Finasteride and Its Influence on the Gut Microbiome

What we have been missing in the assessment and treatment of the Post-Finasteride Syndrome.

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Introduction

Finasteride, a 5-alpha reductase inhibitor, is a widely prescribed medication for the treatment of benign prostatic hyperplasia (BPH) and androgenic alopecia, exerting its effects by inhibiting the conversion of testosterone to dihydrotestosterone (DHT) (1). While its primary mechanism of action is centered on androgen metabolism, recent research has begun to unveil its broader physiological impact, extending beyond hormonal regulation to the gut microbiome and immune homeostasis (2).

Emerging evidence suggests that Finasteride may significantly alter the composition and diversity of gut microbiota, with downstream effects on intestinal permeability, immune modulation, and systemic inflammation (3). The gut microbiome, a dynamic ecosystem of trillions of microorganisms, plays a crucial role in maintaining metabolic balance, regulating neurotransmitter production, and modulating inflammatory responses (4). Disruptions to this delicate microbial equilibrium—such as those potentially induced by Finasteride's impact on androgen-sensitive bacterial populations—may contribute to gut dysbiosis, neuroinflammatory disorders, and metabolic dysfunction (5).

Furthermore, the gut's intricate relationship with the endocrine and immune systems raises concerns that Finasteride-induced shifts in microbiota may have far-reaching consequences. Changes in short-chain fatty acid (SCFA)-producing bacteria, such as *Faecalibacterium prausnitzii* and *Roseburia spp.*, could impair gut barrier integrity, leading to increased gut permeability ("leaky gut") and heightened systemic inflammation, factors implicated in autoimmune disorders and neurological conditions (6).

Given the growing recognition of the gut-brain-endocrine axis, understanding Finasteride's potential role in microbiome disruption is of paramount importance. This article explores the intricate connections between Finasteride, the gut microbiome, and overall health, highlighting the emerging evidence on its impact on microbial composition, immune regulation, and metabolic stability (7).

Androgen-Microbiome Interactions

Androgens, particularly dihydrotestosterone (DHT) and testosterone, play a crucial role in shaping microbial communities within the gut, directly influencing bacterial diversity, immune function, and overall gut homeostasis (8). By inhibiting 5-alpha reductase, Finasteride significantly reduces DHT levels, triggering a hormonal shift that may have profound consequences on the gut microbiome. This reduction in DHT can alter the abundance of commensal bacteria that rely on androgen signaling, disrupting the balance of microbial populations that contribute to intestinal health (9). Furthermore, with DHT's well-established immunoregulatory role in maintaining microbial diversity, its depletion may create an environment favoring an estrogen-dominant microbiota, further influencing immune responses and potentially predisposing individuals to gut dysbiosis. Shifting microbial populations toward estrogen-dominant microbiota, as DHT has immunoregulatory effects that balance gut microbial diversity (10).

Potential Dysbiosis and Gut Barrier Impairment

Research indicates that reduced DHT levels may contribute to intestinal barrier dysfunction by significantly altering gut microbiota composition. The suppression of androgens through Finasteride use can lead to a decline in beneficial butyrate-producing bacteria such as *Faecalibacterium prausnitzii* and *Roseburia spp.*, both of which play a critical role in maintaining gut integrity and modulating inflammation (11). A depletion of these protective microbes weakens the gut lining, making it more susceptible to increased permeability, commonly referred to as "leaky gut syndrome"(12). This compromised barrier function allows endotoxins like lipopolysaccharides (LPS) to translocate into the bloodstream, triggering systemic immune activation. The resulting chronic low-grade inflammation has been strongly associated with a range of metabolic and neurological disorders, potentially exacerbating conditions such as mood disturbances, cognitive dysfunction, and autoimmune dysregulation (13).

Influence on Short-Chain Fatty Acids (SCFAs)

Short-chain fatty acids (SCFAs), particularly butyrate, are essential for maintaining gut homeostasis, modulating immune responses, and providing neuroprotective effects. Disruptions in the gut microbiome caused by Finasteride-induced androgen suppression may lead to a significant reduction in SCFA production (14). This decline in butyrate and other SCFAs can contribute to increased gut permeability and systemic inflammation, exacerbating conditions associated with immune dysfunction and metabolic imbalance. Furthermore, SCFAs influence neurotransmitter synthesis and signaling, particularly through their impact on the gut-brain axis (15). A reduction in butyrate levels may impair the regulation of key neurotransmitters such as GABA and serotonin, potentially contributing to the neuropsychiatric symptoms observed in some individuals experiencing Post-Finasteride Syndrome (PFS), including depression, anxiety, cognitive impairment, and emotional instability (16).

Immune Dysregulation and Autoimmunity Risk

Finasteride's suppression of androgen levels may significantly disrupt the intricate balance between pro- and anti-inflammatory cytokines, leading to immune dysregulation within the gut (17). This disturbance can have profound consequences, including an increased risk of autoimmune conditions due to the modification of T-regulatory cell function. Androgens play a crucial role in modulating immune tolerance, and their reduction may lead to heightened immune reactivity, increasing susceptibility to autoimmune responses. Additionally, the Finasteride-induced shift in gut microbiota may promote dysbiosis-associated inflammation, further exacerbating systemic immune activation. This chronic inflammatory state has been implicated in a range of symptoms frequently reported by long-term Finasteride users, including persistent brain fog, fatigue, and mood disturbances (18). The interplay between immune dysregulation and gut microbiome alterations underscores the need for a more comprehensive understanding of how androgen deprivation therapies impact both neurological and systemic health.

Psychological and Physical Complaints from Finasteride-Induced Gut Dysbiosis

If Finasteride disrupts the gut microbiome, it can initiate a chain reaction of dysfunctions across the gut-brain, gut-immune, and gut-endocrine axes, leading to a broad spectrum of neurological, immune, and metabolic disturbances (19). The resulting imbalance in microbial composition can weaken gut barrier integrity, trigger systemic inflammation, and alter neurotransmitter production—mechanisms that may contribute to both psychological and physical symptoms as discussed in these following sections.

Psychological Complaints

Anxiety and Depression

Finasteride's impact on the gut-brain axis may contribute to the development of anxiety and depression by altering the production of key neuroactive compounds. The reduction of short-chain fatty acids (SCFAs), particularly butyrate, diminishes their anti-inflammatory and neuroprotective effects, potentially impairing mood regulation (20). Additionally, a decline in beneficial bacterial strains such as *Lactobacillus spp.* and *Bifidobacterium spp.* may lead to decreased production of serotonin and GABA, two neurotransmitters essential for maintaining emotional stability. This dysbiosis-driven neurotransmitter imbalance could manifest as heightened anxiety, mood instability, and depressive symptoms (21). Furthermore, increased gut permeability—commonly referred to as "leaky gut"—allows endotoxins to enter systemic circulation, triggering chronic inflammation and neuroinflammatory responses. This inflammatory cascade may exacerbate symptoms such as anhedonia, fatigue, and brain fog, further linking gut dysfunction to the neuropsychiatric effects observed in some Finasteride users (22).

Cognitive Impairment ("Brain Fog")

Cognitive impairment, often described as "brain fog," may arise from Finasteride-induced disruptions in gut microbiota. A reduction in short-chain fatty acid (SCFA)-producing bacteria, such as *Faecalibacterium prausnitzii* and *Roseburia spp.*, may lead to decreased vagal nerve stimulation, which plays a critical role in maintaining cognitive clarity and mental sharpness (23). Furthermore, alterations in microbial composition may promote systemic inflammation, increasing the levels of pro-inflammatory cytokines such as TNF-α and IL-6. These cytokines can cross the blood-brain barrier, triggering neuroinflammatory responses that impair cognitive function and contribute to mental fatigue (24). Many individuals suffering from post-Finasteride syndrome (PFS) report persistent symptoms of poor memory, difficulty concentrating, and slowed processing speed. These cognitive disturbances may be directly linked to gut-driven neuroinflammation, reinforcing the critical role of microbiome health in maintaining optimal brain function (25).

Sleep Disturbances and Insomnia

Sleep disturbances and insomnia may be linked to Finasteride-induced gut dysbiosis, which disrupts essential neurotransmitter pathways involved in sleep regulation. The gut microbiome plays a critical role in serotonin production, a key precursor to melatonin, the hormone responsible for maintaining circadian rhythm and sleep quality. A reduction in beneficial bacteria such as *Lactobacillus* and *Bifidobacterium* may lead to decreased serotonin availability, impairing melatonin synthesis and contributing to sleep-onset and maintenance difficulties. Additionally, increased systemic inflammation, driven by gut permeability and dysbiosis, may exacerbate sleep fragmentation, leading to restless sleep, frequent awakenings, and non-restorative sleep patterns. This chronic disruption in sleep architecture can further perpetuate the neuropsychiatric symptoms observed in post-Finasteride syndrome (PFS), including fatigue, mood instability, and cognitive dysfunction (26).

Emotional Dysregulation and Irritability

Emotional dysregulation and irritability in post-Finasteride syndrome (PFS) may stem from disruptions in the gut microbiome that impair neurotransmitter balance and stress response mechanisms. Beneficial gut bacteria, such as *Lactobacillus* and *Bifidobacterium* spp., play a crucial role in GABA production, a key inhibitory neurotransmitter that promotes emotional stability and resilience to stress. A decline in these microbial populations may lead to reduced GABA availability, increasing susceptibility to heightened emotional reactivity, mood swings, and irritability. Additionally, alterations in the gut microbiome can

exacerbate stress responses by promoting systemic inflammation and dysregulating the hypothalamic-pituitary-adrenal (HPA) axis, leading to persistently elevated cortisol levels. This dysregulated stress response further amplifies emotional instability, contributing to heightened anxiety, agitation, and difficulty managing frustration. Addressing gut health may therefore be a critical component in mitigating the psychological symptoms associated with PFS (27).

Physical Complaints

Gastrointestinal Disturbances

Gastrointestinal disturbances associated with post-Finasteride syndrome (PFS) may be driven by gut microbiome imbalances that impair digestion, nutrient absorption, and immune regulation. A reduction in butyrate-producing bacteria, such as *Faecalibacterium prausnitzii* and *Roseburia* spp., can lead to decreased production of short-chain fatty acids (SCFAs), which are essential for maintaining gut barrier integrity and modulating inflammation. This shift may result in bloating, excessive gas, and altered bowel habits, manifesting as diarrhea or constipation. Additionally, microbial imbalances may favor the proliferation of pro-inflammatory species, triggering abdominal discomfort and cramping. Increased gut permeability, commonly referred to as "leaky gut," can allow microbial metabolites and endotoxins like lipopolysaccharides (LPS) to enter circulation, exacerbating systemic inflammation and potentially contributing to widespread symptoms beyond the gastrointestinal tract. Addressing these gut-related disruptions may be crucial in alleviating both digestive and systemic complaints in PFS patients (28).

Chronic Fatigue and Low Energy

Chronic fatigue and low energy in post-Finasteride syndrome (PFS) may be linked to disruptions in gut microbiota that impact mitochondrial function and overall metabolic health. A compromised gut ecosystem can lead to decreased production of essential metabolites, such as short-chain fatty acids (SCFAs), which play a role in cellular energy regulation. Additionally, dysbiosis may contribute to mitochondrial dysfunction by increasing oxidative stress and reducing ATP production, ultimately resulting in persistent fatigue, weakness, and reduced physical endurance. The imbalance in microbial populations may also lead to systemic inflammation, further exacerbating feelings of exhaustion and muscle soreness. Addressing gut health through microbiome restoration and inflammation reduction may be a critical step in alleviating fatigue-related symptoms in PFS patients (29).

Immune Dysregulation and Inflammation

Finasteride-induced alterations in gut microbiome diversity may profoundly impact immune regulation, leaving individuals more vulnerable to infections and inflammatory conditions. A reduction in beneficial microbial species compromises mucosal immunity, weakening the body's ability to mount appropriate defense responses. Simultaneously, a shift toward pro-inflammatory bacterial populations may trigger a persistent state of low-grade systemic inflammation, leading to widespread physiological repercussions. This chronic inflammatory response often manifests as joint pain, muscle aches, and general malaise, further contributing to fatigue and diminished overall well-being. Moreover, the persistent immune activation associated with dysbiosis may exacerbate neuroinflammation, potentially linking gut dysfunction to the cognitive impairment, mood disturbances, and neurological symptoms frequently reported by post-Finasteride syndrome (PFS) patients. Addressing these gut-related immune disruptions may hold the key to mitigating both physical and neuropsychiatric symptoms associated with Finasteride use (30).

Hormonal Imbalances and Sexual Dysfunction

Finasteride-induced disruptions in gut microbiota composition may have far-reaching consequences on hormonal balance, potentially exacerbating sexual dysfunction and metabolic disturbances. The gut

microbiome plays a crucial role in androgen metabolism, and a shift in microbial populations may impair testosterone synthesis, leading to a cascade of symptoms such as diminished libido, erectile dysfunction, and reduced physical stamina. Additionally, an increase in estrogen-dominant microbiota may further tip the hormonal scales, promoting estrogenic effects such as gynecomastia (male breast tissue growth), fat redistribution, and mood fluctuations. These hormonal imbalances may not only affect sexual health but also contribute to broader metabolic dysfunctions, including weight gain, insulin resistance, and fatigue. Given the intricate interplay between the gut, hormones, and overall physiological function, restoring microbial equilibrium may be a key therapeutic approach to mitigating these debilitating side effects (31).

Skin Issues and Autoimmune Reactions

Disruptions in gut microbiota balance can have profound effects on skin health and immune regulation, leading to a cascade of dermatological and autoimmune manifestations. The gut-skin axis plays a pivotal role in maintaining skin integrity and immune tolerance, and Finasteride-induced dysbiosis may fuel systemic inflammation that manifests externally as eczema, rosacea, persistent acne, or unexplained rashes. Additionally, increased intestinal permeability—commonly referred to as "leaky gut"—may allow microbial metabolites and toxins to enter circulation, triggering immune overactivation and heightening the risk of autoimmune conditions such as psoriasis, lupus, and inflammatory arthritis. By altering the gut microbial landscape and impairing immune homeostasis, these changes may not only exacerbate chronic skin conditions but also contribute to broader inflammatory and immune-mediated disorders. Addressing gut health through microbiome restoration strategies could be a critical step in mitigating these adverse effects (32).

Conclusion

Finasteride-induced disruption of the gut microbiome can trigger a cascade of adverse effects spanning neurological, psychological, immune, and metabolic health (33). By altering androgen levels—particularly reducing DHT—Finasteride indirectly reshapes microbial communities, leading to dysbiosis, increased intestinal permeability, and systemic inflammation. These changes may manifest as cognitive impairment ("brain fog"), anxiety, depression, chronic fatigue, immune dysregulation, and even hormonal imbalances contributing to sexual dysfunction.

Key bacterial populations affected include butyrate-producing species such as *Faecalibacterium prausnitzii* and *Roseburia spp.*, which are critical for maintaining gut barrier integrity and reducing inflammation. Additionally, reductions in *Lactobacillus spp.* and *Bifidobacterium spp.* may compromise serotonin and GABA production, exacerbating mood instability, sleep disturbances, and stress responses. A shift toward estrogen-dominant microbiota, coupled with a decline in beneficial bacteria, may further contribute to systemic inflammation and immune dysregulation.

Although direct research on Finasteride's impact on the gut microbiome remains limited, its known hormonal effects strongly suggest a role in microbial imbalance, intestinal permeability, and immune dysfunction. Restoring microbial diversity through targeted probiotic supplementation, prebiotic intake, and dietary interventions may help mitigate these effects and improve long-term health outcomes for affected individuals. Further research is necessary to fully elucidate the relationship between Finasteride, gut dysbiosis, and the development of post-Finasteride syndrome (PFS).

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Epilogue

The Millennium Health Centers, Inc. specializes in all things Brain.

In 2014, we started getting patients with symptoms that were associated with the use of either Finasteride or Dutasteride (5ARi). This was a unique group of patients with a complex array of symptoms inclusive of mood disorders, musculoskeletal dysfunction, and major issues with libido. AT that time, the focus was on the primary effects of these medications reducing the conversion of Testosterone to DHT. Additionally, from my neuroendocrine training, the 5ARi also affects the conversion of Progesterone to Allopregnanolone, a major mood stabilizing neurosteroid. When we returned both DHT and Allopregnanolone to normal levels, the majority of our cases improved. But there was a subpopulation who continued with symptoms regardless of what we corrected. The missing relationship, in my clinical opinion, is between the influence of 5-ARi on DHT and its importance nourishing the gut's microbiome. Now that this relationship is clearly supported by the scientific literature, the Millennium now includes an extensive GI evaluation to document the presence of Dysbiosis and then to correct this as well..