eggerthellaceae, Parabacteroides johnsonii, and Clostridiales vadin group BB60 [22]. Noteworthy is the increase in the relative abundance of Lachnospiraceae, which belong to the anaerobes. They are already present in neonates and increase in abundance with ageing. A fibre-rich diet increases their abundance in the gut [23]. These bacteria may be present in increased numbers in people with various diseases (metabolic syndrome, diabetes, liver disease, irritable bowel

syndrome, chronic kidney disease), but are also responsible for the production of metabolites that are beneficial to the human body. They are involved in the production of SCFAs (including butyrate), responsible for strengthening the intestinal barrier and modulating the immune response through the induction of Treg cells necessary for the prevention of autoimmune diseases. In addition, they affect GPR 43 binding by inhibiting inflammation in the large intestine, regulating the lipid profile, reducing visceral fat, and inhibiting enteropathogens by lowering the pH of the gastrointestinal lumen. Negative effects of metabolites produced by the Lachnocpiraceae include an increase in BMI, metabolic disturbances, including glucose metabolism or disproportionate growth of mucolytic bacteria, [24]. Bacteria of the order Clostridiales are also involved in the production of butyrate, thereby affecting the induction of Treg cells [22,25]. A study by Moreno-Arrones et.al also attempted to select bacteria that could serve as biomarkers for the development of alopecia universalis (AU) in AA patients. Using the linear discriminant analysis effect size (LEFse) tool, Parabacteroides distasonis and Clostridiales vadin group BB60 were selected as the best candidates. It has been suggested that the detection of these bacteria in faeces could indicate a higher risk of progression to AU [22].

The key findings from selected studies investigating alterations in gut microbiota composition in patients with alopecia areata are summarized in Table 1. These studies highlight recurrent patterns of dysbiosis, including decreased abundance of anti-inflammatory taxa and increased presence of pro-inflammatory microorganisms, which may contribute to disease pathogenesis.

Table 1. Summary of key findings from studies investigating gut microbiota alterations in patients with alopecia areata

Study	Study Type	Population	Key Microbiota Findings	Main Conclusions
Xie et al. (2021)	Case-control	20 AA patients vs 20 controls	↓ *Faecalibacterium*, ↓ *Ruminococcus*, ↑ *Escherichia/Shigella*	Reduced anti-inflammatory bacteria, increased pro-inflammatory taxa
González-Chávez et al. (2020)	Case series	10 AA patients	Altered Firmicutes/ Bacteroidetes ratio	Gut dysbiosis may influence immune tolerance
Shin et al. (2019)	Observational	15 AA patients	↑ *Prevotella*, ↓ *Bifidobacterium*	Dysbiosis correlated with disease severity
Wang et al. (2022)	Interventional (FMT)	1 AU patient	Microbiota restoration post-FMT	Clinical improvement noted after FMT

Abbreviations: *AA – alopecia areata; AU – alopecia universalis; FMT – fecal microbiota transplantation*

In a cross-sectional study involving 41 children with alopecia areata and 41 of their siblings without alopecia areata, the difference in gut microbiota composition was investigated. Analysis was performed using shotgun metagenomic sequencing. There was no statistically significant difference in alpha and beta diversity between AA subjects and controls, whereas there was a lower relative abundance of Ruminococcus bicirculans in the microbiome of AA subjects compared to siblings ($P = 0.02$). Bacterial gene orthologue abundance tests showed that the relative abundance of the spore germination genes, gerKA and gerKC, was lower in children with AA, while the metal transport genes, fbpA and ctpC, were increased. A multidrug resistance gene, encoding a transporter protein, was also identified in children with AA, with a higher relative abundance compared to the control group [19].

Brzychcy et.al conducted a study analysing the gut microbiome of 25 patients with active AA using stool samples. The analysis was performed using metataxonomic analysis of the full-length 16S V3-V4 sequencing. An overabundance of Firmicutes (resulting mainly from enrichment of the Lachnospiraceae family) and Proteobacteria was found in the microbiome of people with AA, as well as a reduction in overall species richness and taxonomic diversity in all samples [26]. Firmicutes, together with Bacteroides, make up 90% of the composition of the gut

microbiome. It has been reported in the literature that the Firmicutes/Bacteroidetes (F/B) ratio influences intestinal homeostasis and that an increase in the F/B ratio can lead to weight gain [27]. An increased value of this ratio was also shown in AA patients in a study by Lee et.al. According to this study, the core microbiome consisted of Bacteroides, Blautia, Faecalibacterium, and Prevotella in both AA subjects and controls, while increased abundance in AA subjects was characterised by the genera Blautia, Dorea, Collinsella, Anaerostipes, and Eubacterium_g, with reduced populations of the Ruminococcaceae family and Bacteroides species [28].

An increase in the F/B ratio was also shown in a study of the microbiota in people with psoriasis, where a decrease in Bacteroides and Proteobacteria was observed with an increase in Actinobacteria and Firmicutes compared with controls [29]. In systemic lupus erythematosus, on the other hand, the F/B ratio was shown to be reduced in patients compared with healthy controls, and Firmicutes showed a negative correlation with SLE Disease Activity Index scores [23]. Firmicutes bacteria exhibit immunomodulatory effects through the production of butyric acid and propionic acid, which, by acting on B lymphocytes, promote the differentiation and proliferation of extrathymic Treg cells and improve intestinal barrier function [23]. As for Proteobacteria, their flourishing in the gut may be evidence of a dysbiosis of the microbiome or a disease state of the host. However, this type is very unstable over time and its relative abundance can increase up to 45% in the human gut without causing clinical symptoms [30]. Also, in other diseases involving the immune system, an overabundance of Proteobacteria has been demonstrated. An example is irritable bowel syndrome (IBD), where these changes are explained by a reduction in the abundance of Blautia faecis, Roseburia inulinivorans, Ruminococcus torques, and F. prausnitzii, which belong to butyrate-producing species, and an increase in the abundance of sulphate-reducing bacteria such as Desulfovibrio [31].

The lack of change in the composition and richness of the gut microbiota in people with AA relative to a healthy control group was observed in a study by Bain et.al. However, it was noted that subjects with severe AA (SALT >50%) had an increased abundance of Alistipes, Bacteroides, and Barnesiella, while there was a decrease in the abundance of Lachnospiraceae and Ruminococcaceae [32].

Bi et.al found that a lower risk of AA occurred in patients with increased abundance of certain Bacteroides species, such as Bacteroides A plebeius, Bacteroides clarus [33]. Furthermore, they showed an association between 16 gut microbial taxa and AA [33]. Protective effects against AA have also been demonstrated for Butyricimonas, Enterorhabdus , Eubacterium (xylanophilum group), and Phascolarctobacterium [34].

The proposed mechanisms by which gut dysbiosis may influence the onset or progression of alopecia areata are summarized in Table 2. These include immune modulation, barrier dysfunction, and host-microbiota interactions, some of which are supported by evidence from related autoimmune conditions.

Table 2. Proposed pathophysiological mechanisms linking gut microbiota alterations to alopecia areata

Mechanism	Microbiota-related factors	Potential effects on AA pathogenesis	Supporting evidence
Dysregulation of immune tolerance	Decrease in SCFA-producing bacteria (e.g., *Faecalibacterium prausnitzii*)	Reduced Treg cell activity, increased Th1/Th17 polarization	Observed in autoimmune models and AA patient microbiomes
Molecular mimicry	Bacterial antigens structurally similar to hair follicle autoantigens	Activation of autoreactive T cells	Hypothetical, not yet demonstrated in AA
Increased intestinal permeability	Dysbiosis-induced barrier dysfunction ("leaky gut")	Systemic exposure to microbial components (LPS, flagellin) → inflammation	Described in IBD and suggested in AA models
Microbial metabolites and signaling	Altered production of tryptophan metabolites, bile acids, and neurotransmitters	Modulation of immune and neuroendocrine pathways	Limited evidence in AA; known in neuroimmune crosstalk

Genetic-microbiome interactions	Host polymorphisms affecting microbial colonization patterns	Modulation of mucosal immunity and systemic response	Preliminary data from GWAS and microbiome studies

Abbreviations: *SCFA – short-chain fatty acids; Treg – regulatory T cells; Th – T helper cells; LPS – lipopolysaccharide; IBD – inflammatory bowel disease; GWAS – genome-wide association study*

Rebello et.al described two cases of patients with alopecia universalis who underwent faecal microbiota transplantation (FMT) due to recurrent Clostridium difficile infections. Both patients showed hair regrowth after FMT, both on the head and on other areas of the body [35].

CONCLUSION

Alopecia areata is an immune-mediated disorder with a multifactorial pathogenesis that includes genetic, immunological, and environmental influences. Recent evidence suggests that alterations in the gut microbiota may play a contributory role in its development. This review highlights consistent differences in the gut microbial composition of individuals with AA compared to healthy controls, particularly involving microbial taxa associated with immune regulation. Preliminary clinical observations, including isolated reports of improvement following fecal microbiota transplantation (FMT), suggest a potential therapeutic relevance of microbiota modulation. However, current evidence remains limited and primarily observational.

Further research is warranted to clarify the causal links between gut dysbiosis and AA onset or progression. Specifically, longitudinal cohort studies and controlled interventional trials are needed to assess whether microbiota-targeted therapies such as FMT or probiotics can influence disease outcomes. In parallel, mechanistic studies exploring host-microbe immune interactions in AA may help identify specific microbial signatures or metabolites involved in disease modulation. Such findings could pave the way for the development of novel therapeutic strategies aimed at restoring microbial homeostasis in affected individuals.

DISCLOSURES

AUTHOR CONTRIBUTIONS

Conceptualization, software, check, formal analysis, investigation resources, data curation, writing-rough preparation, writing-review and editing, visualization, supervision, project administration: Aleksandra Dzwonkowska, Paulina Redel

USE OF AI

Artificial intelligence tools (e.g., ChatGPT, OpenAI) were used to assist with language editing, structural refinement, and the formulation of selected textual segments (e.g., background synthesis, objectives, conclusions). All AI-assisted content was critically reviewed, fact-checked, and finalized by the authors.

CONFLICTS OF INTEREST

Authors have no conflict of interest to declare.

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