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Mitochondrial Dysfunction in Lichen Planopilaris with Focus on Oxidative Stress and Metabolic Reprogramming

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Abstract

Mitochondrial dysfunction plays a notable role in the pathogenesis of lichen planopilaris (LPP), a chronic scarring alopecia characterized by perifollicular inflammation and progressive follicular destruction. Emerging evidence suggests that oxidative stress, driven by mitochondrial dysfunction, acts as a central mechanism underlying cellular damage and immune dysregulation in LPP. Damaged mitochondria contribute to the overproduction of reactive oxygen species (ROS), which exacerbate inflammatory responses, impair cellular repair processes, and promote the apoptosis of follicular keratinocytes. Additionally, mitochondrial dysfunction in LPP has been linked to metabolic reprogramming, characterized by a shift from oxidative phosphorylation to glycolysis, further amplifying ROS production and sustaining chronic inflammation. This metabolic shift disrupts follicular stem cell homeostasis, compromising the regenerative capacity of hair follicles and contributing to permanent scarring. Therapies targeting mitochondrial dysfunction and oxidative stress are gaining attention as potential treatments for LPP. Antioxidants, such as N-acetylcysteine and coenzyme Q10, have demonstrated promise in reducing oxidative damage and mitigating inflammation in preclinical models. Furthermore, interventions aimed at improving mitochondrial function, including PGC-1α agonists and mitochondrial-targeted ROS scavengers, offer potential for halting disease progression. The integration of these approaches with existing therapies, such as immunosuppressants and topical corticosteroids, may enhance treatment outcomes by addressing both inflammatory and metabolic aspects of LPP. Understanding the connection between mitochondrial dysfunction, oxidative stress, and metabolic reprogramming provides a comprehensive framework for advancing therapeutic strategies and improving management of this disorder.

1. Introduction

Lichen Planopilaris (LPP) is an inflammatory primary cicatricial alopecia, a form of inflammatory, scarring hair loss [1]. The etiology of LPP is unknown. However, due to the histopathology findings of lichenoid perifollicular inflammation, it is thought to be a follicular variant of lichen planus [2]. It is the most common cause of scarring hair loss, with a prevalence of approximately 13.4 per 100,000 patients [3]. There are three forms of LPP – classic LPP, frontal fibrosing alopecia (FFA), and Graham-Little-Piccardi-Lasseur syndrome (GLPLS) [1]. In classic LPP, hair loss occurs in an irregular pattern in one

or several portions of the scalp, most common in the vertex region. FFA causes hair loss in a bandlike distribution, and the patient may also experience eyebrow hair loss. GLPLS, the least common of the three variants, the patient presents with “scarring alopecia on the scalp, non-scarring hair loss in the axillary and pubic regions, and the appearance of lichenoid follicular papules on the trunk and extremities” [4].

The most affected patient demographic is women between 40 and 60 years old. There is also an association with mucocutaneous lichen planus in about half of the patients [2]. This association further supports the theory that LPP is a variant of lichen planus. LPP can have a devastating impact on the mental health of affected patients. Several studies have noted an association between anxiety, depression, and lower reported quality of life and LPP [5], [6], [7]. This association can be due to many reasons, including the burden of managing the pain and pruritus caused by LPP, or decreased self-esteem related to hair loss.

Mitochondria are the organelles responsible for cellular metabolism and homeostasis. These responsibilities include generating ATP through oxidative phosphorylation, regulating cellular redox reactions by releasing reactive oxygen species (ROS), regulating calcium homeostasis, and initiating apoptosis [8], [9]. These functions are important to the cell's overall health and, therefore, the person's overall health. Several studies have investigated the theory that the ROS released by mitochondria causes oxidative damage over time, leading to aging and age-related conditions such as Alzheimer's, heart disease, and cancer [10]. Mitochondria also serve an important function in our skin and hair's health by providing the energy necessary and meeting the cellular demands of rapid skin cell turnover. Mitochondria dysfunction may be involved in dermatological conditions such as vitiligo due to mitochondria-driven cell death of melanocytes and psoriasis due to the promotion of inflammation by ROS [11], [12], [13]. If mitochondria play a role in these conditions, it is logical to investigate mitochondria's role in other dermatological conditions, such as LPP.

Lichen Planopilaris is due to an autoimmune attack on the bulge of the hair follicle. The exact trigger of this attack is unknown [14]. However, recent studies have identified dysfunctional mitochondria within the bulge of lesional LPP hair follicles on biopsy [15]. Mitochondrial dysfunction drives oxidative stress and metabolic reprogramming, creating the cellular conditions in which conditions such as LPP, lichen planus, and others can develop [16]. If mitochondrial dysfunction plays a central role in the etiology of LPP, further exploration of mitochondria-focused treatment options is necessary. The following literature review will synthesize current knowledge regarding the role of mitochondrial dysfunction in LPP and highlight potentially helpful therapeutic options. This review aims to bridge the gap between understanding mitochondrial dysfunction and the inflammatory process seen in LPP.

2. Mitochondrial Dysfunction In LPP

Mitochondrial dynamics are essential in determining hair cell differentiation and proliferation. During the transition of hair follicles into the growth phase (anagen), hair follicle stem cells experience a metabolic shift towards oxidative phosphorylation, leading to increased mitochondrial activity [17]. This further demonstrates that mitochondria play a crucial role in regulating energy metabolism, which supports stem cell renewal in response to injury. It suggests that mitochondrial dynamics are critical for hair follicle cycling and indicates that mitochondrial dysfunction could hinder hair follicle regeneration, as seen in disorders like lichen planopilaris. A critical component to complete hair growth is the follicle's movement into the dermis and subcutis during the anagen phase. This process significantly contributes to the energy demands of the anagen phase [18]. The need for efficient mitochondrial biogenesis and function is crucial, as disruptions can compromise ATP availability and contribute to hair loss disorders such as lichen planopilaris.

Mutations in mitochondrial DNA (mtDNA) have the ability to impact follicular cells. mtDNA mutations contribute to increased hyperplasia with hyperkeratosis in the hair follicles of mice, leading to abnormal hair follicle development and reduced hair shaft production [19]. The findings suggest that mtDNA mutations significantly impair hair follicle function and hair growth, potentially contributing to the pathogenesis of LPP. These mitochondrial dysfunctions disrupt hair follicle structure and hinder mitochondrial biogenesis, a process crucial for hair growth that is largely regulated by peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α). Deletion of PGC-1α has been observed to reduce OXPHOS protein content and citrate synthase activity [20], [21]. This disruption

affects oxidative phosphorylation (OXPHOS), leading to a depletion of ATP in follicular keratinocytes. Similarly, the knockdown of the mitochondrial transcription factor A (TFAM) results in mitochondrial DNA (mtDNA) depletion and dysfunction of the electron transport chain (ETC) complexes, compromising ATP production. Elevated levels of reactive oxygen species (ROS) have also been shown to reduce TFAM function (Stout & Birch-Machin, 2019). These observations suggest a potential link between improving mitochondrial function and mitigating the pathogenesis of Lichen Planopilaris (LPP).

Oxidative stress induced by reactive oxygen species (ROS) production contributes to the underlying pathophysiology of various diseases. High levels of reactive oxygen species (ROS) disrupt macromolecular and cellular structures, resulting in cellular damage. Additionally, the increased production of reactive oxygen species (ROS) impairs TFAM, leading to a reduction in hair follicle proliferation and increased apoptosis [11], [23]. The perifollicular immune response seen in Lichen Planopilaris (LPP) in addition to oxidative stress can further impair follicle function and impact disease progression. This highlights the importance of considering therapies targeting oxidative stress when evaluating a patient with Lichen Planopilaris.

3. Oxidative Stress In LPP

Reactive oxygen species (ROS) are generated from multiple sources which contribute to the pathophysiology of oxidative stress in Lichen Planopilaris. Mitochondria are the primary source of reactive oxygen species (ROS), making them vulnerable to ROS-induced mitochondrial DNA (mtDNA) damage [24]. This process contributes to mitochondrial dysfunction which further promotes ROS production, thereby creating a cycle of oxidative stress and inflammation. Similarly, inflammatory cytokines disrupt mitochondrial function, leading to increased ROS levels. ROS has been observed to form a “ring” within the hair follicle, indicating a localized region of oxidative stress [25]. This demonstrated the relationship observed in Lichen Planopilaris (LPP) patients, who exhibit increased immune activity in affected hair follicles. Furthermore, environmental factors such as UV radiation, pollutants, and smoking have been shown to increase the severity of alopecia [26], [27]. However, there is limited research on the impacts of environmental factors on Lichen Planopilaris pathogenesis, potentially leading to their underestimation in disease progression.

Emerging evidence suggests that Lichen planopilaris (LPP), a follicular form of lichen planus affecting hair follicles, is influenced by oxidative stress [28]. Understanding the clinical evidence of oxidative stress in LPP is crucial to improving diagnosis, treatment, and disease management. Peroxisome proliferator-activated receptor gamma (PPARγ) plays an important role in reducing lipid peroxidation, with levels of PPARγ significantly decreased in LPP [29], [30]. This finding suggests that PPARγ may play a significant role in regulating the MDA biomarker, associated with lipid peroxidation and commonly used as an indicator of oxidative stress. Similarly, oxidative stress is involved in the pathogenesis of alopecia areata and psoriasis, where MDA biomarkers have been identified as a valuable indicator for assessing disease severity [31]. Since oxidative stress is also involved in LPP, further research is necessary to determine if biomarkers of oxidative stress in patients with Lichen planopilaris can assess disease severity and improve treatment strategies.

Antioxidants have been recognized as a promising therapeutic option for various diseases, including Lichen planopilaris (LPP). Evidence from case studies and randomized controlled trials highlights their potential in combating the production of reactive oxygen species (ROS). Elbaky et al. reported that the administration of N-acetylcysteine (NAC) or coenzyme Q10 (CoQ10) alone or combined increased glutathione (GSH) levels [32]. An increase in GSH levels indicates a more effective neutralization of reactive oxygen species (ROS), leading to reduced levels of ROS. Additionally, Abdelsamie et al. conducted a randomized control clinical trial demonstrating that CoQ10 decreases malondialdehyde (MDA) levels in oral lichen planus (OLP) [33]. Since lower MDA levels indicated reduced lipid peroxidation, this suggests a decrease in ROS levels. Although OLP and LPP are two different diseases, they share several similarities and highlight the potential of therapies targeting oxidative stress in LPP. Mitochondrial-targeted antioxidants, such as Mitoquinone (MitoQ) present as a promising treatment for reducing mitochondrial oxidative stress. Mizuguchi et al discussed the successful use of MitoQ in reducing psoriatic symptom severity by inhibiting mitochondrial reactive oxygen species (mtROS) [13]. These examples highlight the necessity for additional research to clarify the connection between

oxidative stress and mitochondrial dysfunction in Lichen planopilaris, as this could yield valuable insights for reducing disease severity.

4. Metabolic Reprogramming in LPP

Cancer cells undergo metabolic reprogramming that includes increased aerobic glycolysis, known as the Warburg effect, where there is a shift towards glycolysis even in the presence of oxygen. Mitochondrial dysfunction plays a key role in this process [34]. In inflammatory and fibrotic skin conditions, a metabolic shift similar to the Walburg effect takes place. TGFβ regulates fibrosis and strongly activates glycolysis, which also promotes fibroblast activation. Keloid scarring and atopic dermatitis are linked to a metabolic shift that upregulates glycolysis and increases the use of glycolytic substrates, resulting in skin fibrosis and inflammation [24], [35]. A component found in hair follicles is the mammalian target of rapamycin (mTOR) signaling pathway proteins, which are known to have altered expression in patients with LPP. Additionally, dysregulation of mTOR is associated with multiple inflammatory disorders [36]. mTOR is known to result in glycolysis stimulation suggesting that mTOR may contribute to the increased glycolytic activity in hair follicle cells during LPP and have a significant role in initiating perifollicular inflammation. However, no studies have yet directly investigated the link between the mammalian target of rapamycin pathways and the glycolytic activity in hair follicle cells during lichen planopilaris, and further research is needed.

Lichen planopilaris (LPP) is marked by a decrease in lipid metabolic pathways, which are involved in the inflammatory response and progression of the disease. Histological analysis of LPP reveals perifollicular infiltrate and fibrosis concentrated in the hair follicle bulge, where hair follicle stem cells are located. This inflammatory response results in the replacement of hair follicles with fibrotic tracts, leading to the loss of hair follicles, a process absent in unaffected scalp tissue [37], [38]. This highlights the importance of metabolic reprogramming in LPP pathogenesis and suggests that targeting metabolic modulators could be beneficial in LPP treatments. Peroxisome proliferator-activated receptor-γ coactivator (PGC-1α) is a well-established regulator of mitochondrial biogenesis linked to promoting oxidative phosphorylation (OXPHOS) gene expression. PGC-1α is regulated by Sirtuin 3 (SIRT3) which yields an anti-Warburg effect [39]. This suggests that PGC-1α could be utilized to counteract mitochondrial dysfunction, restore balance, and reduce oxidative stress in LPP. Further research exploring the link between LPP and mitochondrial dysfunction could offer valuable insights into the pathogenesis and treatment of Lichen planopilaris.

5. Immune Dysregulation and Perifollicular Inflammation in LPP

Lichen Planopilaris (LPP) is a chronic, immune-mediated scarring alopecia characterized by an aberrant inflammatory response targeting the hair follicle. It is considered a follicular variant of lichen planus and primarily affects the bulge region of the hair follicle, where hair follicle stem cells reside. The immune dysregulation observed in LPP is driven by T cell-mediated mechanisms, particularly the involvement of Th1 and Th17 pathways, leading to sustained perifollicular inflammation. This inflammatory milieu results in progressive destruction of the follicular epithelium, ultimately leading to permanent alopecia. Recent studies have elucidated the roles of key cytokines and oxidative stress in propagating this immune response, reinforcing LPP as an autoimmune disorder with complex inflammatory signaling.

The immune pathophysiology of LPP is predominantly orchestrated by Th1 and Th17 cells, which mediate the inflammatory response against hair follicle keratinocytes. Th1 cells secrete interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-α), both of which contribute to follicular apoptosis and recruitment of cytotoxic T cells (CD8+) [40]. Th17 cells, on the other hand, release interleukin-17 (IL-17), a potent pro-inflammatory cytokine that amplifies neutrophilic infiltration and sustains chronic inflammation. These immune cascades are further reinforced by the upregulation of key inflammatory cytokines, including interleukin-1 beta (IL-1β), interleukin-6 (IL-6), TNF-α, and IFN-γ [40]. Elevated levels of these cytokines have been detected in lesional scalp biopsies, highlighting their central role in LPP pathogenesis. The recruitment and activation of immune cells at the perifollicular region ultimately disrupts the immune privilege of the hair follicle, making it susceptible to further immune-mediated destruction.

Oxidative stress plays a critical role in driving immune dysregulation in LPP by perpetuating inflammation and cellular damage. Reactive oxygen species (ROS), generated as a byproduct of chronic inflammation, act as signaling molecules that further recruit immune cells to the site of injury. Increased ROS levels contribute to the activation of nuclear factor kappa B (NF-κB) and mitogen-activated protein kinase (MAPK) pathways, both of which enhance the transcription of pro-inflammatory cytokines [41]. This creates a self-sustaining feedback loop where oxidative stress exacerbates immune activation, and in turn, immune cell infiltration leads to further ROS production. Additionally, mitochondrial dysfunction has been observed in LPP-affected hair follicles, further implicating oxidative stress as a key driver of disease progression [41]. The interplay between ROS-induced damage and immune-mediated follicular destruction suggests that targeting oxidative stress may offer a novel therapeutic approach in LPP management.

Current treatment strategies for LPP focus on dampening immune-mediated inflammation to halt disease progression. Corticosteroids, both topical and intralesional, remain the first-line therapy due to their potent anti-inflammatory effects in suppressing T cell activity and cytokine release. Calcineurin inhibitors, such as tacrolimus and pimecrolimus, have also demonstrated efficacy in reducing T cell-mediated inflammation by inhibiting interleukin-2 (IL-2) production [42]. More recently, Janus kinase (JAK) inhibitors, including tofacitinib and ruxolitinib, have been explored as potential therapies due to their ability to block multiple cytokine signaling pathways, particularly IFN-γ-driven inflammation [43]. Beyond conventional immunosuppressive strategies, novel therapeutic approaches targeting oxidative stress have gained interest. Mitochondrial-targeted antioxidants, such as coenzyme Q10 (CoQ10) and MitoQ, hold promise in mitigating ROS-induced tissue damage and breaking the inflammatory cycle [33]. Combining immune modulation with antioxidant therapy may provide a more comprehensive approach to LPP treatment, offering the potential to both suppress immune-mediated follicular destruction and protect against oxidative injury. Future research should focus on elucidating the precise mechanisms by which oxidative stress contributes to LPP pathology and exploring targeted interventions that can effectively modulate both immune dysregulation and oxidative damage.

6. Therapeutic Strategies for Lichen Planopilaris: Antioxidants, Mitochondrial Enhancement, and Metabolic Modulation

Lichen planopilaris (LPP) is characterized by perifollicular inflammation, progressive hair follicle destruction, and irreversible scarring. While immune dysregulation is central to LPP pathogenesis, emerging evidence highlights the role of mitochondrial dysfunction and oxidative stress as upstream contributors to disease progression. Oxidative stress, defined as an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, is a well-established driver of cellular damage, immune activation, and fibrotic remodeling [44]. Given this, therapeutic strategies aimed at reducing oxidative stress, enhancing mitochondrial function, and modulating metabolic pathways hold promise in mitigating LPP pathophysiology.

ROS are byproducts of mitochondrial respiration, and in a healthy state, the body maintains a balance between ROS generation and scavenging by endogenous antioxidants. However, in LPP, excessive ROS production due to mitochondrial dysfunction leads to oxidative damage of lipids, proteins, and DNA, resulting in chronic inflammation and apoptosis of follicular keratinocytes [45]. This is further exacerbated by immune cell infiltration and cytokine release, reinforcing a cycle of oxidative stress and inflammation.

Several biomarkers of oxidative stress have been detected in other inflammatory skin conditions, and preliminary findings suggest similar mechanisms in LPP. Elevated levels of 8-hydroxy-2′-deoxyguanosine (8-OHdG), a marker of oxidative DNA damage, and malondialdehyde (MDA), an indicator of lipid peroxidation, have been observed in patients with other scarring alopecias and inflammatory dermatological conditions, suggesting that oxidative damage may be an underexplored aspect of LPP pathogenesis [46].

Therapeutic approaches aimed at restoring redox balance have gained interest. N-acetylcysteine (NAC), a precursor to glutathione, is a well-known ROS scavenger that has been studied in various inflammatory diseases, including oral lichen planus. A study evaluating antioxidant therapy in oral lichen planus demonstrated a reduction in pain scores and disease severity following NAC supplementation in addition to pentoxyfyllin, suggesting its potential therapeutic benefit in LPP [47].

Similarly, Coenzyme Q10 (CoQ10), a mitochondrial antioxidant crucial for electron transport chain function, has been investigated for its role in reducing oxidative stress and mitochondrial dysfunction in dermatological disorders with trials showing comparable results to topical corticosteroids in LPP management [33]. While there is limited data on CoQ10 in LPP, its role in mitigating ROS-driven inflammation makes it a promising candidate for future clinical trials.

Traditional antioxidants face limitations in efficacy due to poor mitochondrial penetration and inadequate accumulation at the primary sites of ROS production. To address this, mitochondrial-targeted antioxidants such as MitoQ and SkQ1 have been developed, engineered to selectively localize within mitochondria and neutralize ROS at their source [48]. MitoQ, a ubiquinone derivative, and SkQ1, a plastoquinone-based antioxidant, have shown promising results in preclinical studies by improving mitochondrial function, reducing oxidative stress, and preserving cellular integrity in ROS-driven diseases. Given that LPP is associated with mitochondrial dysfunction, these targeted antioxidants present a potential therapeutic avenue to halt follicular destruction and promote hair follicle survival.

Beyond direct ROS scavenging, enhancing mitochondrial biogenesis and function represents another strategy to counteract oxidative stress in LPP. Peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α) is a master regulator of mitochondrial biogenesis and oxidative metabolism, playing a crucial role in energy homeostasis. PGC-1α activation has been explored as a potential therapeutic approach in neurodegenerative and metabolic disorders, given its ability to enhance mitochondrial efficiency and reduce ROS production [49]. While studies on PGC-1α in LPP remain limited, its role in modulating mitochondrial health, maintaining follicular stem cell populations, and preventing apoptosis suggests potential therapeutic relevance.

Another approach to mitigating oxidative stress is mitochondrial uncoupling, which reduces mitochondrial membrane potential and subsequently lowers ROS production. However, excessive uncoupling may lead to ATP depletion and energy deficits, necessitating precise modulation. While mitochondrial uncouplers have not been widely studied in dermatology, their potential role in conditions characterized by excessive ROS accumulation and metabolic dysfunction warrants further investigation.

Metabolic reprogramming is a hallmark of chronic inflammatory conditions, with cells shifting from oxidative phosphorylation (OXPHOS) to glycolysis in response to stress, a phenomenon similar to the Warburg effect observed in cancer cells. This shift allows immune and inflammatory cells to sustain their activation, fueling chronic inflammation and tissue destruction. Emerging evidence suggests that a similar metabolic shift occurs in LPP, where hair follicle cells may favor glycolysis over OXPHOS, contributing to disease pathology.

Targeting metabolic pathways provides a novel strategy to counteract inflammation and oxidative stress in LPP. Glycolysis inhibitors, such as 2-deoxy-D-glucose (2-DG), have been explored in inflammatory diseases due to their ability to suppress immune cell activation and ROS production [50]. Additionally, AMP-activated protein kinase (AMPK) activators, such as metformin, have demonstrated potential in reducing oxidative stress and inflammation by promoting mitochondrial efficiency [51].

Conversely, inhibition of the mechanistic target of rapamycin (mTOR) pathway, which plays a role in cellular metabolism, has been investigated for its anti-inflammatory effects. mTOR inhibitors have been shown to regulate T-cell activation and cytokine production, mechanisms that are central to LPP pathogenesis and have been shown [36]. Given the metabolic reprogramming observed in scarring alopecias, targeting these pathways may help restore energy homeostasis and reduce inflammatory damage.

Despite growing evidence linking oxidative stress, mitochondrial dysfunction, and metabolic reprogramming to LPP, clinical validation of targeted therapies remains in its infancy. Future research should focus on biomarker-driven studies to assess oxidative stress markers such as 8-OHdG and MDA in LPP patients and track their response to antioxidant therapy. Additionally, randomized controlled trials evaluating mitochondrial-targeted therapies, glycolysis inhibitors, and metabolic modulators are needed to determine their clinical efficacy. Given the multifaceted nature of LPP, a multi-targeted therapeutic approach integrating mitochondrial repair, metabolic reprogramming, and immune modulation may offer the most promising strategy.

By addressing the underlying metabolic and oxidative stress-driven mechanisms, future treatment strategies for LPP could shift beyond symptomatic immunosuppression to disease modification and prevention.

7. Therapeutic Strategies for LPP

Utilizing combination therapies in the treatment of LPP may lead to the most efficacious outcomes. Given the importance of targeting mitochondrial dysfunction, integration of such remedies with current, evidence-based treatment regimens offers a synergistic, multi-layered attack towards the underlying disease. Clinicians must carefully consider the extent of disease and severity of symptoms prior to initiating any therapies in combination [52]. Nevertheless, addressing both the inflammatory and metabolic aspects of LPP could significantly enhance long-term results.

In addition to the interventions described above, alternative treatment strategies may benefit certain patient populations as well. Incorporating such approaches either in tandem with or in place of classic therapeutic regimens help to curb serious adverse effects associated with the long-term use of medications like corticosteroids and immunomodulators. Platelet-rich plasma (PRP) therapy, a regenerative treatment approach utilizing a patient's natural blood products, has been recently studied in cicatricial alopecias, including LPP. Specific byproducts of PRP called alpha granules store and release a number of growth factors to enhance hair density, bolster epidermal thickness, and increase the number of follicular bulge cells [53]. Another supplemental strategy is the use of low-level light therapy (LLLT), which employs photobiomodulation to both decrease inflammation and optimize overall cellular function. Modulation of the natural hair cycle by LLLT specifically augments the anagen phase of hair growth to lead to improvements in hair density and diameter while minimizing shedding [54]. Lastly, though not extensively studied, certain nutritional and lifestyle interventions may curtail oxidative stress and global inflammation. Anti-inflammatory diets rich in antioxidants like polyphenols assist in stabilizing androgen levels to reduce hair turnover. Further, healthy outlets for stress management in day-to-day life slow the body's heightened response, thereby delaying disease progression [55]. Additional research focusing on these alternative measures is certainly warranted, but their promise in transforming the course of LPP remains strong.

8. Research Gaps and Future Research

There is currently no consensus on the exact mechanism of LPP. Many believe that the process involves autoimmune CD8 cytotoxic T-cell destruction of epithelial hair follicle stem cells (eHFSC) [56]. One study found that there has been accumulation of damaged and oxidised proteins, such as oxidized glutathione and the accumulation of medium and long chain fatty acids, which indicates mitochondrial dysfunction in frontal fibrosing alopecia (FFA) [56]. Due to the similar histopathological features between FFA and LPP, mitochondrial dysfunction has potential to contribute to the pathophysiology of LPP [56]. This assertion however, is an active area of research that has largely been unexplored. One pilot study identified mitochondrial cristae degeneration, a sign of severe mitochondrial damage, in those with LPP [15]. This same study also found that mitochondrial Transcription factor A (TFAM), which is essential for mitochondrial DNA transcription, was decreased in those with LPP [15]. Additionally, this study also artificially induced epithelial-to-mesenchymal transition and immune privilege collapse similar to that of LPP in healthy scalp hair follicles which subsequently induced decreased TFAM as well decreased O2 consumption [15]. These findings are highly suggestive of a link between LPP and mitochondrial dysfunction in the pathogenesis of the condition.

Further research is needed to expand on this pilot study to identify if this mitochondrial dysfunction is the cause or consequence of the follicular damage of LPP. Future investigation into the presence of TFAM, reactive oxygen species, and oxygen consumption rate in those with LPP are needed to fully establish a link between mitochondrial dysfunction and LPP. These can serve as biomarkers that could transform LPP management with new non-invasive methods of diagnosis, the ability to track disease severity, and the potential to monitor response to treatment. Additionally, future research can further investigate the microstructure of mitochondria at different stages of the disease utilizing electron microscopy or fluorescence-based mitochondrial staining [57]. Further research is also needed to understand the metabolomic and proteomic shifts in LPP. Metabolomics can help identify changes in ATP production, accumulation of medium and long chain fatty acids, and reactive oxygen species metabolism in the hair follicle [56], [57]. Proteomic profiling can illuminate which mitochondrial

proteins and enzymes are implicated, if at all, in response to LPP in the hair follicle [57]. As more information is uncovered on the role of mitochondrial dysfunction, clinical trials targeting PPAR-y signaling in hair follicles, which has been seen to be decreased in LPP, may show therapeutic benefit in preventing disease progression [15]. It would also be reasonable to conduct a clinical trial on potential benefits of mitochondrial antioxidants like Nicotinamide adenine dinucleotide (NAD+) and coenzyme Q-10 that have shown promise in conditions such as vascular dementia, migraines, and Peyronie disease [58], [59]. As more is learned about the exact pathogenesis of mitochondrial dysfunction in LPP, more targeted therapies will emerge with the potential to slow, mitigate, or reverse LPP.

9. Conclusion

Recent research linking mitochondrial dysfunction, oxidative stress, and metabolic reprogramming to the pathogenesis of LPP offers strong evidence on this complex disease. Mitochondrial dysfunction contributes to an overproduction of reactive oxygen species, perpetuating chronic inflammation and immune dysregulation. These findings highlight the need for more integrative treatment approaches that targets immune-mediated inflammation while also addressing underlying metabolic and mitochondrial disturbances. Emerging medical therapies such as mitochondrial-targeted antioxidants, metabolic modulators, and methods to improve mitochondrial biogenesis hold potential for clinical intervention. Their integration into current clinical practice in addition to traditional immunosuppressive therapies may lead to more effective, multifaceted treatment regimens that address and alleviate both inflammatory and metabolic aspects of LPP. By shifting the therapeutic paradigm from symptomatic management of disease management, the next phase of LPP research holds potential to significantly improve patient prognosis and quality of life. Continued exploration and research will be essential in understanding new methods to stop and even reverse progression of this scarring alopecia.

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